Brachial plexopathy/nerve root avulsion in a football player: the role of electrodiagnostics.

Joseph H Feinberg

Jeffrey Radecki MD
Lehigh Valley Health Network, jeffrey.radecki@lvhn.org

Scott W Wolfe

Helene L Strauss

Douglas N Mintz

Published In/Presented At

Feinberg JH, Radecki J, Wolfe SW, Strauss HL, Mintz DN. Brachial plexopathy/nerve root avulsion in a football player: the role of electrodiagnostics. HSS J. 2008 Feb;4(1):87-95. doi: 10.1007/s11420-007-9064-1. Epub 2008 Jan 4.

HSSJ (2008) 4: 87–95
DOI 10.1007/s11420-007-9064-1

ELECTRODIAGNOSTIC CORNER

Brachial Plexopathy/Nerve Root Avulsion in a Football Player: The Role of Electrodiagnostics

Joseph H. Feinberg, MD · Jeffrey Radecki, MD · Scott W. Wolfe, MD · Helene L. Strauss, BA · Douglas N. Mintz, MD

Received: 5 November 2007/Accepted: 6 November 2007/ Published online: 4 January 2008

Abstract Electromyography (EMG) studies are a useful tool in anatomical localization of peripheral nerve and brachial plexus injuries. They are especially helpful in distinguishing between brachial plexopathy and nerve root injuries where surgical intervention may be indicated. EMG can also assist in providing prognostic information after nerve injury as well as after nerve repair. In this case report, a football player presented with weakness in his right upper limb after a traction/traumatic injury to the right brachial plexus. EMG studies revealed evidence of both pre- and postganglionic injury to multiple cervical roots. The injury was substantial enough to cause nerve root avulsions involving the C6 and C7 levels. Surgical referral led to nerve grafts targeted at regaining function in shoulder abduction and elbow flexion. After surgery, the patient's progress was monitored utilizing EMG to assist in identifying true axonal regeneration.

Key words electromyography · EMG · electrodiagnostics · brachial plexopathy · nerve root avulsion · nerve graft

J. H. Feinberg, MD · J. Radecki, MD
Department of Physiatry,
Hospital for Special Surgery,
523 East 72nd Street, 2nd Floor, New York, NY 10021, USA

S. W. Wolfe, MD · H. L. Strauss, BA
Center for Hand and Upper Extremity Surgery,
Hospital for Special Surgery,
523 East 72nd Street, 4th Floor, New York, NY 10021, USA

D. N. Mintz, MD
Department of Radiology and Imaging,
Hospital for Special Surgery,
535 East 70th Street, New York, NY 10021, USA

J. H. Feinberg, MD (✉)
Hospital for Special Surgery,
East River Professional Building, 523 East 72nd Street, 2nd Floor,
New York, NY 10021, USA
e-mail: FeinbergJ@HSS.EDU

Introduction

The "burner", sometimes referred to as "stinger syndrome," is one of the most common injuries in sports medicine. Frequently underreported by athletes, stingers have been found to affect as many as 65% of college football athletes at least once during their collegiate career [1]. Symptoms typically include a burning or stinging pain that radiates down one of the upper limbs with or without associated parasthesias and weakness. In most cases, symptoms are transient and self-limiting. However, recurrences are common and subtle neurologic deficits may persist without being detected. Symptoms of burner syndrome that persist may indicate a more serious neurologic injury or chronic syndrome [2–4].

Burners are primarily felt to be either an upper cervical root (C5–C6) or upper brachial plexus injury. Controversy exists whether these injuries commonly involve the cervical roots, the brachial plexus, or a combination of the two. Developing an understanding of the anatomic localization would help in diagnosing these injuries. This type of injury results from nerve traction and/or direct compression of the involved nerve fibers depending on mechanism of injury [5, 6]. Rare, but catastrophic, sequelae of severe traumatic upper cervical nerve root injury may result in permanent nerve root injury, namely, nerve root avulsion [7, 8]. These injuries are usually limited to high-velocity impacts, such as motorcycle collisions, and have not yet been documented in the literature pertaining to football injury.

Case report

History

A 30-year-old semiprofessional football player presented, after a helmet-to-helmet collision while tackling, with persistent right arm weakness and parasthesias. At the time of the injury, the patient reported that the impact caused his head to laterally flex to the contralateral side. He then felt an

