# Cervical Transforaminal Epidural Injection in the Management of a Stinger

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## INTRODUCTION

A "stinger," otherwise known as a "burner," is a transient, reversible peripheral nerve injury of the upper limb caused by injury to the cervical spine and shoulder. This injury usually occurs during participation in contact sports [1-3]. Stingers are considered to be underreported by athletes and are most common in American-style football, hockey, gymnastics, and wrestling [4]. In a study of collegiate American-style football players, Levitz et al [5] reported that stingers occur in 50%-65% of these athletes during the course of their career; they also reported high rates of recurrence. Mechanisms of injury include traction, compression, and direct blows [4,6-9]. Traction injuries result from ipsilateral shoulder depression and contralateral neck flexion, effectively resulting in traction on the nerve root and/or brachial plexus.

At the high school level, football players are more likely to have traction injuries. Conversely, compression injuries are more likely sustained at the collegiate and professional levels [10]. Compression injuries of the nerve root stem from a combination of forced hyperextension with ipsilateral rotation and lateral flexion leading to transient neuroforaminal narrowing. Finally, a direct blow to the supraclavicular region (Erb point), where the brachial plexus is most superficial, can result in direct trauma to the plexus. Most stingers, according to the Seddon classification scheme, are characterized as first- or second-degree peripheral nerve injuries, which refer to neurapraxia (demyelination) or axonotmesis (axonal loss), respectively. A third-degree injury, or neurotmesis (complete nerve transection), is not considered within the spectrum of this disorder [11,12].

Because tackling and blocking are the two most common mechanisms of stingers, defensive backs and offensive linemen are most susceptible [10]. Symptoms appear immediately after contact [1] and generally affect the upper trunk of the brachial plexus or C5/C6 nerve roots [6-8,13-15]. The primary symptom is burning pain referring to the upper limb, which may be accompanied by weakness, numbness, and paresthesias [1,16]. Symptoms typically do not last more than 24 hours [2]. A wide range of clinical courses have been described after a stinger injury, ranging from full recovery in seconds or hours to the development of a chronic syndrome [1,5]. The distribution of weakness is dependent on the source of the nerve injury, but whether it is the root (C5 or C6) or upper trunk of the brachial plexus, the most common pattern of muscle weakness involves the deltoid, supraspinatus, and/or infraspinatus for several weeks after the injury [17].

Conflicting views exist regarding the location of the neurological lesion [13]. Some authors who included an electrodiagnostic evaluation in their studies reported a greater incidence of brachial plexus involvement [8,14,15,18]. However, Slipman et al [19] demonstrated that diagnostic cervical selective nerve root blocks frequently provoke symptoms outside the classic dermatomal distribution in nonathletes. Thus it is plausible that injury to a single nerve root can produce a more circumferential pattern of symptoms in the upper limb [20].

Several factors suggest that most stingers are caused by cervical nerve root injury [5,21]. It has been shown that there is a statistically significant 10%-13% decrease in neuroforaminal diameter when the neck is positioned in 20°-30° of extension, as is common during a stinger injury [22]. Anatomically, the cervical nerve roots appear to be more vulnerable to injury than the brachial plexus because of their orientation and

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structure. Furthermore, the brachial plexus is surrounded by more perineural tissue compared with the nerve roots and is more resilient to injury [9].

Despite increased efforts to better identify and predict stingers, research regarding treatment options is lacking. Epidural injections in the management of a stinger have not been studied previously. In the following case, we describe a collegiate American football player who sustained a C5 nerve root/stinger injury and experienced prompt and complete resolution after a cervical transforaminal epidural steroid injection.

