Case Report

Spinal Cord Injury from Fluoroscopically Guided Intercostal Blocks with Phenol

Narayan R. Kissoon, MD, Jonathan Graff-Radford, MD, James C. Watson, MD, and Ruple S. Laughlin, MD

From: Department of Neurology, Mayo Clinic, Rochester, MN

Address Correspondence: Ruple S. Laughlin, MD, Department of Neurology, Mayo Clinic 200 First Street SW, Rochester, Minnesota, 55905 E-mail: laughlin.ruple@mayo.edu

Disclaimer: There was no external funding in the preparation of this manuscript. Conflict of interest: Each author certifies that he or she, or a member of his or her immediate family, has no commercial association (i.e., consultancies, stock ownership, equity interest, patent/licensing arrangements, etc.) that might pose a conflict of interest in connection with the submitted manuscript.

Manuscript received: 10-02-2013
Revised manuscript received: 12-09-2013
Accepted for publication: 01-16-2014

Free full manuscript: www.painphysicianjournal.com

Background: Image guided intercostal blocks are commonly performed and considered relatively safe. Chemical denervation is commonly used in clinical practice for treatment of chronic non-cancer associated pain.

Objective: To report a case of spinal cord injury resulting from fluoroscopically guided intercostal blocks with phenol.

Study Design: Case report.

Setting: Inpatient hospital service.

Results/Case Report: A 53 year-old women was transferred from her local facility for acute onset of lower extremity paresis beginning shortly after right intercostal nerve injections of 2 mL of preservative-free phenol at the T7, 8, 9 levels. She had previous intercostal blocks for chronic right-sided mid thoracic/abdominal pain every 3 months for at least one year without sequelae. Within 20 minutes of the injection, she developed a sensation of right leg weakness and heaviness. Over several hours she developed worsening right leg weakness, and then left leg weakness, followed by urinary retention. Admission examination revealed severe right greater than left leg weakness, right lower extremity hyperesthesia to T10, absent lower extremity reflexes, and bilateral extensor plantar responses. Magnetic resonance imaging (MRI) of the entire spine demonstrated extensive T2/DWI hyperintensity in the central spinal cord from T1 to L1 with mild cord enlargement and enhancement at T7-9 (sites of injection). Extensive serum and cerebrospinal fluid (CSF) evaluation did not show any evidence of an infectious, inflammatory, or metabolic cause to her myelopathy. Repeat MRI of the entire spine demonstrated near complete resolution of the T2 signal abnormality. One month after presentation, despite radiographic improvement, the patient showed some clinical improvement, but remained walker dependent and with neurogenic bowel and bladder.

Limitations: This report describes a single case report.

Conclusion: This case offers several lessons for a pain specialist including 1) the potential for a neurologic catastrophe (spinal cord injury) from aqueous neurolytic intercostal blocks despite "safe" contrast spread; 2) potential mechanisms of neurogenic injury with intercostal blocks; 3) review of modifiable factors to decrease the risk of neurogenic injury; and 4) review of potential interventions (steroids, lumbar drain) to improve outcome in the setting of iatrogenic procedural related spinal cord injury.

Key words: Phenol, myelopathy, paraparesis, chemical denervation, neurolysis, intercostal block, epidural spread, chronic pain, complication, transverse myelitis

Pain Physician 2014; 17:E219-E224

Chronic pain affects one in 5 adults (1). Chronic pain practice guidelines support the use of cryoablation, thermal intradiscal procedures, or radiofrequency ablation in selected cases for treatment of refractory chronic pain but do not recommend treatment with chemical denervation in the routine treatment of non-cancer associated pain (2). Chemical neurolysis of the celiac plexus or splanchnic nerves

with phenol or alcohol is considered standard of care for refractory epigastric abdominal pain related to pancreatic cancer (3). Despite varying opinions on the appropriate scenario for the use of chemical neurolysis in chronic non-cancer associated pain, chemical denervation with phenol or alcohol is commonly used in clinical practice for chronic non-cancer pain (4-7).

Adverse neurological events associated with phenol injections have been reported, with case reports highlighting complications with subarachnoid, intrathecal, stellate ganglion, and intercostal injection sites (8-12). Spinal cord injury after phenol injection has been rarely reported. We report a case of phenol induced myelopathy with serial magnetic resonance imaging (MRI) findings and clinical follow-up.