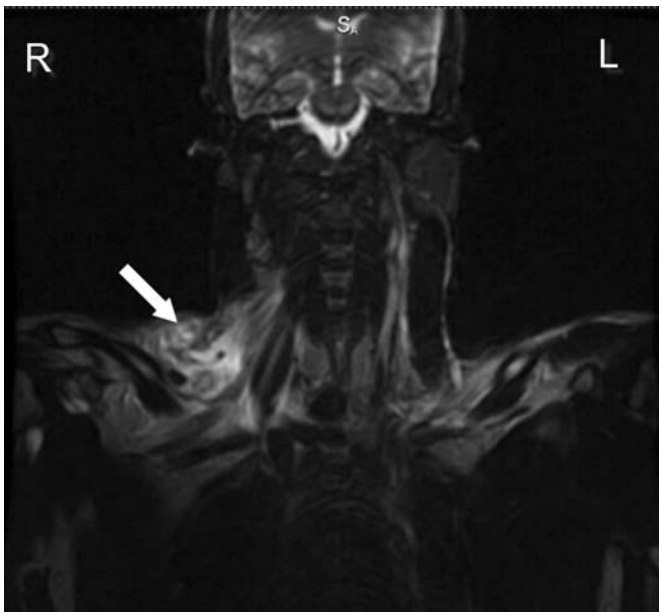

Fig. 1. Right supraclavicular hematoma. Low-resolution coronal T2, fat-suppressed MR image displays asymmetry of soft tissues with right-sided hematoma (arrow)

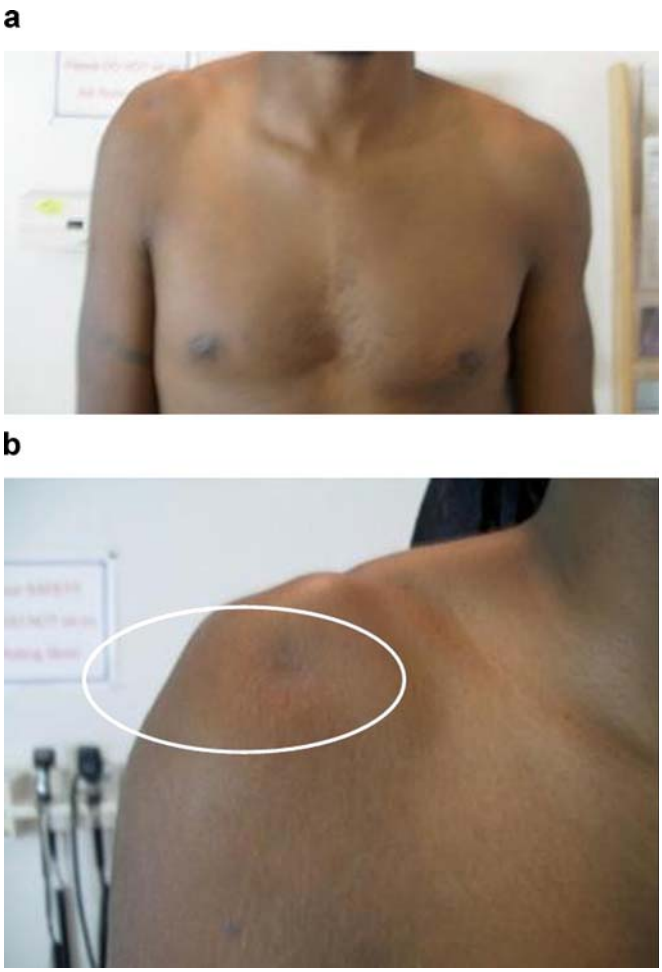

Fig. 2. Anterior view of patient—4 weeks post-injury. There is evidence of subacromial subluxation and deltoid atrophy, **a** and **b** (open circle)

immediate "frostbite" sensation in the first to third digits of his right hand and was unable to move much of his right arm. The "frostbite" sensation progressed to a severe, sharp right shoulder and neck pain, with a burning sensation encompassing the right arm and hand.

The patient reported no loss of consciousness, confusion, headache, other motor/sensory changes, or bowel/bladder changes at the time of injury or after. He was immediately transported to a local emergency room and hospitalized for 5 days for evaluation.

During hospitalization, a number of diagnostic studies were performed including a noncontrast computed tomography (CT) of head, CT of cervical spine, right shoulder x-ray, and magnetic resonance imaging (MRI) of cervical spine with and without contrast. Studies were virtually unrevealing, with the exception of a large infiltrative lesion, consistent with hematoma, localized at the right supraclavicular region compressing the brachial plexus (Fig. 1). Evaluation of the spinal cord was also normal, with the exception of a noted "limited" evaluation at the C5–C6 levels. The patient was discharged home.

At the time of presentation, the patient reported improved numbness in his right arm and hand but still limited by weakness. The patient also did provide a history of prior "stinger" that caused numbness in his right hand, which quickly resolved, but reportedly limited his strength and range of motion of the right shoulder.

Physical examination

On general examination, the patient presented as a well-developed athlete utilizing a sling to support the right upper extremity. An inspection revealed evidence of subacromial subluxation of the right shoulder and atrophy of the right deltoid (Fig. 2) and the right infraspinatus (Fig. 3).