### **CASE PRESENTATION**

The patient was a 21-year-old, right-hand dominant starting center for a Division I collegiate football team. In his 3 collegiate seasons, he reported an average of 3-5 stingers per year without side predilection. Previous episodes lasted minutes to a few hours and never with noticeable weakness. He presented to our clinic with 5 days of persistent left-sided neck and shoulder pain associated with paresthesias and upper limb weakness. During a game, symptoms began immediately after he was struck on the right side of his helmet, which induced left lateral flexion and extension of his neck. The team physician diagnosed the stinger and treated him with rest and a methylprednisolone dose pack on postinjury day 1, but his symptoms persisted. He denied any loss of consciousness, headaches, or symptoms involving other extremities associated with the injury.

Upon physical examination, motor testing of the left shoulder abduction, external rotation, and elbow flexion was 4/5 (Medical Research Council scale). Sensation to light touch and pinprick revealed deficits in the left lateral shoulder. All other upper extremity myotomes and dermatomes were intact. The biceps muscle reflex was slightly diminished compared with the right side. Incorporation of the Spurling maneuver to the left reproduced paresthesias to the left lateral shoulder. He had a symmetric gait pattern with a narrow base of support and did not demonstrate upper motor neuron signs. Cervical spine magnetic resonance imaging (MRI) showed a small left paracentral disk protrusion at C7-T1 but without significant central or neuroforaminal narrowing. No disk abnormalities were seen at the C4-5 level corresponding to the C5 nerve root. Foraminal stenosis is noted, right greater than left, at the C4-5 level primarily due to uncovertebral hypertrophy and disk protrusion (Figure 1).

The patient reported a compression mechanism of injury that most likely resulted in an injury to the nerve root and not the brachial plexus. This history, along with his symptoms and physical examination, were highly suggestive of a left C5 nerve root injury. Because his shoulder pain, paresthesias, and weakness had not improved and prompt return to play was desired, a cervical transforaminal epidural steroid injection was offered. The risks and benefits of the procedure,



Figure 1. (A) Cervical spine T2-weighted sagittal magnetic resonance imaging scan. (B) Cervical spine T2-weighted axial magnetic resonance imaging scan at the C4-5 level.

including serious complications, were discussed with the patient. On the following day, a fluoroscopic-guided left C5 transforaminal epidural steroid injection was performed with contrast under real-time fluoroscopy and digital subtraction angiography. After a negative lidocaine test dose (1 mL of 1% preservative-free lidocaine), 10 mg of dexamethasone was injected (Figure 2). During instillation of the injectate, the patient experienced reproduction of his typical paresthesias followed by concordant pain relief in the recovery room.



Figure 2. Anteroposterior view of left C5 transforaminal epidural steroid injection with contrast outlining the proximal and distal nerve root (arrows).

Since the injection, the patient has had lasting resolution of his pain. He reported a dramatic improvement in strength 3 days after the procedure when an electrodiagnostic examination (ordered by the team physician before the consultation) was performed on postinjury day 10. Physical examination revealed only trace weakness with shoulder abduction and external rotation. The electrodiagnostic study revealed evidence of an acute C5 radiculopathy on the basis of 1+ spontaneous activity (fibrillation potentials) in the left deltoid, infraspinatus, and rhomboid muscles, with increased insertional activity of cervical paraspinal muscles. The left deltoid and infraspinatus were noted to have mild decreased recruitment.

Two weeks after the injury, the patient was cleared by his team physician to return to play. The results of a 2-week postinjection follow-up examination revealed an asymptomatic patient with normal muscle strength. We discussed his increased risk for future episodes and greater potential for a sustained motor deficit because of previous stingers. Although imaging demonstrated a normal cervical lordosis, he was counseled on the importance of maintaining optimal cervical posture and appropriate strength of the cervical, thoracic, scapular, and core stabilizers.

The rehabilitation program was overseen by the team physician, and re-education regarding proper blocking techniques was directed by coaching staff. Although cervical rolls have not demonstrated a reduction in the incidence of stingers [10], the team implemented their use. Despite optimizing football technique and participating in a comprehensive rehabilitation program, the patient sustained a total of 8 stinger episodes during the course of 2 seasons. However, none of these episodes lasted longer than a few hours and were devoid of weakness or side predilection.

