

Paraplegia after lumbosacral nerve root block: report of three cases

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Abstract

Background context: Lumbar nerve root blocks and epidural steroid injections are frequently employed in the management of degenerative conditions of the lumbar spine, but relatively few papers have been published that address the complications associated with these interventions. Serious complications include epidural abscess, arachnoiditis, epidural hematoma, cerebrospinal fluid fistula and hypersensitivity reaction to injectate. Although transient paraparesis has been described after inadvertent intrathecal injection, an immediate and lasting deficit has not been previously described as sequelae of a nerve root block. **Purpose:** We present three cases in which either persisting paraplegia or paraparesis occurred immediately after administration of a lumbar nerve root block and propose a mechanism for this devastating but previously unreported complication.

Study design/setting: Case reports of three patients.

Patient sample: Three patients, two women and one man ranging in age from 42 to 64 years, underwent three procedures performed at three different facilities, in the hands of two different injectionists. In each instance, penetration of the dura was not thought to have occurred. In two procedures the needles were placed transforamenally, one at L3–4 on the left and one at L3–4 on the right, and in the third the needle tip was placed immediately lateral to the S1 nerve root.

Outcome measures: Patient follow-up data from medical office records.

Methods: In each case, needle placement was verified with injection of a contrast media in conjunction with either computerized tomography or biplanar fluoroscopy. No backbleeding or cerebrospinal fluid was encountered upon aspiration in any of the procedures. Magnetic resonance imaging (MRI) was performed within 48 hours of injury in all patients.

Results: In each patient, paraplegia suddenly ensued after instillation of the steroid solution and, in each instance, postprocedure MRI revealed increased signal in the low thoracic spinal cord on T2-weighted imaging consistent with edema. The sudden onset of neurological deficit and the imaging changes noted in the spinal cord point to a vascular explanation for these injuries. We postulate that in these patients the spinal needle either penetrated or caused injury to an abnormally low dominant radiculomedullary artery, a recognized anatomical variant. This vessel, also known as the artery of Adamkiewicz, in 85% of individuals arises between T9 and L2, usually from the left, but in a minority of people may arise from the lower lumbar spine and rarely even from as low as S1. The artery of Adamkiewicz travels with the nerve root through the neural foramen and irrigates the anterior spinal artery. Injury of it or injection of particulate matter into it, as what may happen with the commonly used epidural steroid injectates, may result in infarction of the lower thoracic spinal cord, producing the clinical and imaging findings seen in these three patients.

Conclusions: We present the cases of three patients who had lasting paraplegia or paraparesis after the performance of a nerve root block. We propose that the mechanism for this rare but devastating complication is the concurrence of two uncommon circumstances, the presence of an unusually low origin of the artery of Adamkiewicz and an undetected intraarterial penetration of the procedure needle.

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Introduction

Lumbosacral nerve root blocks and epidural steroid injections are frequently used in the management of numerous degenerative conditions of the lumbar spine, including lumbar spinal stenosis, herniated lumbar intervertebral disc disease and facet arthropathy. Relatively few papers, however, have been published that address the complications associated with these interventions. A survey of large series of fluoroscopically guided epidural steroid injections reveals complication rates ranging from 0% to 9.6% [1]. The most commonly reported complication is headache, which is generally self-limited. Serious complications include epidural abscess, arachnoiditis, epidural hematoma, cerebrospinal fluid fistula and hypersensitivity reaction to injectate. Transient paraparesis resulting from epidural steroid injections was previously reported in a case involving inadvertent penetration of the thecal sac by an injection of local anesthetic [2]. We report two cases of paraplegia and one case of serious paraparesis in which neurologic function did not recover after performance of a lumbosacral nerve root block. In addition, we postulate the mechanisms of these neurological injuries. The injuries occurred at three different facilities, in the hands of two different injectionists, and in instances where penetration of the dura was not thought to have occurred. None of the patients had prior symptoms referable to the spinal cord. Magnetic resonance imaging (MRI) was performed within 24 hours of injury in each case revealed signal abnormality in the low thoracic spinal cord on T2-weighted imaging consistent with edema not present on MRIs of any of the three patients before the procedures.