The range of motion of the cervical spine was within normal limits without pain. On further examination of the right upper limb, strength testing demonstrated flaccid muscles in shoulder abduction and bicep flexion. There was substantial weakness in elbow extension with lesser

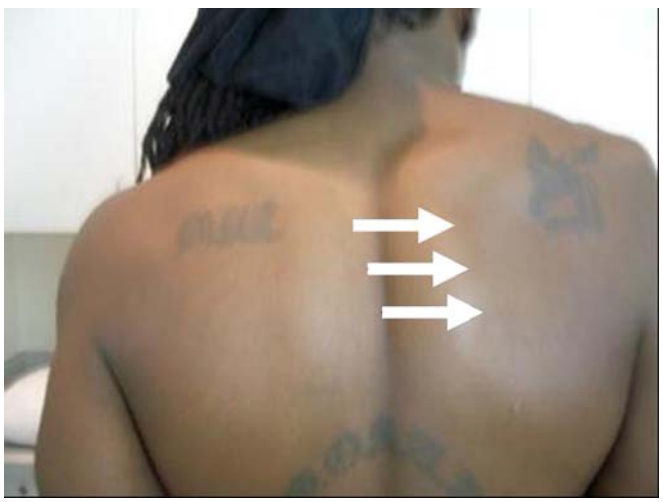

Fig. 3. Posterior view of patient—4 weeks post-injury. Exam reveals right infraspinatus atrophy (arrows)