## DISCUSSION

Corticosteroids are used to treat radiculopathies and compressive neuropathies such as carpal tunnel syndrome. Methylprednisolone-soaked Gelfoam is routinely applied to the facial nerve during acoustic neuroma resection to decrease neural edema and improve postoperative nerve function [23]. Animal models support the notion that phospholipase A2, a key enzyme in proinflammatory states, is involved in the early process of myelin breakdown and Wallerian degeneration after nerve injury. By inhibiting this enzyme and lipid peroxidation, it is believed that steroids decrease inflammation, improve regional blood flow, and become neuroprotective [24].

The findings on the electromyogram (EMG) suggest a component of neurapraxia and/or axonotmesis. This combination is a likely mechanism of nerve root injury in radiculopathy cases [25]. The EMG was performed at the earliest point when spontaneous activity would be present, and therefore they may not have been fully expressed. An EMG was considered before the procedure, but it only would have demonstrated decreased motor unit recruitment. Nerve conduction studies could be used to help identify an upper trunk plexopathy (which was normal in our case), but like spontaneous activity, it would have been too early to be identified before the procedure. Dexamethasone may have allowed for faster recovery of the neurapraxic injury, perhaps by reversing nerve root ischemia. The reported half-life of dexamethasone is 36-54 hours. On the basis of the patient's rapid recovery 3 days after the injection, we believe his injury was mainly neurapraxic in nature because this pattern of recovery is not expected with axonotmesis. However, the presence of fibrillation potentials on EMG suggests at least partial axonotmesis.

An anesthetic test dose before the corticosteroid injection is used to identify potentially dangerous vascular entry of medications undetected by other methods [26]. The test dose may be considered positive if the following occurs: agitation or other sudden central nervous system change; gross motor deficits and/or paresthesias in the trunk, legs, or contralateral limb; or systemic symptoms of anesthetic toxicity, including cardiac arrhythmia, perioral numbness, metallic taste, dizziness, and/or ringing in the ear [27].

The lidocaine test dose used before the corticosteroid was injected also may have had a benefit. The authors of one study reported that selective nerve root injections with anesthetic alone may be effective in avoiding operative intervention in patients with radicular pain, although they concluded that the combination of a corticosteroid and an anesthetic is more effective than an anesthetic alone [28]. Other authors have shown that lidocaine may have anti-inflammatory effects [29]. Animal models have demonstrated that local anesthetics increase intraradicular blood flow, which may improve intraneural metabolism and reduce anti-inflammatory mediators [30]. Other factors that potentially lead to improvement also were considered, including previous courses of oral steroids and the natural course of stinger improvement despite treatment.

In this case, although the foraminal stenosis is not severe in the supine position, an argument can be made for the role of dynamic stenosis as a pathoanatomic explanation for nerve root injury. Our patient clearly had a C5 radiculopathy, although the exact location of injury along the nerve root remains unclear. As demonstrated in this case, transforaminal epidural injections typically spread along the path of the proximal and distal nerve root (Figure 2). For this reason, a transforaminal approach was used to deliver an injectate directly at the site of suspected pathology [31].

Although the patient had a positive outcome, treatment with cervical transforaminal epidural injections requires caution. Serious complications have been reported, including spinal cord and brain injuries [32-37]. The true incidence of these complications is unknown by prospective investigation, but they are rare [38,39]. The most often hypothesized mechanism is inadvertent intra-arterial injection of particulate corticosteroids with a resulting embolus and infarction [40]. In our case we injected dexamethasone, a nonparticulate corticosteroid, after a negative lidocaine test dose. To date, no serious complications have been reported with the use of nonparticulate corticosteroids during cervical transforaminal injections.

