Epidural Steroid Injection for Low Back Pain and Sciatica: Fifty Years of Clinical Controversy

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INTRODUCTION

Low-back pain (LBP) is a common medical complaint in the general population, with significant socio-economic impact on the society[1,2]. Sources from developed countries cite the incidence of LBP as 5% per year with a prevalence of 60% to 90%[3,4]. In Australia, health costs for back problems were estimated to be A$700 million in 1993-94, while direct costs for back pain in the United Kingdom in 1998 were in excess of £1500 million[5,6]. Physicians continue to face significant difficulties managing low back pain despite the increased awareness of its magnitude.

Epidural steroid injections (ESI) performed at the lumbar or caudal epidural spaces are one of the commonest, non-operative interventions used in the management of LBP and sciatica. ESI has been a traditional form of treatment for these medical complaints for nearly 50 years[7,8]. Despite its long history and widespread use, the practice of ESI for the management of LBP and lumbosacral radiculopathy remains highly controversial, because of the inconclusive evidence to support its efficacy, and the possibility of rare, but potentially devastating adverse events. The controversy over the use of ESI has even boiled over into the public domain, especially in Australia, where sensationalized news in the mass media reporting disastrous consequences of ESI has led to increased anxiety among the general public, and a reduction in frequency of use of this technique, for fear of medico-legal consequences.

This article aims to provide an overview of the history, efficacy, complications and current role of ESI in the management of low back pain and sciatica. Although ESI can also be performed at the cervical and thoracic epidural spaces, this review will limit its discussion to ESI at the lumbosacral epidural spaces (which includes caudal epidural space), where the practice is most established, and yet most controversial.

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HISTORY OF EPIDURAL STEROID INJECTIONS

Epidural injections for low back pain and sciatica were first reported in 1901 and these involved the injection of cocaine via the sacral hiatus (caudal epidural space)[9,10,11]. Viner, in 1925, injected mixtures of procaine in normal saline, Ringer's solution, or "liquid petrolatum" into the caudal epidural space[12]. In 1930, Evans reported that 24, out of 40 patients with unilateral sciatica treated by caudal epidural injection of procaine and saline, had symptomatic pain relief[13]. In 1952, Robecchi and Capra reported using "periradicular" hydrocortisone to treat lumbar disk herniation. They speculated that their patient's "lumbago and sciatica" were caused by "inflammation" [14]. The following year, Lievre et al reported that five out of 20 patients with low back pain improved after caudal epidural corticosteroid injection with hydrocortisone[15]. In 1960, Brown and Goebert et al, in separate studies, also demonstrated highly successful response rates in patients with back pain and sciatica after caudal epidural injections with local anaesthetic and steroids[16,17,18]. MacNab first described transforaminal epidural injections (in contrast to "classical" translaminar epidural injections) in 1971, and since then, various studies have shown that these blocks could be used for diagnostic and therapeutic purposes in patients with radicular pain[19].

MECHANISM OF ACTION OF ESI

Mixter and Barr, in 1934, proposed that radicular pain could be caused by mechanical compression of spinal nerve roots by herniated intervertebral disks[20]. However, other researchers demonstrated that there was histological evidence of inflammation present in the nerve roots of patients with sciatica when they presented for surgery, and that the inflammation of these nerve roots corresponded to clinical symptoms[21,22]. Since then, other authors have commented that radiculopathic symptoms may occur from chemical irritation of the nerve roots even when there is no direct mechanical compression of nerve roots[23,24]. In fact, Rydevik et al observed that inflammation of the nerve root and the dorsal root ganglion appears to be the critical feature in the pathogenesis of radiculopathy[25].

McCarron et al provided further proof that inflammation of spinal nerve roots may be responsible for LBP and radiculopathy, when he injected autologous nucleus pulposus into the epidural space of dogs for five to seven days[26]. Microscopic analysis of the spinal cords and the

nerve roots of these dogs showed intense inflammation, which were not present in dogs of the control group given epidural normal saline. This study showed that even small amounts of nucleus pulposus could result in intense inflammatory response of the surrounding spinal neural tissues. This team hypothesized that high concentration of phospholipase A2 (PLA-2) and other enzymes present within the nucleus pulposus of the intervertebral disc were responsible for initiating the inflammatory reaction.