Case reports

Case 1

A 64-year-old woman presented with 2.5 months of complaints of low back pain, bilateral buttock and leg pain and numbness in her left leg after falling on her back. Four years before presentation, she had undergone laminectomy of L4–5 and a fusion with pedicle screw instrumentation to treat symptoms of progressive low back and leg pain from lumbar spinal stenosis and spondylolisthesis at this level. After surgery, she had good relief of symptoms for 2 years. She then began to complain of back and right leg pain that initially resolved without intervention only to return after several months and persist. Her past medical history was unremarkable. The neurologic examination was normal. Straight leg raising was negative. MRI revealed diffuse facet arthropathy, L3–4 moderate central stenosis and foraminal stenosis worse on the left and changes related to the surgery at L4–5 but without stenosis. As a diagnostic and therapeutic intervention, the patient underwent biplanar fluoroscopically guided contrast-enhanced right L3–4 and L4–5 transforaminal nerve root blocks. Using a two-needle technique, 25-g spinal needles were advanced into the right L3 and L4 nerve foramina to access the epidural space. After syringe aspiration that pro-

duced no cerebrospinal fluid or blood, 1-cc of Omnipaque (Nycomed Inc., Princeton, NJ, USA) was injected at each level to confirm accurate needle tip placement. Then 3 cc of an injectate consisting of preservative-free marcaine 0.25% with 12 mg betamethasone (Celestone; Schering-Plough, Kenilworth, NJ, USA) was introduced at each level. The patient rapidly developed lower extremity paraparesis with an L1 motor level and no sensory or sphincter deficits. An emergency MRI performed within several hours of injury (Fig. 1, bottom left) revealed high signal intensity upon T2 imaging in the distal thoracic spinal cord consistent with spinal cord edema. At 1-month follow-up, her lower extremity strength has improved from 3/5 to 4/5 bilaterally. Follow-up MRI at 1 month reveals resolution of the abnormal spinal cord signal.

Case 2

A 51-year-old woman with a history of three prior lumbar spine surgeries, including a fusion of L5–S1 with pedicle screw instrumentation, presented with symptoms consistent with a left L3 radiculopathy. Imaging revealed a left-sided foraminal disc herniation producing compression of the left L3 nerve root. Under computed tomography (CT) guidance, a nerve root block was attempted. A 20-g spinal needle was advanced into the left L3–4 foramen. After aspiration produced no backbleeding or cerebrospinal fluid, 1 cc of preservative-free Lidocaine (Astra Zeneca, London, UK) 1% was instilled, producing symptoms of low back pressure and reproduction of the lower extremity radiculopathy. After a second negative aspiration, 1 cc of 40 mg/cc methylprednisolone acetate injectable suspension (Depo-Medrol; Pharmacia and Upjohn, Peapock, NJ, USA) mixed with 0.2 cc of Isovue 300 (Bracco Diagnostics, Princeton, NJ, USA) contrast was instilled. A CT scan was performed revealing contrast extravasation around the left L3 nerve root in the neural foramen. The patient complained of low back and chest pain, shortness of breath and numbness and paralysis of the lower extremities. Neurological examination was that of a complete spinal cord injury at the low thoracic level including loss of sphincter function. MRI of the thoracolumbar area performed in evening the same day (Fig. 1, bottom right) revealed swelling T2 signal change in the distal spinal cord consistent with edema. At 8-month follow-up, there has been no recovery of neurological function.

Case 3

A 42-year-old man presented with a long history of low back pain and episodic left lower extremity pain for which he had undergone lumbar laminotomy and microdiscectomy on two prior occasions. Over the course of 3 years, he was treated with lumbosacral steroid injections on at least 13 occasions, which generally produced at least partial relief of his symptoms. He returned with a left-sided radiculopathy consistent with MRI findings of spondylosis at L5–S1 most prominent on the left. Under CT guidance, a 22-g spinal needle was advanced until the tip was just lateral to the left