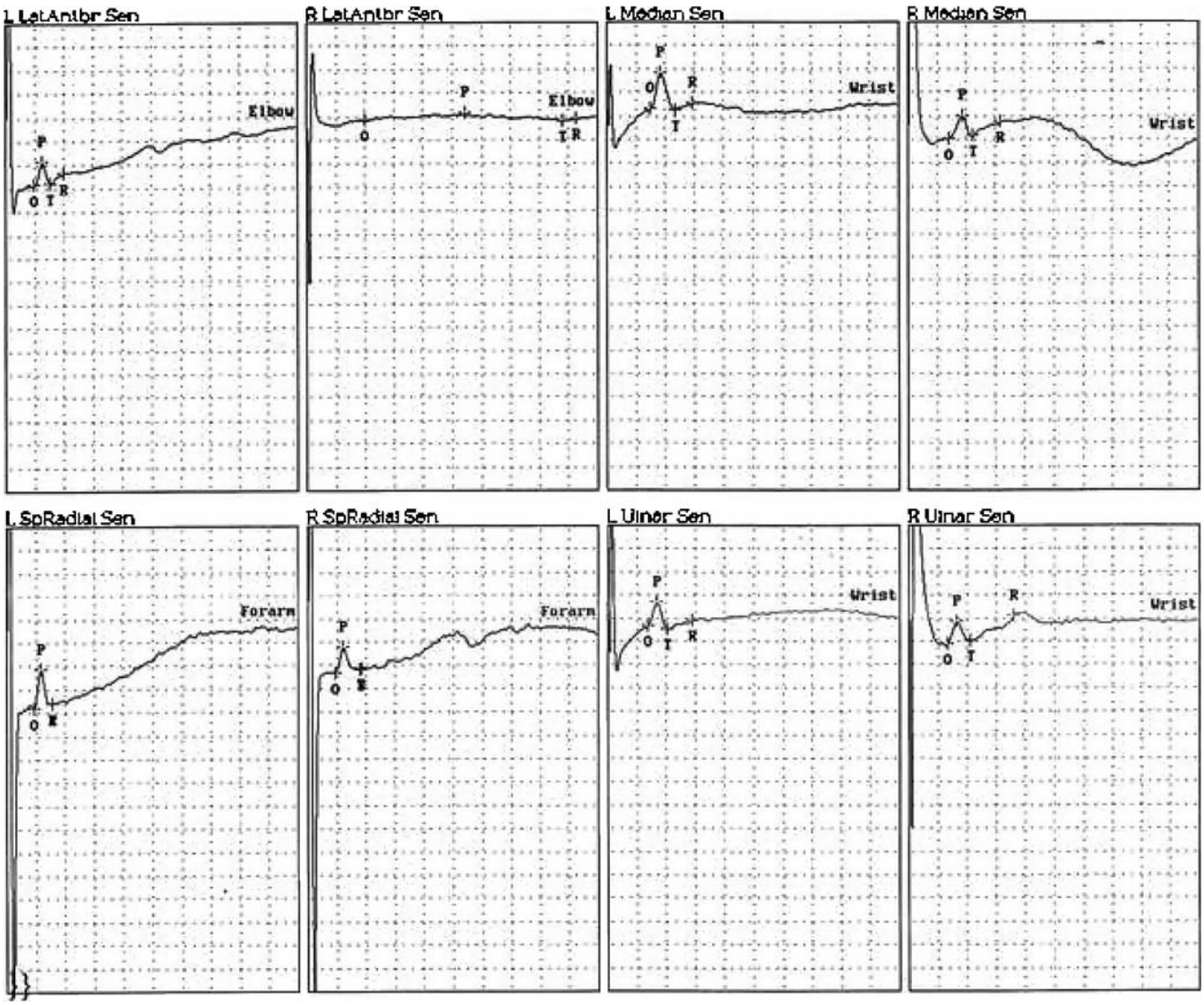

Fig. 4. Nerve conduction tracings of sensory nerves

Table 1 Nerve conduction values of sensory nerves

Nerve	Site	Onset Lat (ms)	Norm Onset	Peak Lat (ms)	Amplitude	Norm Amp	Duration (ms)	Seg Name	Distance (cm)	Velocity (m/s)	Norm Vel
L Lat/Antbr	Forarm				O-P (μV)		Neg.				
	Elbow	1.94		2.50	18.85		–	Elbow–Forarm	12.50	64.5	
R Lat/Antbr	Forarm				O-P (μV)		Neg.				
33.2°C	Elbow	NR		NR							
L Median	3rd Dig				O-P (μV)		Neg.				
	Wrist	3.00	<3.6	3.66	30.41		1.56				
R Median	3rd Dig				O-P (μV)		Neg.				
33.8°C	Wrist	2.84	<3.6	3.72	19.23		–				
L SpRadial	FWS				O-P (μV)		Neg.				
	Forarm	1.84		2.31	17.02		–	Forarm–FWS	11.00	59.7	
R SpRadial	FWS				O-P (μV)		Neg.				
33.8°C	Forarm	1.91		2.41	10.89		–	Forarm-FWS	11.50	60.3	
L Ulnar	5th Dig				O-P (μV)		Neg.				
	Wrist	2.72	<3.4	3.34	20.43		1.16	Wrist–5thDig	–	–	
R Ulnar	5th Dig				O-P (μV)		Neg.				
33.7°c	Wrist	2.66	<3.4	3.28	19.49		–	Wrist-5thDig	–	–	

Table 2 Electromyographic needle examination

Side	Muscle	Nerve	Root	INS	FIBS	PSW	FAS	AMP	DUR	CONFIGURATION	PAT	REC INT
R	1st Dorint	Ulnar	C8-T1	Nml	0	0	0	Nml	Nml	Di/Tri	Phasic	Full	Normal
R	AP8	Median	C8-T1	Nml	0	0	0	Nml	Nml	Di/Tri	Phasic	Full	Normal
R	Bicep	Musc	C5–6	Nml	2+	2+	0	Non	None	None		None	None
R	Brachialiss	Musc	C5–6	Nml	3+	3+	0	Non	None	None		None	None
R	Cervpara Low	Rami	C6–7	Nml	3+	3+	0	Non	None	None		None	None
R	Cervpara Up	Rami	C5–6	Nml	3+	3+	0	Non	None	None		None	None
R	Deltoid	Axilla	C5–6	Nml	3+	3+	0	Non	None	None		None	None
R	ExtCarRad	Radial	C7–8	Nml	2+	2+	0	Nml	Nml	Di/Tri	Phasic	Dec	Normal
R	ExtDigCom	Radial	C7–8	Nml	1+	2+	0	Nml	Nml	Di/Tri	Phasic	Dec	Normal
R	ExtIndPro	Radial	C7–8	Nml	1+	2+	0	Nml	Nml	Di/Tri	Phasic	Dec	Normal
R	FlexCar Rad	Median	C6–8	Nml	3+	3+	0	Nml	Nml	Di/Tri	Phasic	Discrete	Dec
R	Infraspin	Supra	C5–6	Nml	3+	3+	0	Non	None	None		None	None
R	Latriceps	Radial	C6-7-8	Nml	1+	2+	0	Nml	Nml	Di/Tri	Phasic	Dec	Normal
R	LongHdTricep	Radial	C6-7-8	Nml	0	1+	0	Nml	Nml	Di/Tri	Phasic	Full	Normal
R	PalmarisLong	Median	C8-T1	Nml	0	0	0	Nml	Nml	Di/Tri	Phasic	Full	Normal
R	Pronatorter	Median	C6–7	Nml	2+	3+	0	Nml	Nml	Di/Tri	Phasic	Discrete	Dec
R	Rhomboids	DorsS	C5	Nml	0	0	0	Nml	Nml	Di/Tri	Phasic	Full	Normal
R	SerratAnt	LnTho	C4–6	Nml	2+	2+	0	Non	None	None		None	None
R	Supraspin	Supra	C5–6	Nml	3+	3+	0	Non	None	None		None	None
R	Trapezius	Spin	C3–4	Nml	0	0	0	Nml	Nml	Di/Tri	Phasic	Full	Normal

degrees of weakness in the wrist flexors/extensors and finger flexors. The intrinsic muscles of the hand were grossly within normal limits. Sensation exam was diminished over the lateral aspect of the right arm extending down over the first two digits. Reflexes were diminished/absent in biceps, brachioradialis, and triceps testing.

Diagnosis and management

Electromyographic studies were performed 3–5 weeks post-injury. Nerve conduction studies of sensory nerves demonstrated an absent right lateral antebrachial cutaneous nerve response (C5, C6) and decreased responses in the right superficial radial (C6) and right median nerve to the third digit (C7) (Fig. 4). More specifically, there was a 50% amplitude reduction in the right superficial radial sensory nerve and a 30% amplitude reduction in the right median sensory response (Table 1). Up to a 50% reduction in amplitude is acceptable in electrodiagnostic testing. However, these decreased amplitudes likely represent a component of postganglionic nerve injury to the C6 and C7 nerve roots. The ulnar sensory amplitudes (C8) were found to be symmetric.