PLA-2 enzymes can leak out of "degenerated" or herniated intervertebral discs, and release arachidonic acid from cell membranes, which then sets off the inflammatory cascade[27]. Saal et al showed that samples from patients undergoing spinal disc surgeries had concentration of PLA-2 that was 20 to 10,000 times higher than normal tissue[28]. This inflammatory response causes neural oedema, and sensitises adjacent nerve roots and the dorsal root ganglia, which leads to clinical signs and symptoms of radiculopathy[29,30]. It has been shown that the nerve root and the dorsal root ganglion normally are only marginally sensitive to mechanical stimuli, but a significant increase in mechanical hyperalgesia (or sensitisation) occurs in the presence of inflammation[31,32]. The analgesic effect of steroids is therefore derived mainly from its ability to inhibit the action of PLA-2 on cell membranes to release arachidonic acid, thus preventing the initiation of the inflammatory cascade.

Besides possessing intense anti-inflammatory properties, epidural steroid has also been shown to inhibit neural transmission of normal nociceptive C fibres. In rat plantar nerve specimens, methylprednisolone acetate suppressed transmission in unmyelinated C fibres, and this effect was reversed when the steroid was removed, suggesting a direct, membrane stabilising effect on the neural membrane[33].

Glucocorticoid receptor sites have also been found on the norepinephrine, epinephrine, and 5-hydroxytryptamine neurons located in the lower brainstem, nucleus of the spinal tract of the trigeminal nerve, and the dorsal horn substantia gelatinosa. Thus, steroids may also act at the level of the spinal cord and the brain to modulate neural responsiveness to nociceptive input from the peripheral nociceptors[34].

EFFICACY

Efficacy Of "Classical" Translaminar / Caudal ESI

ESI is time-honored, having been performed for 50 years. A recent observational study of over 25,000 patients with back and radicular pain in the United States showed that ESI continues to be a common method of treatment in these patients[35]. However, efficacy of ESI has not been established, and its safety profile continues to be questioned.

Unfortunately, definitive outcome studies are sadly lacking with many studies being anecdotal reports, retrospective reviews, or uncontrolled studies. Treatment response rate obtained with these uncontrolled lumbar and caudal ESI studies varied from 20-100%, with an average response rate calculated from many studies at 60% by Kepes and Duncalf and 75% by White[36,37].

Although there are a number of randomised controlled clinical trials on the efficacy of ESI, many of these studies suffer from questionable methodologies. Some controlled studies had small sample sizes, while others had patient populations that were often poorly defined and not homogenous – with some patients having had both acute and chronic pain, while others had had back surgery. Furthermore, treatment protocols were variable, with variations in type and dosage of corticosteroid used, injection technique, patient diagnosis, length of follow-up, and outcome criteria[38,39]. According to Bogduk et al., no statistically valid conclusions could be drawn from the results of many of these controlled studies due to the small sample sizes or poorly controlled study designs[40]. (Table 1)

Table 1. Summary of Controlled Epidural Corticosteroid Studies

Study	Number of Patients	Approach	Follow-up	Steroid Benefit
Beliveau, 1971[41]	48	Caudal	1-3 mths	No
Dilke, 1973[42]	100	Lumbar	2 wk & 3 mths	Yes
Breivik, 1976[43]	35	Lumbar	3 wk	Yes
Snoek, 1977[44]	51	Lumbar	48 hr & 8-20 mths	No
Yates, 1978[45]	20	Caudal	1 mth	Yes
Klenerman,1984[46]	63	Lumbar	2 wk & 20 mths	No
Helliwell, 1985[47]	39	Lumbar	1 mth & 3 mths	Yes
Cuckler, 1985[48]	73	Lumbar	24 hr & 21 mths (average)	No
Ridley, 1988[49]	39	Lumbar	2 wk	Yes
Bush & Hillier, 1991[50]	23	Caudal	1 mth & 1 yr	Yes
Carette, 1997[51]	156	Lumbar	3 & 6 wk; 3 & 12 mths	No

Dilke et al, in randomised, double blind, placebo-controlled study, showed that patients given ESI, had significant greater improvement (60% vs. 31%) when assessed for initial pain relief, compared to the control group who received an interspinous ligament (non-epidural) injection[42]. Furthermore, ESI-treated patients had persistent benefit after 3 months, with lower use of analgesics, less referral for surgery and higher percentage of return to work.