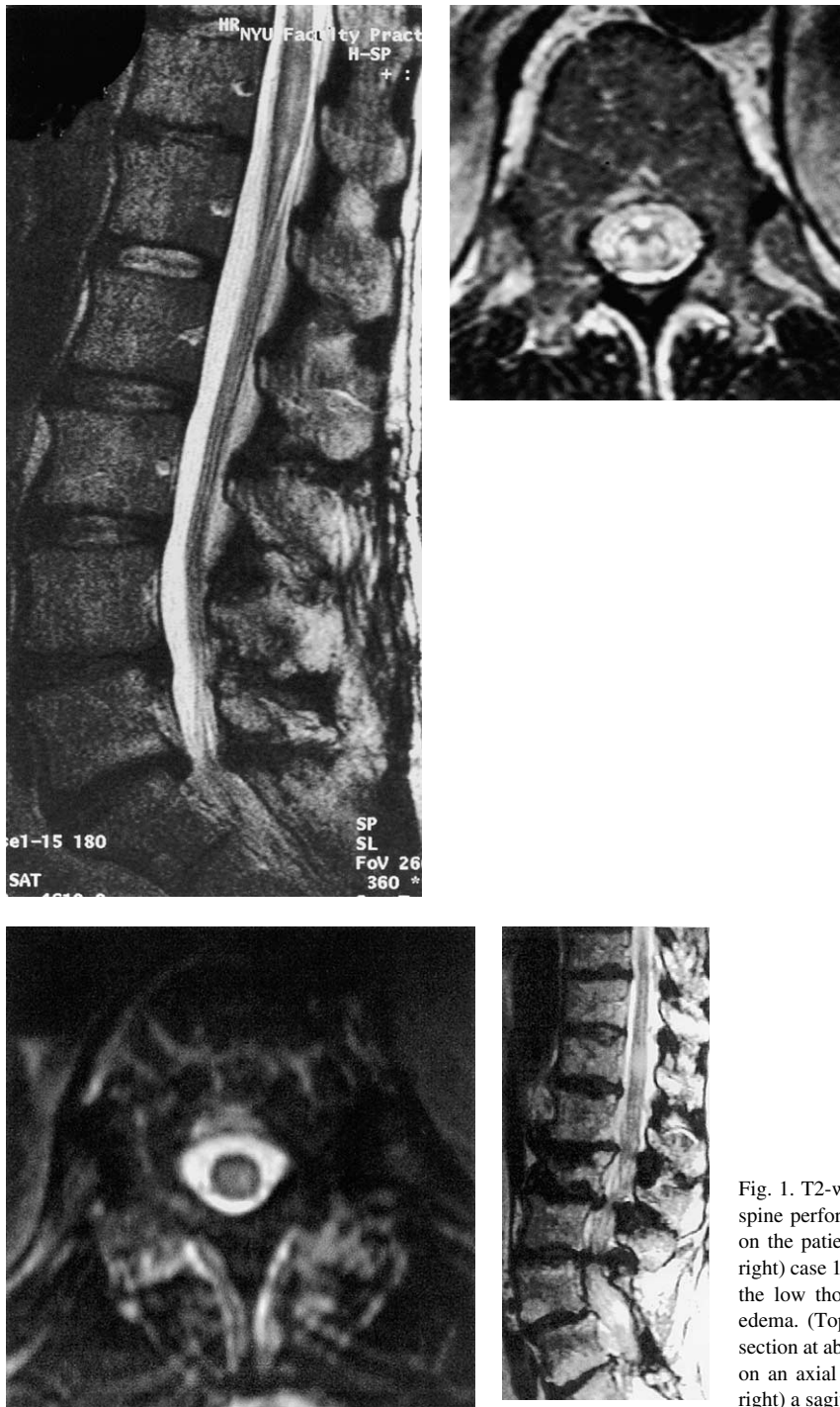


Fig. 1. T2-weighted magnetic resonance image of the thoracolumbar spine performed within 48 hours of the onset of neurological deficit on the patient from (top left and right) case 3 and (bottom left and right) case 1. (Top left) A sagittal section reveals marked expansion of the low thoracic spinal cord with increased signal consistent with edema. (Top right) These findings are also appreciated on an axial section at about the T12 level. (Bottom left) Similar findings are noted on an axial slice at about T12 in the patient in case 1 and (bottom right) a sagittal slice taken from the patient in case 2.