Case Report

A 53 year-old right-handed woman with a past medical history of type 2 diabetes, atrial fibrillation with sick sinus syndrome status post pacemaker placement, and chronic right-sided mid thoracic/abdominal pain of presumed neuropathic origin (without identifiable cause despite a dermatomal distribution and extensive evaluation including a myelogram) was receiving intermittent intercostal neurolysis with phenol every 3 months for at least one year at an outside medical facility. She was transferred to our facility 2 days following the development of acute lower extremity paresis following intercostal neurolysis with phenol. In review of her outside records, all prior injections were performed using the same injectate.

Two days prior to transfer to our hospital, the patient underwent T7, T8, and T9 right intercostal nerve injections, each with 2 mL of 3% preservative-free phenol under fluoroscopic guidance. Review of outside procedure notes documented that the right T7, 8, and 9 ribs were contacted approximately 7 cm lateral to the spinous process with a 27 gauge 2.5 inch needle to gauge depth, which was then advanced off the inferior edge, and advanced slightly. Injection of contrast was reported to demonstrate spread along all 3 intercostal nerves without clear intravascular uptake or tracking into the paravertebral or epidural spaces. This was followed by injection of 2 mL of 0.5% bupivacaine with epinephrine and then by the injection of 2 mL of 3% preservative-free phenol at each level. A half mL of saline was then injected followed by another 2 mL of 0.5% bupivacaine. Following the procedure the tract was infiltrated with 1% lidocaine as the needle was withdrawn.

Twenty minutes following dismissal from the facility where she received the injection, the patient developed a sensation of right leg weakness and heaviness. Over several hours, she began to feel spasms in her toes bilaterally, followed by worsening bilateral lower extremity weakness. When she awoke in the middle of night to void, she found she was unable to move either leg, with the right leg being much worse than the left. She was also unable to urinate and felt constipated. She called for an ambulance and was taken to her local emergency department where it was found that she had urinary retention of one liter.

The patient was admitted to her local institution and underwent a thoracic spine computed tomography (CT) scan followed the next day by CT myelogram which were both normal. Cerebrospinal fluid (CSF) analysis revealed an elevated protein of 208 mg/dl, glucose 99 mg/dl, and 36 white blood cells (WBC) with neutrophilic predominance. CSF testing including Lyme polymerase chain reaction (PCR), venereal disease research laboratory (VDRL) test, cryptococcus antigen screen, cytomegalovirus PCR, enterovirus PCR, Epstein-Barr virus PCR, herpes simplex virus PCR, varicella zoster virus PCR, human T-lymphotrophic virus antigen screen, West Nile IgM and IgG, and paraneoplastic panel was negative.

The patient was transferred to our facility for further evaluation, management, and imaging. At admission, she had severe, diffuse right leg weakness and moderate left proximal with mild distal weakness. Hyperesthesia was present throughout the right lower extremity to the T10 level with intact sensation to light touch, pain, and proprioception. Lower extremity reflexes were diminished on the left and absent on the right with bilateral extensor plantar responses.

Serological evaluation showed a normal vitamin B12, methylmalonic acid, vitamin E, copper, zinc, angiotensin converting enzyme, serum protein electrophoresis, TSH, myeloperoxidase, proteinase 3, cyclic citrullinated peptide antibody, anti-nuclear antibody, extractable nuclear antibodies, neuromyelitis optica antibody, and anti-phospholipid antibodies. MRI of the cervical, thoracic, and lumbar spine revealed extensive abnormal T2/DWI hyperintensity in the central cord with associated mild cord enlargement from T1-L1 and enhancement of cord at T7-10 (Figs. 1 and 2). MRI interpretation raised concern for an inflammatory myelopathy or spinal cord infarct. Based on this interpretation, normal serological testing, a potentially inflammatory

appearing CSF, and a potential role for corticosteroids in treatment of acute spinal cord injury (13,14), we empirically treated our patient with intravenous methylprednisolone one gram daily for 5 days. Her neurological examination at dismissal to her local rehabilitation facility showed little improvement from her admission examination. She remained non-ambulatory with inability to bear weight on either leg and continued to have a neurogenic bowel and bladder. CSF phenol level was attempted from her original study done at the outside facility but there was not an adequate sample left for testing.