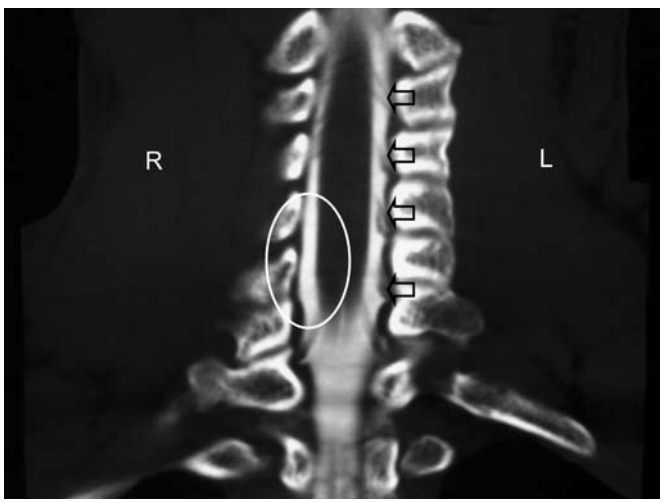

Fig. 5. Nerve root avulsion. Coronal reconstruction of CT myelogram demonstrates normal visualized left-sided nerve roots (open arrows) at multiple levels with absent/partial nerve roots on the right C6 and C7 levels (open oval)

Electromyographic needle examination revealed abnormal spontaneous activity in the form of positive sharp waves and fibs with no motor units in largely C5 and C6 innervated muscles (Table 2). There was minimal abnormal spontaneous activity in the form of PSWs in the long head of the triceps, but recruitment was normal. There was abnormal spontaneous activity in the form of positive sharp waves and fibs, with normal motor unit configuration and reduced recruitment in predominantly mixed C6, C7, and C8 innervated muscles (Table 2). Muscles innervated by cervical roots cranial to C5 and caudal to C7 were all normal. The only other normal tested muscle was the C5 innervated rhomboids, which, in light of the evidence of denervation of other C5 muscles, indicates a lesion in the C5 nerve root distal to the take off of the dorsal scapular nerve and, therefore, postganglionic.

Electrodiagnostic studies in this patient, therefore, reflect a severe right brachial plexopathy involving predominantly the C5 and C6 nerve roots as well as incomplete C7 nerve root denervation. The study also indicates that the C5 nerve root injury is postganglionic and the C6 root injury to be primarily preganglionic.

A further examination of the nerve roots was then undertaken by way of a CT myelogram. This study demonstrated normal cervical nerve roots on the left (Fig. 5). However, evaluation of the individual nerve root levels on the right revealed no visualization of dorsal or ventral nerve roots at the C5–C6 levels and partial roots at

the C6–C7 levels confirming both complete C6 and incomplete C7 nerve root avulsions (Fig. 6).

Final Diagnosis

1. Postganglionic C5 nerve root injury
2. Preganglionic complete C6 and partial C7 nerve root avulsions

Treatment and outcomes

The patient was subsequently referred to a surgical specialist for evaluation and was enrolled in a physical therapy regimen to maintain range of motion and perform strengthening exercises. The patient was deemed an appropriate surgical candidate and underwent nerve transfer surgery with the ultimate goal of restoring elbow flexion and shoulder abduction. Surgical technique involved transfer of spinal accessory nerve to suprascapular nerve, median nerve to brachialis, ulnar nerve to musculocutaneous nerve, and radial nerve to axillary nerve (Fig. 7).