S1 nerve root. The needle position was confirmed after introduction of a small amount of a contrast agent. Aspiration produced no backbleeding or cerebrospinal fluid, and 1 cc of preservative-free Lidocaine 1% was injected, producing no significant clinical change. At this point, 1 cc of 40 mg/cc methylprednisolone acetate injectable suspension (Depo-Medrol; Pharmacia and Upjohn) was instilled. The patient complained of chest pain and bilateral lower extremity weakness and numbness. Neurological examination revealed a T10 sensory level, 0/5 function in all lower extremity

muscle groups and loss of sphincter tone. MRI performed within 6 hours (Fig. 1, top left and right) demonstrated expansion of the lower thoracic spinal cord and increased signal on T2-weighted imaging. At over 5-year follow-up, the patient has had no recovery of neurological function.

Discussion

Serious complications of lumbosacral steroid injections include epidural abscess [3–6], epidural hematoma [7,8]

and hypersensitivity reactions to injectate [9–11]. Neurological dysfunction has been reported as a late complication of dural penetration and subarachnoid injections with development of arachnoiditis [12,13]. Although transient paraparesis has been reported as an immediate sequelae of an intrathecal injection with local anesthetic [2], our report is the first to describe immediate and lasting neurological deficit.

The pattern of neurological deficit, paraparesis or paraplegia, loss of sphincter function and a sensory level in the low thoracic area is consistent with that seen in infarction of the thoracolumbar spinal cord [14]. The presence of edema in the lower spinal cord and the abrupt onset of neurological deficit seen in these three cases point to a vascular cause of injury. We postulate that in these patients the spinal needle either penetrated or caused injury to an abnormally low dominant radiculomedullary artery, a recognized anatomical variant.

The blood supply to the spinal cord normally comes from a single anterior spinal artery and two posterior spinal arteries. The anterior spinal artery is formed at its rostral end near the cervicomedullary junction by the joining of two anterior spinal branches of the vertebral arteries and runs the length of the spinal cord supplying its anterior two thirds. The posterior spinal arteries are smaller vessels, and their course may be discontinuous. They run in the posterior lateral aspects of the spinal cord and provide the blood supply to its posterior third. Radicular arteries that arise bilaterally from the aorta at every vertebral level and travel with the segmental nerve roots into the neural foramen also supply the anterior spinal artery, but most of these provide a blood supply only to the nerve root and do not supply the spinal cord. A few large radicular branches are termed radiculomedullary arteries, because they give off branches that ascend and descend within the dural tube to perfuse the anterior spinal artery and spinal cord. Although there are an average of three radiculomedullary arteries supplying the cervical spinal cord, below T8 the major supply to the anterior spinal artery is by means of a single, large radiculomedullary branch: the artery of Adamkiewicz. The artery of Adamkiewicz arises in 85% of individuals between T9 and L2, usually from the left [15]. Its origin, however, is highly variable [16] and in a minority of people may arise from the lower vertebrae in the lumbar spine [17] (Fig. 2) and rarely even from as low as S1.

Spinal cord infarction in its vascular territory would be expected to produce the clinical and imaging findings seen in the three patients presented [14,18].

We suggest that the mechanism of injury is a spinal cord infarction caused by the concurrence of two uncommon circumstances: the presence of an unusually low origin of the artery of Adamkiewicz and an undetected intraarterial penetration of the procedure needle. The subsequent spinal cord infarction could have resulted in one of two ways. Intraarterial injection of the steroid preparation led to embolization of particulate matter into the spinal cord, because the available steroid preparations are suspensions that are insoluble

in water. Alternately, direct injury to the artery of Adamkiewicz could have led to its thrombosis. Although the radicular or segmental arteries of the thoracolumbar area are frequently ligated to create exposure during spine surgery, this only very rarely leads to vascular insufficiency of the spinal cord, because there are anastomoses with collateral arteries as it enters the neural foramen. Injury of the artery of Adamkiewicz in the neural foramen may occur distal to collateral vessels and result in spinal cord infarction.