The patient was seen back in outpatient follow-up one month after hospital dismissal. At this visit, there was clear but mild improvement in the patient's strength with the right lower extremity demonstrating only antigravity strength proximally and moderate weakness distally. Only trace weakness remained in the left lower extremity. She was now able to ambulate, but required a walker. Her sensory exam was unchanged with persistent hyperesthesia to the umbilicus as well as persistent bowel and bladder dysfunction. Repeat MRI of the cervical, thoracic, and lumbar spine demonstrated marked interval improvement in the previously described extensive T2 signal abnormality and cord expansion with the follow-up study only showing mild asymmetrical T2 signal change on the right (Figs. 1 and 2).

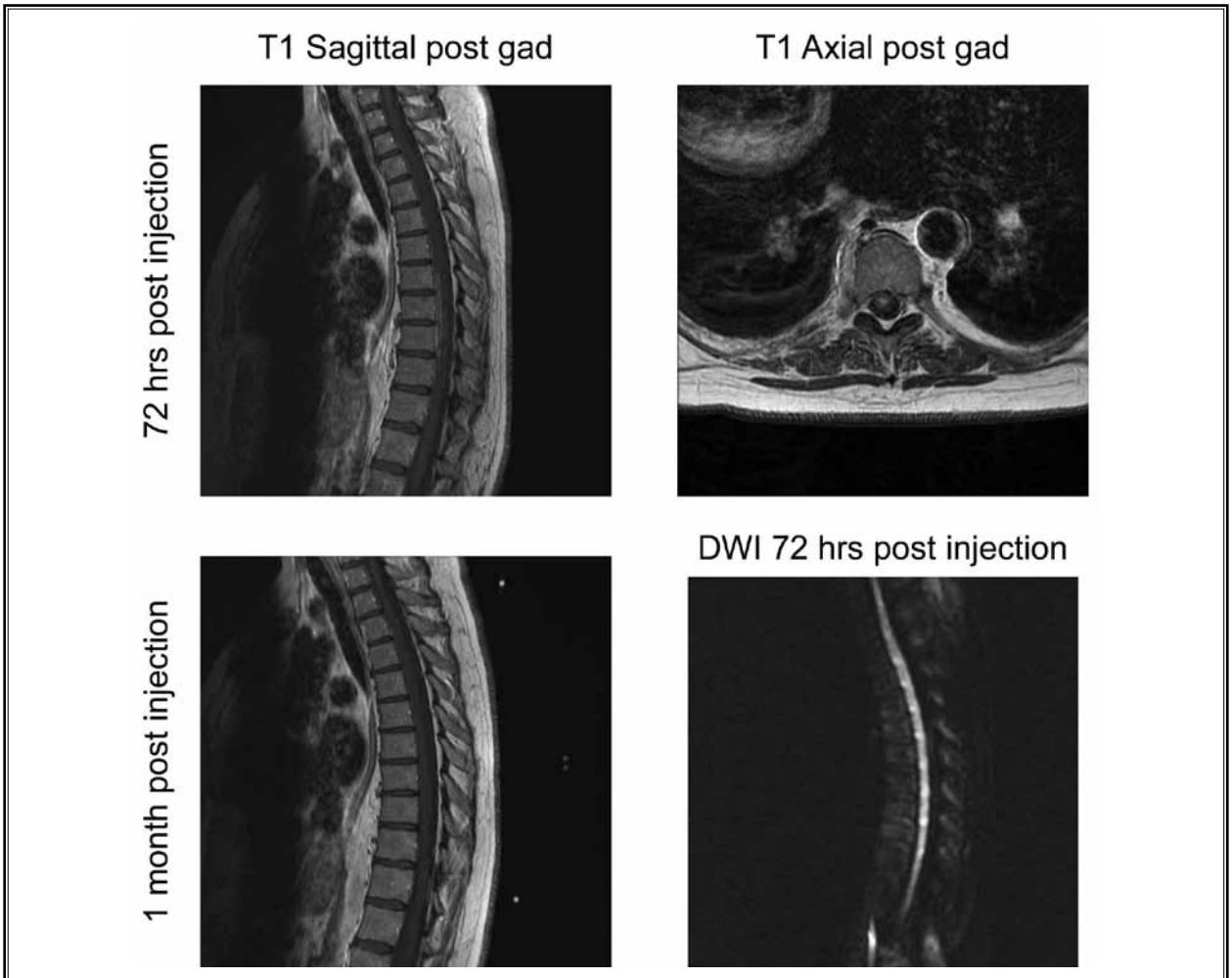

Fig. 1. *T1 weighted magnetic resonance images 72 hours following 3 2 mL injections of 3% preservative-free phenol into the right T7, T8, and T9 intercostal spaces had shown contrast enhancement at the levels of injection of the central cord with resolution of enhancement one month following the injection. Diffusion weighted imaging performed 72 hours following injection displayed extensive restricted diffusion throughout the entire thoracic cord.*