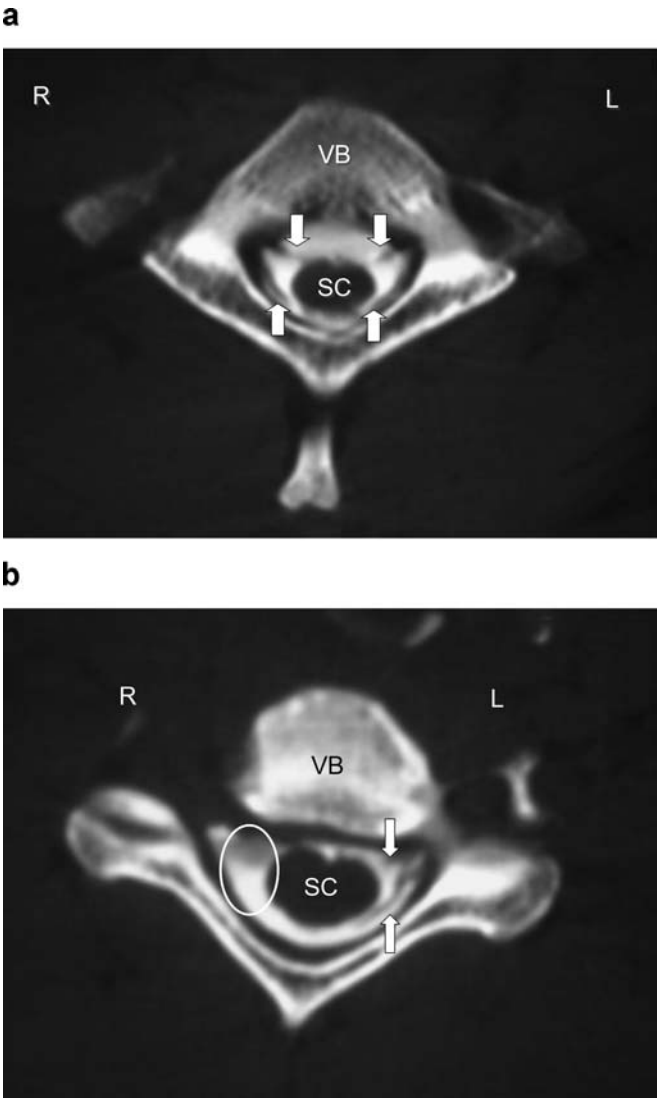

Fig. 6. Nerve root avulsion. (**a**.) Axial images of CT myelogram demonstrating intact ventral (downward arrow) and dorsal (upward arrow) nerve roots at an unaffected cervical spinal level. (**b**.) Axial images of CT myelogram demonstrating intact ventral and dorsal nerve roots on left (arrows) and absence of nerve roots on the right (open oval). (VB=vertebral body, SC=spinal cord)

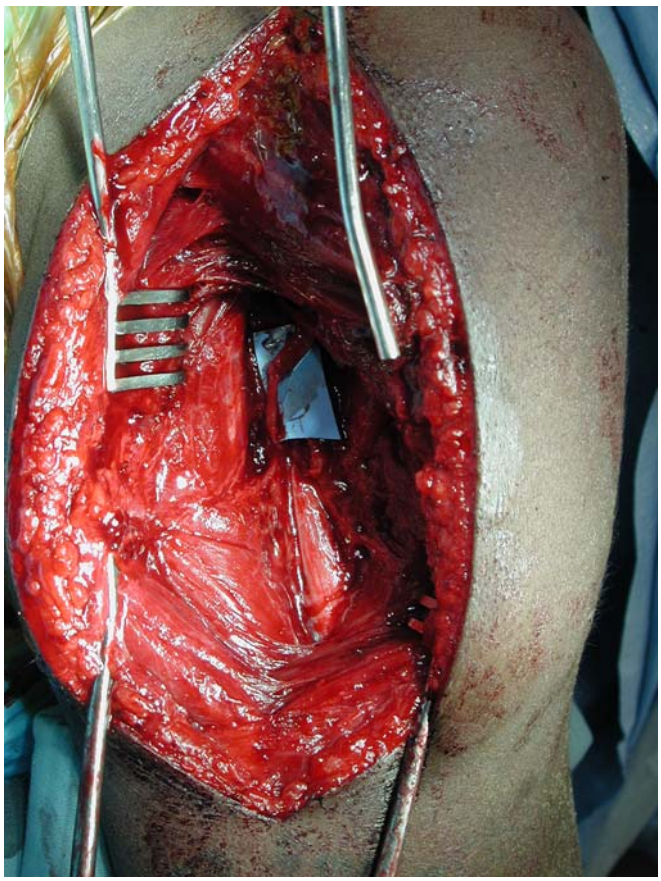

Fig. 7. Intraoperative photo of nerve transfer

The patient was evaluated at 6 weeks postoperatively with no evidence of gross muscle motion at shoulder or elbow. At 3-month follow-up, the patient was able to demonstrate a hint of supraspinatus activity and biceps contraction (Fig. 8). Physical therapy program continued with focus on EMG biofeedback to learn activation of muscle by transferred nerves. Six months postoperatively, the patient displayed evidence of increasing biceps bulk and decreased shoulder subluxation. Motor testing revealed active muscle activity with the ability to flex elbow to 130° (Fig. 9) and weakly abduct the shoulder to 20° (Fig. 10) with deltoid and supraspinatus contractions evident.

The patient was then reexamined with EMG 9 months after surgical repair. Sensory nerve conduction studies revealed further reduced amplitude on testing the right median nerve supplying the third digit. The right lateral antebrachial cutaneous (LAC) and superficial radial sensory nerve studies were not significantly changed from the initial study (Table 3, Fig. 11).