Acute transverse myelitis (ATM) is a rare condition resulting from an infectious or inflammatory process involving the spinal cord. Patients with ATM typically present with a subacute onset of paraparesis or paraplegia. Preceding infection is present in up to 50% of these patients, whereas other cases have been reported to occur after vaccinations or in association with autoimmune disorders [19]. ATM has also been reported after injection of chymopapain, occurring approximately 3 weeks after the procedure [20]. The immediate deficits seen in the cases under present consideration, however, led us to consider a vascular cause for injury in favor of ATM. Upon review of the literature, we found that there are reports of acute onset lower extremity paralysis thought to be caused by inadvertent intraarterial injection during intramuscular injection of penicillin that were reported using the term “transverse myelitis,” used in the past interchangeably with spinal cord infarction [21–26]. The clinical presentation in these cases was remarkably similar to that of the present cases: patients experienced rapid and total loss of motor and sensory neurological function at about the T10 level. These patients were young children in whom intraarterial injection into the superior gluteal artery of a highly viscous penicillin injectate was thought to have refluxed into the aorta and then moved antegrade into the dominant radiculomedullary artery supplying the lower spinal cord [22–25].

An emergent arteriogram was not performed in any of these cases. Only recently, after discussion with our interventional neuroradiology colleagues, have we become convinced of a vascular cause for these injuries. Although performing late angiography would be interesting, we have decided not to request it; our patients all have long-standing fixed deficit and would not expect any benefit to offset the risk of an additional invasive procedure. If, however, acute neurological deficit is seen in the course of future procedures, we plan to perform emergent arteriography and would recommend the same to other practitioners.

The use of fluoroscopic or CT guidance has improved the accuracy of these procedures and reduced the frequency of inadvertent intrathecal injections. More reliable placement of procedure needles in the vicinity of the neural foramen, a highly vascular area, however, may increase the risk of intravascular penetration. Supportive evidence of an intraarterial injection in our patients was the transient complaints of flushing, dyspnea and chest pain, consistent with symptoms of intraarterial injections of Lidocaine reported elsewhere [1]. Unfortunately, a negative aspiration is not a



Fig. 2. Anteroposterior projection (left) digitally subtracted and (right) partially subtracted spinal angiogram in the lumbar region of a different patient undergoing angiography for reasons unrelated to this report. The catheter tip is situated in the right L3 radicular artery and the artery of Adamkiewicz is shown entering the spinal canal by means of the right L3–4 neural foramen and constituting an anterior spinal artery. Note the ascending and descending limbs and characteristic “hairpin” turn the vessel makes on the ventral surface of the spinal cord. It is conceivable that in this patient a nerve root block performed at the right L3–4 foramen could penetrate the artery of Adamkiewicz.

reliable indicator in excluding vascular penetration [27]. Furman found in assessing 761 transforaminal injections that even when aspiration is routinely performed, the rate of intravascular injection was 11.2% [28]. Reasons for a falsely negative aspiration include a needle tip lodged against the vessel’s inner wall or aspiration causing the vessel to collapse.

An injection into the venous plexus of the epidural space and neural foramen would not be expected to cause injury, because the venous network in this area is richly anastomotic; thrombosis of a few vessels should not produce venous hypertension. In addition, injected material should move away from the spinal cord by means of the intervertebral veins. Although all three patients in this series had a history of prior surgery, it is unclear whether this could have been a contributing factor.

A common factor in all three cases presented in this report is the use of biplanar fluoroscopy or CT to provide precise placement of the needle tip at the point where the nerve root emerges from the neural foramen. Because the radiculomedullary artery travels with the nerve root at this point, perhaps it was this effort to obtain precise needle placement that actually increased the risk of the procedure. Consequently, it may be that injections further from the foramen or by means of a translaminar route are safer. An additional point may be that a less particulate or viscous injectate may be safer to employ.

In summary, we present the cases of three patients that had lasting paraplegia or paraparesis after the performance of a lumbosacral nerve root block. We propose that the mechanism for this rare but devastating complication is the concurrence of two uncommon circumstances, the presence of an unusually low origin of the artery of Adamkiewicz and an undetected intraarterial penetration of the procedure needle.

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References

- [1] Botwin KP, Gruber RD, Bouchlas CG, Torres-Ramos FM, Freeman TL, Slaten WK. Complications of fluoroscopically guided transforaminal lumbar epidural injections. *Arch Phys Med Rehabil* 2000; 81(8):1045–50.
- [2] McLain RF, Fry M, Hecht ST. Transient paralysis associated with epidural steroid injection. *J Spinal Disord* 1997;10(5):441–4.
- [3] Shealy CN. Dangers of spinal injections without proper diagnosis. *JAMA* 1966;197(13):1104–6.