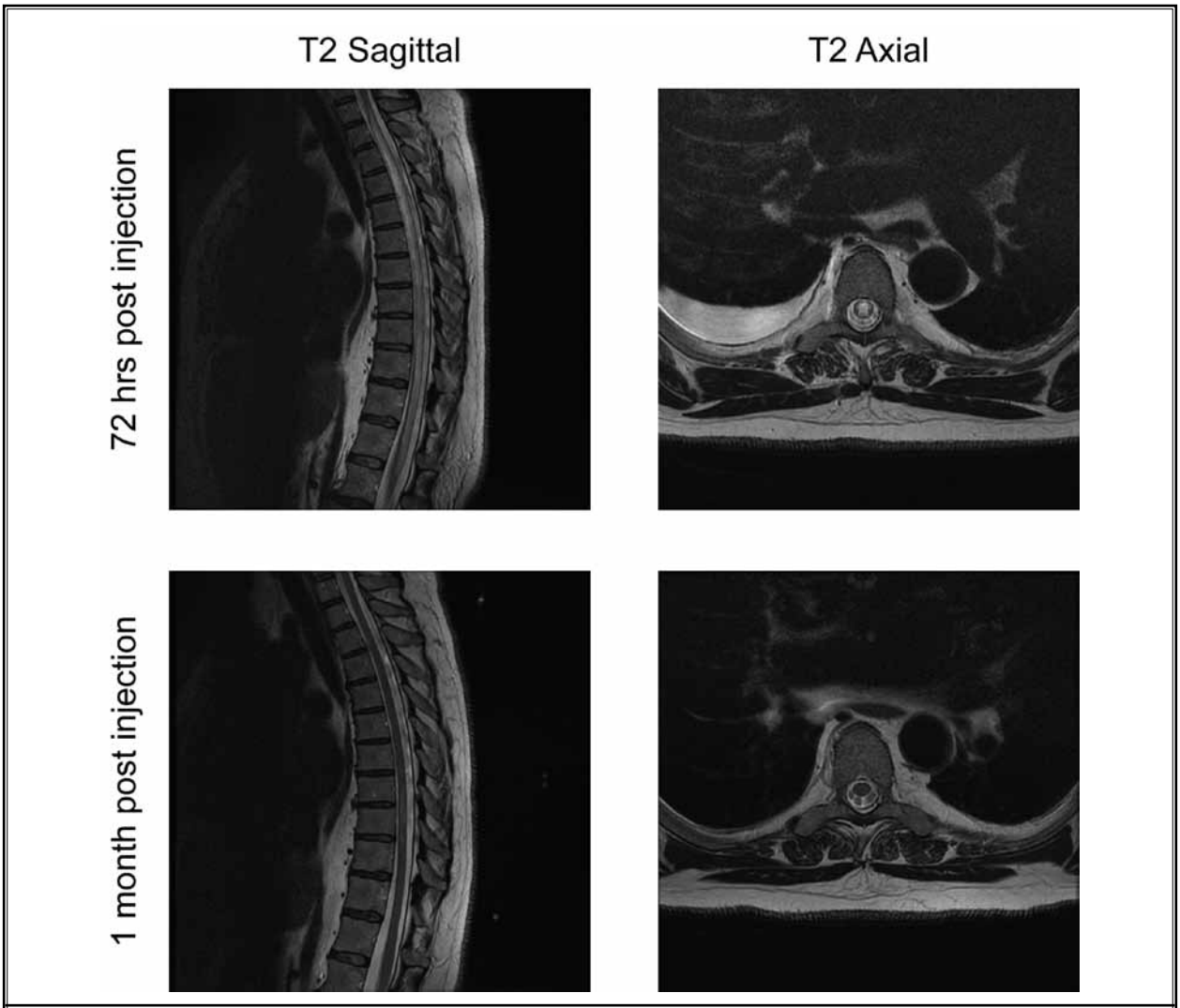

Fig. 2. *T2 weighted magnetic resonance images 72 hours following 3 2 mL injections of 3% preservative-free phenol into the right T7, T8, and T9 intercostal spaces demonstrated longitudinally extensive T2 hyperintensity of the central cord with improvement one month following the injection.*