EMG needle examination of tested muscles continued to show abnormal spontaneous activity (Table 4). Nascent potentials were identified in the parts of the deltoid and the biceps (Fig. 12). Muscle recruitment was found to be discrete

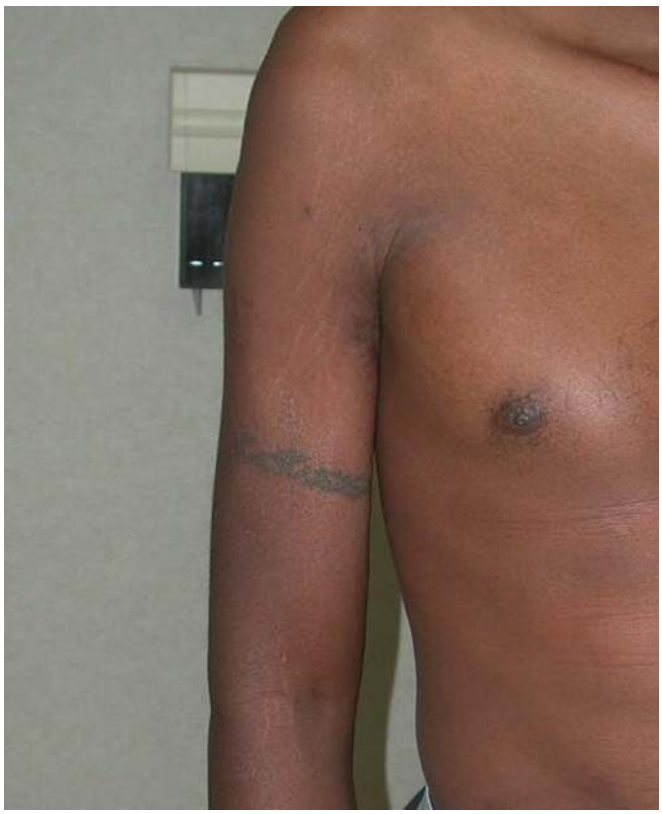

Fig. 8. Three-month post-surgical follow-up. Patient with evidence of biceps contraction

in the deltoid and decreased in the biceps. Polyphasic potentials were identified in the supraspinatus and infraspinatus. These polyphasics had a more mature and developed configuration than seen in a typical nascent potential. They had longer durations and were larger in amplitude, but did not have the configuration of the polyphasic potentials that arise from terminal collateral sprouting. These polyphasics appeared to represent matured nascent potentials, giving them a more developed appearance. EMG testing of the flexor carpi radialis demonstrated polyphasics that had developed from terminal collateral sprouting from axons that were intact at the time of the initial injury. There were also nascent potentials in the serratus anterior. We believe these arose from axonal regeneration from the incomplete C7 nerve root avulsion injury.

A review of the current EMG findings suggests a right brachial plexopathy. There is evidence of some axonal regeneration to the median sensory fibers to digit 3. No significant change was found in the superficial radial nerve or recovery of the LAC. The deltoid displayed evidence axonal regeneration, but limited recruitment and the patient had difficulty activating motor units. There is also evidence of axonal regeneration to the biceps, supraspinatus, and infraspinatus with good recruitment.

Discussion

Electrodiagnostic testing, more commonly known as EMG, can be helpful in nerve root and brachial plexus injuries to