Bush and Hillier, in another randomised, placebo-controlled study of clinically well-defined patients with radicular pain, paraesthesias, and positive straight leg raising, showed that there was significantly better pain relief at 4 weeks for ESI (via the caudal route) compared to placebo[50]. After one year, this treatment group continued to have a tendency (although not statistically significant) of less pain and more mobility and there were significantly fewer patients with a positive straight leg raise test. Hickey showed that there were additive benefits from serial ESI injections. After the first ESI injection, only 17% of his 250 patients had benefit; a further second injection 2 weeks later increased 44% more patients to the improved category, and a subsequent third ESI improved the remaining 39%[52].

Unfortunately, not all studies of ESI showed beneficial effects. Snoek et al treated 51 patients who had unilateral radicular pain (12 days to 36 weeks symptom duration) with lumbar ESI of either 2 ml of saline or 80 mg of methylprednisolone (2 ml) [44]. He found no significant differences in pain relief between the two groups after 48 hours. However, Benzon criticised this study's evaluation time of 48 hours as being too early for detecting a difference, as the corticosteroid effect may take as long as 4 to 6 days to become evident[7]. Furthermore, the injection volume of 2 ml may be too small to allow spread of the steroids to the affected nerve roots.

In another randomised, double-blind study involving 73 patients who had well-defined unilateral sciatica or spinal stenosis, Cuckler et al gave either 2 ml water, 80 mg methylprednisolone and 5 ml 1% procaine or 5 ml 1% procaine and 2 ml saline into the epidural space[48]. They too could not detect any statistically significant difference between ESI and control groups during short-term and long-term follow-ups. Criticisms were leveled at this study for the short evaluation period (1 day), and the non-uniformity of the study patients. Furthermore, study subjects received injections at the L3-L4 interspaces regardless of the level of their pain complaints.

More recently, Carette et al, in another randomised, double-blind clinical trial, administered ESI up to three times in patients who had pain and neurological deficits due to significant disc herniation[51]. They found that although ESI provided short-term improvements in leg pain and sensory deficits for up to six weeks, it offered no significant short-term and long-term functional benefits, nor did it reduce the need for surgery.

Investigators who reviewed the literature also came to different conclusions. In a systematic review, Koes et al concluded that ESI would not be useful, especially in patients with chronic low back pain without sciatica[53]. However, they did find that six out of 12 studies showed ESI to be more effective than control treatment for patients with sciatica, while the remaining six studies could not detect any significant differences. Furthermore, of the four best studies, two reported beneficial results and two reported negative results.

A meta-analysis of almost the same trials by Watts and Silagy showed that that epidural corticosteroids do have an analgesic effect on sciatica compared with control[54]. They discovered that the odds ratio for short-term pain relief with ESI (more than 75% improvement for up to 60 days) was 2.61, while for long-term pain relief (up to 12 months), the odds ratio for the ESI group was 1.87. This study quantitatively demonstrated that ESI is effective in the management of lumbosacral radicular pain when injected either via lumbar or caudal route.