Discussion

Phenol was initially utilized as an anesthetic option for severe pain in patients with incurable cancer before its usage expanded to other types of chronic pain (15). Aqueous phenol solutions for neurolysis provide effective pain relief (7). The mechanism of action of phenol is likely 2-fold. Acutely, phenol acts as a local anesthetic by inhibiting nerve conduction based on the size of the nerve fiber (16). The long-term anesthetic results are secondary to non-selective nerve destruction, which may (in part) explain some of the complications associated with phenol use such as reactive meningitis, arachnoiditis, paraplegia, or other unintended damage of nerve fibers (10,17). Severe systemic complications of phenol such as acute respiratory and renal failure can also occur (9). Autopsy specimens of phenol injection complications show cervical spinal cord demyelination with infarction thought to be secondary to direct neurolysis with demyelination and axonal loss as well as ischemic injury related to vascular thrombosis (10,12).

Spread of an anesthetic aqueous solution is a well-documented phenomenon which explains why single

nerve injections can provide anesthesia for multiple dermatomes with both large and small volumes of anesthetic (18,19). Imaging studies of intercostal anesthetic injection have documented lateral spread (along the intercostal space), paravertebral spread, spread to adjacent intercostal spaces via the paravertebral space (20), and both rostral and caudal epidural spread (18). Epidural spread is common once spread reaches the paravertebral space (21). Once phenol reaches the epidural space, small volumes of phenol in glycerin (2 mL – 4 mL) have been observed to spread on average of 12.2 vertebral segments on first injection and 8.3 vertebral segments in subsequent injections (22).

Complications from adverse central migration of injectate (local anesthetic or neurolytic) placed fluoroscopically after demonstration of "safe" contrast spread along the intercostal nerves is rare (23,24). A single case report documented complications of chemical denervation with onset of paraplegia minutes following intercostal injection of 7.5% aqueous phenol (11). Imaging studies showed diffusion of phenol from the intercostal injection site to the intervertebral foramen with the presence of phenol in the CSF, but did not demonstrate MRI evidence of spinal cord injury as we note in our case. Neither our patient nor the patient previously reported had clinical benefit despite acute administration of high dose intravenous methylprednisolone and both had continued and significant disability when seen in follow-up.

Prior histopathological autopsy reports of phenol neurolysis performed within the intrathecal space or at the root level demonstrate spinal cord infarction (10,12). Our patient, however, had mild clinical improvement suggesting inflammation of the spinal cord likely due to chemical irritation and not infarction as one would then not expect clinical improvement. More convincingly, the T2 signal abnormalities on spinal cord MRI were not in a single spinal vascular distribution (as would be expected in a spinal cord infarct) and the resolution of imaging abnormalities upon serial imaging were more consistent with direct nerve injury rather than ischemic infarct. We suspect that our patient's less severe outcome when compared to these other cases was due to a smaller relative phenol dose affecting the spinal cord as a result of the greater distance of anesthetic diffusion from her intercostal phenol nerve injection as opposed to an intrathecal injection.

Interestingly, chemical denervation with alcohol has been shown to have similar efficacy for treatment of spasticity and chronic pain without the reported complications of phenol (2,25). At phenol concentrations higher than 3%, phenol causes axonal degeneration and inflammation leading to nerve destruction (25-27). Animal models have shown that alcohol concentrations of 35% caused demyelination and concentrations of 50 – 100% caused wallerian degeneration with fibrosis (25,28). The higher concentrations of alcohol required for neurolysis provides a safety margin to minimize unwanted adverse events with relatively few reports of adverse effects following intramuscular and perineural alcohol injections as compared to phenol (25).