The patient elected to have the procedure because his main goal was prompt return to play. In highly competitive sports, careers can be made or lost in a short period. The decision to perform the injection was a considered one. Appropriate noninterventional treatments were performed first, and only then did further treatment ensue. To our knowledge, this is the first reported case of a stinger injury being treated with a cervical epidural injection. Further investigation is needed to substantiate the benefits of this treatment approach.

#### REFERENCES

- 1. Kuhlman GS, McKeag DB. The "burner": A common nerve injury in contact sports. Am Fam Physician 1999;60:2035-2042.
- **2.** Rihn JA, Anderson DT, Lamb K, et al. Cervical spine injuries in American football. Sports Med 2009;39:697-708.
- Castro FP Jr. Stingers, cervical cord neurapraxia, and stenosis. Clin Sports Med 2003;22:483-492.
- Sallis RE, Jones K, Knopp W. Burners: Offensive strategy for an underreported injury. Physician Sportsmed 1992;20:47-55.
- Levitz CL, Reilly PJ, Torg JS. The pathomechanics of chronic, recurrent cervical nerve root neurapraxia. The chronic burner syndrome. Am J Sports Med 1997;25:73-76.
- Hershman EB. Injuries to the brachial plexus. In: Torg JS, ed. Athletic Injuries to the Head, Neck, and Face. 2nd ed. St. Louis: Mosby; 1991, 345-350.
- Poindexter DP, Johnson EW. Football shoulder and neck injury: A study of the "stinger" [abstract]. Arch Phys Med Rehabil 1984;65:601-602.

- Di Benedetto M, Markey K. Electrodiagnostic localization of traumatic upper trunk brachial plexopathy [abstract]. Arch Phys Med Rehabil 1984;65:15-17.
- **9.** Weinstein SM. Assessment and rehabilitation of the athlete with a "stinger." A model for the management of noncatastrophic athletic cervical spine injury. Clin Sports Med 1998;17:127-135.
- **10.** Weinstein S. Stingers: Understanding the mechanism, diagnosis, treatment, and prevention. In: Akuthota V, Herring S, eds. Nerve and Vascular Injuries in Sports Medicine. New York, NY: Springer; 2009, 127-138.
- **11.** Hershman EB. Brachial plexus injuries [abstract]. Clin Sports Med 1990;9:311-329.
- Warren RF. Neurologic injuries in football. In: Jordan BD, Tsairis P, Warren RF, eds. Sports Neurology. Rockville, MD: Aspen; 1989, 235-237.
- Standaert CJ, Herring SA. Expert opinion and controversies in musculoskeletal and sports medicine: Stingers. Arch Phys Med Rehabil 2009; 90:402-406.
- **14.** Robertson WC Jr, Eichman PL, Clancy WG. Upper trunk brachial plexopathy in football players [abstract]. JAMA 1979;241:1480-1482.
- Markey KL, Di Benedetto M, Curl WW. Upper trunk brachial plexopathy. The stinger syndrome [abstract]. Am J Sports Med 1993;21:650-655.
- **16.** Presciutti SM, DeLuca P, Marchetto P, et al. Mean subaxial space available for the cord index as a novel method of measuring cervical spine geometry to predict the chronic stinger syndrome in American football players. J Neurosurg Spine 2009;11:264-271.
- Shannon B, Klimkiewicz JJ. Cervical burners in the athlete. Clin Sports Med 2002;21:29-35.
- Clancy WG Jr, Brand RL, Bergfield JA. Upper trunk brachial plexus injuries in contact sports. Am J Sports Med 1977;5:209-216.
- **19.** Slipman CW, Plastaras CT, Palmitier RA, et al. Symptom provocation of fluoroscopically guided cervical nerve root stimulation: Are dynatomal maps identical to dermatomal maps? Spine 1998;23:2235-2242.
- **20.** Kelly JD, Aliquo D, Sitler MR, et al. Association of burners with cervical canal and foraminal stenosis. Am J Sports Med 2000;28:214-217.
- **21.** Meyer SA, Schulte KR, Callaghan JJ, et al. Cervical spinal stenosis and stingers in collegiate football players. Am J Sports Med 1994;22:158-166.
- **22.** Yoo JU, Zou D, Edwards WT, et al. Effect of cervical spine motion on the neuroforaminal dimensions of human cervical spine. Spine 1992; 17:1131-1136.
- Galloway III EB, Jensen RL, Dailey AT, et al. Role of topical steroids in reducing dysfunction after nerve injury. Laryngoscope 2000;110: 1907-1910.
- **24.** Lee HM, Weinstein JN, Meller ST, et al. The role of steroids and their effects on phospholipase A2 (an animal model of radiculopathy). Spine 1998;23:1191-1196.
- 25. Kimura J. Electrodiagnosis in Diseases of Nerve and Muscle. 3rd ed. Oxford: Oxford University Press; 2001.
- Smuck M, Leung D. Inadvertant injection of a cervical radicular artery using an atraumatic pencil-point needle. Spine J 2011;36:E220-E223.
- 27. Smuck M, Maxwell MD, Kennedy D, Rittenberg JD, Lansberg MG, Plastaras CT. Utility of the anesthetic test dose to avoid catastrophic injury during cervical transforaminal epidural injections. Spine J 2010; 10:857-864.
- **28.** Riew KD, Yin Y, Gilula L, et al. The effect of nerve-root injections on the need for operative treatment of lumbar radicular pain. A prospective, randomized, controlled, double blinded study. J Bone Joint Surg Am 2000;82-A:1589-1593.
- **29.** Shoji Y. Effects of lidocaine on nucleus pulposus-induced nerve root injury: A neurophysiologic and histologic study of pig cauda equine. Spine 1998;23:2383-2389.
- **30.** Yakubi S. Nerve root infiltration and sympathetic nerve block. An experimental study of intra-radicular blood flow. Spine 1995;20:901-906.