McQuay and Moore reanalysed the same studies as those examined by Watts and Silagy, (and added the study by Carette et al to their analysis) to assess the extent of the benefit given by the steroids[55]. Of the 11 trials that gave short-term relief data (more than 75% improvement for up to 60 days), they found an overall statistically significant benefit with ESI when compared to controls. The number-needed-to-treat (NNT) for short-term relief was around 7.3. This means that for seven patients treated with epidural steroid, one will obtain more than 75% pain relief for up to 60 days. With regards to long-term improvements, there was again an overall statistically significant benefit but the NNT for long-term (12 weeks up to one year) improvement was about 13 for 50% pain relief. This means that for thirteen patients treated with epidural steroid, one will obtain more pain relief over this longer-term period. The authors commented that while the clinical benefit (NNT) values did appear disappointing, clinical benefits were still significantly better with ESI compared with controls, and that patients may choose to undergo ESI if it helps them reduce the need for other medications or to delay surgery.

Another area of controversy is the use of ESI in patients with symptomatic lumbar spinal stenosis. Ciocon et al showed that elderly patients with radiculopathic pain from lumbar spinal stenosis also responded well after ESI with duration of pain relief ranging from four to ten months. They concluded that ESI offered significant pain relief and would be a therapeutic option among elderly patients with spinal stenosis, especially when these patients are at high risk from the iatrogenic side effects of medications and are likely to be poor surgical risks[56]. Unfortunately recent studies by Rivest et al and Fukusaki et al did not manage to reproduce similar degrees of beneficial effects after ESI in patients with symptomatic spinal stenosis[57,58].

Efficacy Of Transforaminal ESI

The above discussion on the efficacy of ESI is based on ESI studies performed using classical "translaminar" lumbar epidural and caudal epidural approaches. However, Derby et al contends that ESI should be performed via the transforaminal approach to the epidural space, as it more reliably place the corticosteroid in the anterior epidural space where the most pain-sensitive structures are located[59]. Lutz et al evaluated the therapeutic efficacy of fluoroscopic guided, selective transforaminal epidural injections in

pulposus[60]. In their study, 52 of 69 (75%) patients treated had 50% pain relief or greater, and returned to pre-injury physical activities when evaluated at an average of 80 weeks after the injections. However less favorable results were obtained in patients who had pain of greater than six months in duration, or when there was severe lateral bony stenosis.

Performing an epidural via the transforaminal approach allows only selected nerve roots to be blocked, and could be used as a diagnostic tool to identify or confirm a specific nerve root as a pain generator when the diagnosis is not clear based on other clinical evidence[61,62]. Derby et al showed that there was good correlation between success and failure of the transforaminal ESI and surgical outcomes in patients who had radicular pain lasting more than one year[63]. This team discovered that patients with pain lasting more than one year and who have had a positive response to steroid injected into the symptomatic nerve root (roots) had a positive surgical outcome of 85%. Conversely, patients who did not respond to the steroid and had pain for more than one year (95%) generally had poor surgical outcomes.

A recent randomised, double-blind trial of 160 patients with sciatica who had either methylprednisolone or saline injected epidurally via the transforaminal route, did not show any significant long term beneficial effects for the ESI group, although there appeared to better recovery of leg pain and patient satisfaction for the steroid group at two weeks[64]. On the other hand, Vad et al showed in a randomised study that patients receiving transforaminal ESI had a success rate of 84%, compared with 48% in the control group receiving trigger-point injections, after an average follow-up period of 1.4 years[65].

To date, there have been no studies comparing the effects of different steroids injected during ESI, nor the dosages of steroids injected. There was a single study by Kraemer et al that claimed the superiority of epidural transforaminal ESI over "classical" translaminar ESI in patients with lumbar radiculopathy[66]. However, both epidural groups had better results than the control group.

SIDE EFFECTS AND COMPLICATIONS

Technical Complications

Technical side effects include pain at the injection site and transient exacerbation of back pain symptoms, usually lasting for less than two days. Accidental dural puncture during the injection attempt can lead to troublesome, but usually self-limiting headaches. In experienced hands, accidental dural punctures should be less than 1%. In fact, MacDonald cited an incidence of 0.33% for 5865 lumbar epidural injections[67].

Epidural Haematoma

Epidural haematoma is extremely uncommon after ESI, with an incidence of one case per 200,000 epidural blocks[68]. A survey of the literature revealed only two cases after ESI. The most recent case of epidural haematoma occurred in a healthy 34-year-old man who had onset of acute cervical myelopathy from a large cervical epidural haematoma eight days after a cervical epidural steroid block[69]. This patient recovered almost completely after prompt surgical intervention.