While our patient had improvements in her neurological deficits, she still had significant disability that may have been prevented or reduced with use of a more controlled lesioning method such as radiofrequency ablation. Perioperative lumbar spinal drainage has been widely practiced for thoracic aorta repair to decrease the risk of spinal cord ischemia by lowering intrathecal pressure thereby improving cord perfusion (29-31). One case report demonstrated improvement with lumbar drain in a spinal ischemic syndrome after aortic dissection (32). The literature supports CFS diversion 48 hours postoperatively in spinal cord ischemia associated with aortic repair (30). Since our patient presented 48 hours after symptom onset, lumbar drain placement was not a therapeutic option. The potential role of lumbar drain placement to improve outcomes in post interventional spinal cord injury could be an area of future study since these patients did not respond to high dose steroids. If effective, intervention for iatrogenic spinal cord injury needs to be instituted early. This requires early identification of neurological deficits. Our case suggests that extended observation may be appropriate following phenol neurolysis when performed near the neuroaxis.

Conclusion

Although rare, neurological damage from the use of phenol as an agent for chemical denervation may be significant. Phenol induced myelopathy can be avoided by recognition of the possibility of phenol related injury due to diffusion of phenol to adjacent structures and the potential for paravertebral and epidural spread of phenol with intercostal nerve block. Consideration of non-aqueous neurolysis methods for the treatment of chronic pain syndromes is warranted due to the potentially devastating complications from the use of phenol as highlighted by our case.