- [4] Chan ST, Leung S. Spinal epidural abscess following steroid injection for sciatica. Case report. *Spine* 1989;14(1):106–8.
- [5] Goris H, Wilms G, Hermans B, Schillebeeckx J. Spinal epidural abscess complicating epidural infiltration: CT and MR findings. *Eur Radiol* 1998;8(6):1058.
- [6] Bromage PR. Spinal extradural abscess: pursuit of vigilance. *Br J Anaesth* 1993;70(4):471–3.
- [7] Williams KN, Jackowski A, Evans PJ. Epidural haematoma requiring surgical decompression following repeated cervical epidural steroid injections for chronic pain. *Pain* 1990;42(2):197–9.
- [8] Benzon HT, Wong HY, Siddiqui T, Ondra S. Caution in performing epidural injections in patients on several antiplatelet drugs. *Anesthesiology* 1999;91(5):1558–9.
- [9] Christensen FR, Andersen LW. Adverse reaction to extradural buprenorphine. *Br J Anaesth* 1982;54(4):476.
- [10] Simon DL, Kunz RD, German JD, Zivkovich V. Allergic or pseudoallergic reaction following epidural steroid deposition and skin testing. *Reg Anesth* 1989;14(5):253–5.
- [11] DeSio JM, Kahn CH, Warfield CA. Facial flushing and/or generalized erythema after epidural steroid injection. *Anesth Analg* 1995;80(3):617–9.
- [12] Abram S, O'Conner T. Complications associated with epidural steroid injections. *Regional Anesthesia* 1996;21(2):149–62.
- [13] Nelson D, Vates T, Thomas R. Complications from intrathecal steroid therapy in patients with multiple sclerosis. *Acta Neurol Scand* 1973;49:176–88.
- [14] Monteiro L, Leite I, Pinto A, Stocker A. Spontaneous thoracolumbar spinal cord infarction: report of six cases. *Acta Neurol Scand* 1992;86:563–6.
- [15] Connors J, Wojack J. *Interventional neuroradiology: strategies and practical techniques*. Philadelphia: WB Saunders Company, 1999.
- [16] Gillilan L. The arterial blood supply to the human spinal cord. *J Comp Neurol* 1958;110:75–103.
- [17] Tventen L. Spinal cord vascularity. *Acta Radiol* 1976;17:1–16.
- [18] Sandson T, Friedman J. Spinal cord infarction: report of 8 cases and a review of the literature. *Medicine* 1989;68:282–92.
- [19] Berman M, Feldman S, Alter M, Zilber N, Kahana E. Acute transverse myelitis: incidence and etiologic considerations. *Neurology* 1981;31:966–71.
- [20] McDermott DJ, Agre K, Brim M, et al. Chymodiactin in patients with herniated lumbar intervertebral disc(s). An open-label, multicenter study. *Spine* 1985;10(3):242–9.
- [21] Faber HK. Transverse myelitis from injection of penicillin. *Am J Dis Child* 1967;113(4):508–9.
- [22] Bacci R, Mathis I, Baduini G. Acute transverse myelopathy caused by penicillin injection. *Eur Neurol* 1975;13(6):555–9.
- [23] Stafford WW, Mena H, Piskun WS, Weir MR. Transverse myelitis from intraarterial penicillin. *Neurosurgery* 1984;15(4):552–6.
- [24] Weir MR, Fearnow RG. Transverse myelitis and penicillin. *Pediatrics* 1983;71(6):988.
- [25] Wilkins A, Estanol B. Transverse myelitis secondary to intramuscular administration of benzathine penicillin. *Arch Invest Med (Mex)* 1987;18(1):25–9.
- [26] Mehta A, Miyasu U, Baker R. Spinal cord infarctions: a clinicopathological study. *Neurol India* 1970;20:206–16.
- [27] Renfrew DL, Moore TE, Kathol MH, el-Khoury GY, Lemke JH, Walker CW. Correct placement of epidural steroid injections: fluoroscopic guidance and contrast administration. *Am J Neuroradiol* 1991;12(5):1003–7.
- [28] Furman M, O'Brien E, Zglszewski T. Incidence of intravascular penetration in transforaminal lumbosacral epidural steroid injections. *Spine* 2000;25:2628–32.