Infections

Although rare, bacterial meningitis and epidural

Table 2. Reported Cases Of Epidural Abscess After Epidural Steroid Injection

Study	Injections	Findings	Outcome	Medical History
Shealy[75]	L, X 4, M	Squamous cell cancer and inflammatory cells	Foot drop, late death due to cancer	Cancer
Chan and Leung[76]	L, X 1, T	Staphylococcus aureus	T8 paraplegia, near complete recovery	Diabetes
Goucke and Grazioti[77]	L, X 3 , M	Staphylococcus aureus	Death	Diabetes, recent postoperative staphylococcus sepsis
Waldman[78]	C, X 3	Staphylococcus aureus	C6-level quadriparesis	None
Marmourian[79]	L, X 1	Staphylococcus aureus	Paraplegia	Diabetes
Knight[80]	S, X2, T + P	Staphylococcus aureus	Paraplegia	Diabetes
Bromage[81]	Th, X 6, M	Not stated	Quadriplegia	Postherpetic neuralgia
Strong[82]	Th, X1, M + B X 10 via 2 catheters	Staphylococcus aureus	Complete recovery	Resolving acute herpes zoster
Kaul[83]	L, X 1, H	Inflammatory cells	Complete recovery	None
Koka[84]	L	Conservative treatment	Complete recovery	

L, lumbar; C, Cervical; S, Caudal; Th, Thoracic; M, Methylprednisolone acetate; T, triamcinolone diacetate; B, bupivacaine; P, procaine; H, Hydrocortisone
Adapted from Molloy RE & Benzon HT [38].

abscesses have been reported after ESI. Dougherty and Fraser reported two cases of bacterial meningitis after attempted ESI[70]. One of the patients had an accidental dural puncture during the ESI procedure. It is believed that meningitis is unlikely to occur unless unintentional dural puncture occurs and Abram recommends that ESI should be abandoned if dural puncture occurs[71].

From 1966 till 2002, about 10 cases of epidural abscesses occurring after ESI have been reported. This incidence should be taken into the context that tens of thousands of ESI has been performed since 1966. Even allowing for under-reporting, the incidence appears to be less than 0.01%[72]. Furthermore, most epidural abscesses occur spontaneously from haematogenous spread or from adjacent vertebral infection, although it can also occur following spinal and epidural anaesthesia and epidural catheter insertion for postoperative pain relief[73,74]. Note that two of the case reports listed in Table 2 involved patients who had thoracic epidural steroid for treatment of pain secondary to herpes zoster[81,82]. One patient had an epidural catheter inserted and both received multiple steroid injections. Most of the abscesses cultured *Staphylococcus Aureus* and occurred mainly in patients with diabetes mellitus or who were in immunocompromised states. Rapid diagnosis and therapy, which may include surgical drainage, are necessary to avoid any permanent neurologic deficit. (Table 2)

Ophthamological Complications

There have been a few reports of retinal venous haemorrhage and ambylopia occurring after epidural injections of steroids and local anaesthetics for treatment of low back pain and sciatica[85-89]. One common theme in these rare reports were the large volume of injectate used (exceeded 40 ml), and it is believed that this may lead to increased spinal fluid pressure in the optic sheath subarachnoid space which in turn increases the retinal venous pressure.

Hormonal Disturbances

Adrenal suppression has been reported as a side effect after ESI, arising from the systemic effect of the steroid[90,91]. Kay et al demonstrated that decreased levels of adrenocorticotropic hormone (ACTH) and cortisol, as well as abnormal cortisol response to synthetic ACTH could be present up for up to three months post-ESI[92]. Some authors recommend that perioperative corticosteroid replacements should be considered in patients who had recent ESI undergoing surgery, because of the relative adrenal insufficiency[90,92]. Besides suppression of the pituitary-adrenal axis, there have also been reports of Cushing's syndrome, excessive weight gain, fluid retention, hyperglycaemia, acute hypertension and congestive cardiac failure after ESI[18,91].