References

1. Katz WA. Musculoskeletal pain and its socioeconomic implications. *Clin Rheumatol* 2002; 21:S2-S4.
2. Practice guidelines for chronic pain management: An updated report by the American Society of Anesthesiologists Task Force on Chronic Pain Management and the American Society of Regional Anesthesia and Pain Medicine. *Anesthesiology* 2010; 112:810-833.
3. Nagels W, Pease N, Bekkering G, Cools F, Dobbels P. Celiac plexus neurolysis for abdominal cancer pain: A systematic review. *Pain Med* 2013; 14:1140-1163.
4. Manjunath PS, Jayalakshmi TS, Dureja GP, Prevost AT. Management of lower limb complex regional pain syndrome type 1: An evaluation of percutaneous radiofrequency thermal lumbar sympathectomy versus phenol lumbar sympathetic neurolysis—a pilot study. *Anesth Analg* 2008; 106:647-649, table of contents.
5. Pollitt CI, Salota V, Leschinskiy D. Chemical neurolysis of the superior hypogastric plexus for chronic non-cancer pelvic pain. *Int J Gynaecol Obstet* 2011; 114:160-161.
6. van Eijs F, Stanton-Hicks M, Van Zundert J, Faber CG, Lubenow TR, Mekhail N, van Kleef M, Huygen F. Evidence-based interventional pain medicine according to clinical diagnoses. 16. Complex regional pain syndrome. *Pain Pract* 2011; 11:70-87.
7. Weksler N, Klein M, Gurevitch B, Rozentsveig V, Rudich Z, Brill S, Lottan M. Phenol neurolysis for severe chronic nonmalignant pain: Is the old also obsolete? *Pain Med* 2007; 8:332-337.
8. Garcia E, Patt RB, Ginsberg LE. Effective but near disastrous subarachnoid neurolysis for mechanical root pain in a paraplegic cancer patient. *Clin J Pain* 1996; 12:243-249.
9. Gupta S, Ashrith G, Chandra D, Gupta AK, Finkel KW, Guntupalli JS. Acute phenol poisoning: A life-threatening hazard of chronic pain relief. *Clin Toxicol (Phila)* 2008; 46:250-253.
10. Hansebout RR, Cosgrove JB. Effects of intrathecal phenol in man. A histological study. *Neurology* 1966; 16:277-282.
11. Kowalewski R, Schurch B, Hodler J, Borgeat A. Persistent paraplegia after an aqueous 7.5% phenol solution to the anterior motor root for intercostal neurolysis: A case report. *Arch Phys Med Rehabil* 2002; 83:283-285.
12. Superville-Sovak B, Rasminsky M, Finlayson MH. Complications of phenol neuroloysis. *Arch Neurol* 1975; 32:226-228.
13. Bracken MB. Steroids for acute spinal cord injury. *Cochrane Database Syst Rev* 2012; 1:CD001046.
14. Bracken MB, Shepard MJ, Holford TR, Leo-Summers L, Aldrich EF, Fazl M, Fehlings M, Herr, DL, Hitchon PW, Marshall LF, Nockels RP, Pascale V, Perot PL, Piepmeier J, Sonntag VK, Wagner F, Wilberger JE, Winn HR, Young W. Administration of methylprednisolone for 24 or 48 hours or tirilazad mesylate for 48 hours in the treatment of acute spinal cord injury. Results of the Third National Acute Spinal Cord Injury Randomized Controlled Trial. National Acute Spinal Cord Injury Study. *JAMA* 1997; 277:1597-1604.
15. Mark FH, White JC, Zervas NT, Ervin FR, Richardson EP. Intrathecal use of phenol for the relief of chronic severe pain. *N Engl J Med* 1962; 267:589-593.
16. Nathan PW, Sears TA. Effects of phenol on nervous conduction. *J Physiol* 1960; 150:565-580.
17. Nathan PW, Scott TG. Intrathecal phenol for intractable pain: Safety and dangers of the method. *Lancet* 1958; 1:76-80.
18. Middaugh RE, Menk EJ, Reynolds WJ, Bauman JM, Cawthon MA, Hartshorne MF. Epidural block using large volumes of local anesthetic solution for intercostal nerve block. *Anesthesiology* 1985; 63:214-216.
19. Purcell-Jones G, Pither CE, Justins DM. Paravertebral somatic nerve block: A clinical, radiographic, and computed tomographic study in chronic pain patients. *Anesth Analg* 1989; 68:32-39.
20. Moorthy SS, Dierdorf SF, Yaw PB. Influence of volume on the spread of local anesthetic-methylene blue solution after injection for intercostal block. *Anesth Analg* 1992; 75:389-391.
21. Cowie B, McGlade D, Ivanusic J, Barrington MJ. Ultrasound-guided thoracic paravertebral blockade: A cadaveric study. *Anesth Analg* 2010; 110:1735-1739.
22. Salmon JB, Finch PM, Lovegrove FT, Warwick A. Mapping the spread of epidural phenol in cancer pain patients by radionuclide admixture and epidural scintigraphy. *Clin J Pain* 1992; 8:18-22.
23. Benumof JL, Semenza J. Total spinal anesthesia following intrathoracic intercostal nerve blocks. *Anesthesiology* 1975; 43:124-125.
24. Moore DC. Efocaine; complications following its use. *West J Surg Obstet Gynecol* 1953; 61:635-638.
25. Kocabas H, Salli A, Demir AH, Ozerbil OM. Comparison of phenol and alcohol neurolysis of tibial nerve motor branches to the gastrocnemius muscle for treatment of spastic foot after stroke: A randomized controlled pilot study. *Eur J Phys Rehabil Med* 2010; 46:5-10.
26. Gracies JM, Elovic E, McGuire J, Simpson DM. Traditional pharmacological treatments for spasticity. Part I: Local treatments. *Muscle Nerve Suppl* 1997; 6:S61-S91.
27. Carda S, Molteni F. Selective neuromuscular blocks and chemoneurolysis in the localized treatment of spasticity. *Eura Medicophys* 2004; 40:123-130.
28. Kong KH, Chua KS. Neurolysis of the musculocutaneous nerve with alcohol to treat poststroke elbow flexor spasticity. *Arch Phys Med Rehabil* 1999; 80:1234-1236.
29. Khan SN, Stansby G. Cerebrospinal fluid drainage for thoracic and thoracoabdominal aortic aneurysm surgery. *Cochrane Database Syst Rev* 2004; 10:CD003635.
30. Ullery BW, Cheung AT, Fairman RM, Jackson BM, Woo EY, Bavaria J, Pochettino A, Wang GJ. Risk factors, outcomes, and clinical manifestations of spinal cord ischemia following thoracic endovascular aortic repair. *J Vasc Surg* 2011; 54:677-684.
31. Keith CJ, Jr., Passman MA, Carignan MJ, Parmar GM, Nagre SB, Patterson MA, Taylor SM, Jordan WD. Protocol implementation of selective postoperative lumbar spinal drainage after thoracic aortic endograft. *J Vasc Surg* 2012; 55:1-8; discussion 8.
32. Blacker DJ, Wijdicks EF, Ramakrishna G. Resolution of severe paraplegia due to aortic dissection after CSF drainage. *Neurology* 2003; 61:142-143.