- Rathmell J. Atlas of Image-Guided Intervention in Regional Anesthesia and Pain Medicine. Philadelphia, PA: Lippincott and Williams & Wilkins; 2006.
- **32.** Brouwers PJ, Kottink EJ, Simon MA, Prevo RL. A cervical anterior spinal artery syndrome after diagnostic blockade of the right C6-nerve root. Pain 2001;91:397-399.
- **33.** Ziai WC, Ardelt AA, Llinas RH. Brainstem stroke following uncomplicated cervical epidural steroid injection. Arch Neurol 2006;63:1643-1646.
- **34.** Tiso RL, Cutler T, Catania JA, Whalen K. Adverse central nervous system sequelae after selective transforaminal block: The role of corticosteroids. Spine J 2004;4:468-474.
- **35.** Suresh S, Berman J, Connel DA. Cerebellar and brainstem infarction as a complication of CT-guided transforaminal cervical nerve root block. Skeletal Radiol 2007;36:449-452.

- **36.** Beckman WA, Mendez RJ, Paine GF, Mazzilli MA. Cerebellar herniation after cervical transforaminal epidural injection. Reg Anesth Pain Med 2006;31:282-285.
- **37.** Ludwig MA, Burns SP. Spinal cord infarction following cervical transforaminal epidural injection: A case report. Spine 2005;30: E266-E268.
- Ma DJ, Gilula LA, Riew KD. Complications of fluoroscopically guided extraforaminal cervical nerve blocks. An analysis of 1036 injections. J Bone Joint Surg Am 2005;87:1025-1030.
- **39.** Derby R, Lee S-H, Kim B-J, Chen Y, Seo KS. Complications following cervical epidural steroid injections by expert interventionalists in 2003. Pain Physician 2004;7:445-449.
- **40.** Derby R, Lee S, Date E, Lee J, Lee C. Size and aggregation of corticosteroids used for epidural injections. Pain Med 2008;9:227-234.