Neurotoxicity

Drugs used for ESI have been blamed for causing neurotoxicity, and this has been attributed to either the depot steroids or their preservatives. The fear of neurotoxicity from ESI has mainly been extrapolated from reports of neurotoxicity that have occurred from the previously widespread practice of intrathecal steroid injections. Nelson has consistently questioned the efficacy and the safety of intrathecal steroid injections[93,94]. His concern has been that the preservative polyethylene glycol (PEG) – which is present in commonly used depot steroids, Aristocort (triamcinolone depot steroid) and Depo-Medrol (methylprednisolone depot steroid) – has the potential to cause neurotoxicity in the form of arachnoiditis, sterile meningitis and pachymeningitis when injected intrathecally. He recommended that steroid injections should not be administered into the intrathecal space. He also warned against ESI because of the theoretical risk that the steroid might migrate into the subarachnoid space via connecting blood vessels, or as a result of accidental subdural or intrathecal injection. (Table 3-commonly used depot steroids)

Some of Nelson's fears in intraspinal steroid injections have been raised by an earlier work by Seghal et al which revealed that subarachnoid placement of 80 mg methylprednisolone can cause a transient increase in CSF protein and a pleocytosis that persisted for a few weeks[95]. On the other hand, Delaney et al. showed, in a cat epidural model, that a single ESI with Aristocort did not produce evidence of tissue damage when compared to matched controls[96]. Cicala et al also demonstrated that single ESI with Depomedrol in a rabbit did not result in tissue reaction significantly different from a control group[97].

Benzon et al examined the effect of polyethylene glycol on the electrophysiology of rabbit nerves[98]. They did not detect any decrease in neural conduction at the clinically relevant PEG concentration of 3%. This finding is significant as the common drugs used in clinical practice, Aristocort and Depo-Medrol contain only 3% PEG. Furthermore, the concentration of PEG would be further diluted by the concurrent use of saline or local anesthetic. Abram and associates also studied the effects of serial intrathecal steroid injections on the rat spinal cord, and concluded that accidental intrathecal injection during ESI has a low potential to produce neurotoxicity[99]. Abram and O'Connor, in a review of 64 clinical series of ESI involving about 7000 patients, did not manage to detect even a single report of arachnoiditis[100].

There have also been suspicions raised about possibility of neurotoxicity arising from ESI injection of depot steroids containing the preservative benzyl alcohol[101,102]. Abram, however, argued that there has not been any study that has demonstrated the linkage between neurotoxicity with either benzyl alcohol or polyethylene glycol[102]. Other steroid preparations have also been used for ESI injections (Table 3). This includes dexamethasone, which is a soluble steroid preparation. Abram, however, advised against the use of soluble steroids because it is rapidly cleared from the epidural space. Furthermore, dexamethasone has significant mineralocorticoid activity and this could lead to salt and fluid retention[71].

Table 3. Constituents And Comparison Of Commonly

Steroid	Anti-inflammatory Potency	Salt Retention Property
Dexamethasone (soluble steroid)	25-30	30
Depo-Medrol Methylprednisolone PEG 3350 Myristyl-gamma-picolinium PH adjusted to 3.5-7 with NaOH or HCL	5	0
Celestone Betamethasone-sodium- phosphate Betamethasone acetate	25-30	0
Aristocort Triamcinolone diacetate Polysorbate 80 NF PEG 3350 Benzyl alcohol NaCl	5	0
Kenacort-A Triamcinolone acetonide Polysorbate 80 Benzyl alcohol Carmellose sodium NaCl		

CURRENT ROLE OF ESI

In view of the vastly conflicting data on the efficacy of ESI, and the potential for rare but severe side effects, what should be the current stand on ESI? Although the efficacy of ESI has not been conclusively proven, many studies, as discussed above, do point to good short- to intermediate-term success after ESI in selected patients. Many reviews by distinguished researchers have continued to recommended the use of ESI as part of a multidisciplinary treatment programme, especially for patients with acute radicular pain, herniated disc, or an acute on chronic flare-up of back and radicular pain[8,103-105]. According to Bogduk, more than 40 papers have been published on lumbar and caudal epidural corticosteroid injections involving a total of more than 4,000 patients, and only four of these papers have recommended against the use of lumbosacral epidural corticosteroids[40].

Abram states that in order for ESI treatment to be more effective and to minimise side effects, pain physicians should limit its use to patients with evidence of radiculopathy[106]. Evidence showed that ESI was most effective in patients with pain of discogenic origin, especially if the condition was acute and involved a significant disc bulge or herniation, and was associated with significant radicular pain[7,37,107]. Patients having internal disc disruption from an annular tear without significant disc degeneration or radicular pain respond less well to epidural corticosteroids[107].

Jamison listed the following factors as associated with poor response to ESI: (1) numerous previous treatments for pain without any improvement, (2) current use of multiple medications, and (3) back pain that does not increase with activities[108]. Other factors that may reduce chances for ESI success include long symptom duration, previous surgery, history of substance abuse, lack of employment and heavy smoking.

Contraindications for ESI include local or systemic infection, coagulopathy and history of allergy to steroids or its preservatives[109]. There is an increased risk of hyperglycaemia and epidural abscess formation, albeit small, after ESI in diabetic patients. Patients must be informed of the potential risks prior to the procedure.

Abram recommends that only moderate doses of steroids (50 mg triamcinolone diacetate; 80 mg methylprednisolone acetate) be used for each ESI[106]. He advised against any repeat injection if there was no response to the first ESI. However if there is partial pain relief from the initial ESI, a further one or two more ESI injections at two-week intervals could be performed to further enhance and prolong the beneficial effects[40,110]. Further repeat injections should not be offered when the response to ESI is only transient, but could be considered if there are prolonged responses of six to twelve months or longer[38]. The total number of ESI per year should not exceed three, due to the cumulative side effects of the steroids.

Many authors have also recommended that ESI (translaminar and transforminal approaches) should be performed under fluoroscopic guidance to ensure proper and accurate needle placement. McLain states that one possible reason why ESI fails is the inability to place the medications appropriately at the desired target nerve, while Bogduk said that conventional "blind" ESI may not guarantee that the depot corticosteroid is deposited in the vicinity of the pain-generating tissues[111,112]. Other studies have also confirmed that ESI, especially via the caudal approach, performed without fluoroscopy led to a significant incidence of improper needle placement[113]. The use of fluoroscopy would also help detect the presence of significant anatomic anomalies such as a midline epidural septum or multiple separate epidural compartments that might restrict the desired flow of the epidural injectate to the suspected pain generator[114]. Lastly, fluoroscopy helps to prevent accidental intravascular injections, because the absence of blood return with needle aspiration before an injection is not a reliable indicator of intravascular needle placement[115].

Despite such recommendations, a recent national

survey in the United States showed that there are still considerable variations in current practices of ESI[116]. For example, fluoroscopy during ESI was used more often in private practices compared to academic centers. There was also no consensus in the use of ESI employing the transforaminal approach.

CONCLUSION

ESI is not a generic treatment for all patients with complaints of lower back pain. Patients must be carefully, and systematically evaluated, and only appropriate patients should be offered this treatment. Invasive procedures, like ESI, should always be used as part of a comprehensive multidisciplinary treatment programme, which may include medications, physical therapy, psychological therapy and even major surgical procedures. ESI must always be recommended in conjunction with a formal physical therapy program such as dynamic spine stabilization programs, which include spine mobility, strengthening exercises, and postural and dynamic body mechanics training. Epidural injections can provide a period of pain relief, allowing disc and nerve root injuries to recover, while patients can continue with the physical rehabilitation programme without excessive reliance on oral analgesics[117].

It is clear that further comparative studies are required to clearly define the advantages and disadvantages of the use of fluoroscopy during ESI, transforaminal technique of ESI and newer techniques like epidural steroid administration via epiduroscopes.

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