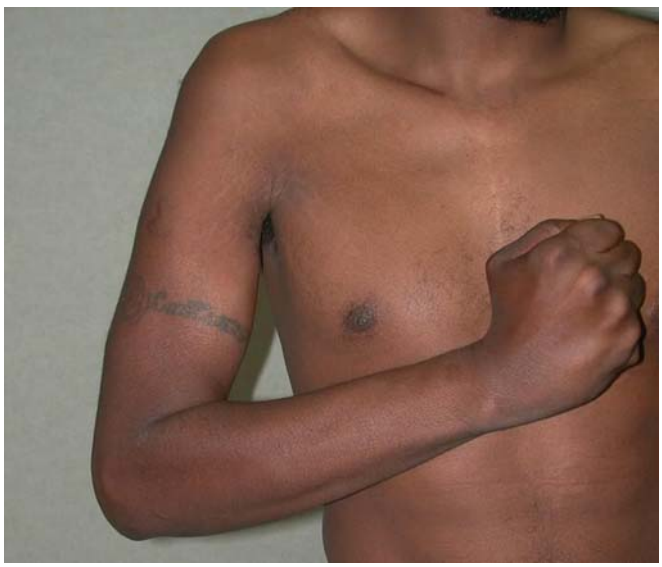

Fig. 9. Six-month post-surgical follow-up. Patient demonstrates elbow flexion

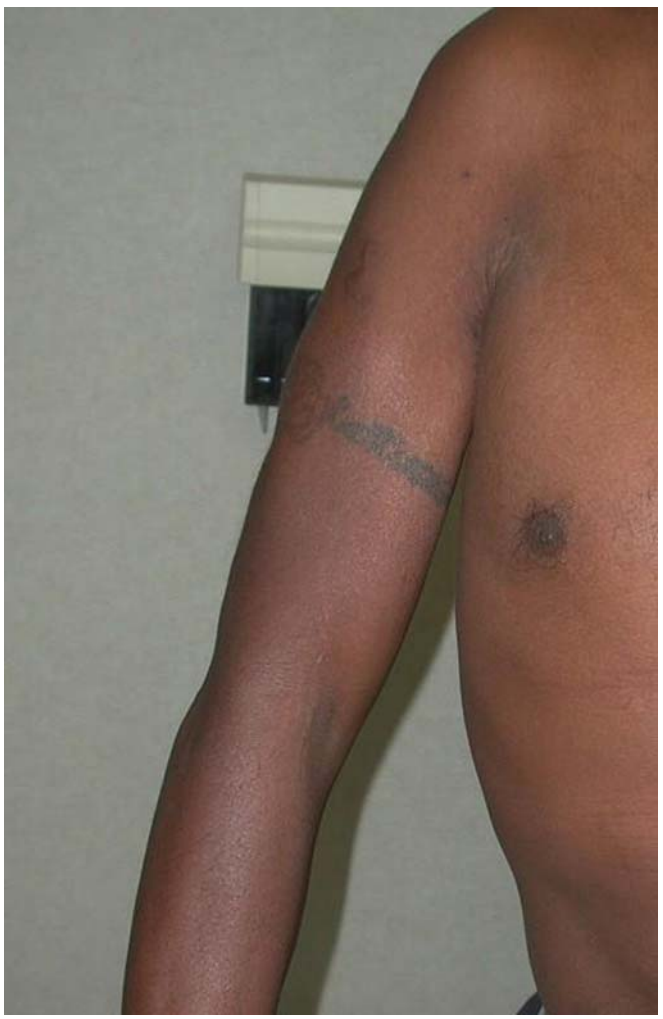

Fig. 10. Six-month post-surgical follow-up. Patient demonstrating shoulder abduction

Table 3 Nerve conduction tracings of sensory nerves at follow-up. Sensory summary table

Site	NR	Onset (ms)	Norm Onset (ms)	O-P Amp (μV)	Norm O-P Amp	Site1	Site2	Delta-0 (ms)	Dist (cm)	Vel (m/s)	Norm Vel (m/s)
Right Lat AnteBrach Cutan Sensory (Lat Forearm) 32.9°C											
Elbow	NR					Elbow	Lat Forearm		0.0		
Right Median D3 Sensory (3rd Digit) 31.8°C											
Wrist		2.9	<3.2	6.4	>10						
Right Sup Radial Sensory (FWS) 32.9°C											
Forearm		2.6		12.3	>10	Forearm	FWS	2.6	12.0	46.2	>45

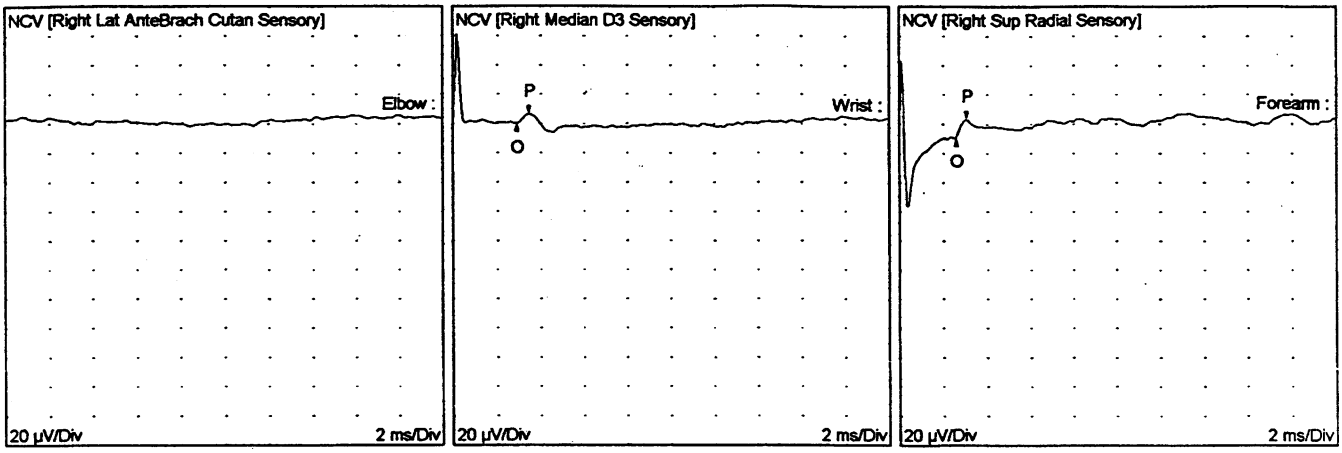

Fig. 11. Nerve conduction tracings of sensory nerves at follow-up. Sensory waveforms

Table 4 Electromyographic needle examination at follow-up

Side	Muscle	Nerve	Root	Ins Act	Fibs	Psw	Fascic	Amp	Dur	Configuration	Int Pat	Rec Int	Comment
Right	Biceps	Musculocut	C5-6	Nml	1+	1+	0	Nml	Nml	Nascents	Dec	Nml	
Right	Supraspinatus	SupraScap	C5-6	Nml	1+	1+	0	Nml	Nml	Inc Polys	Dec	Nml	
Right	Infraspinatus	SupraScap	C5-6	Nml	1+	1+	0	Nml	Nml	Inc Polys	Dec	Nml	
Right	SerratAnt	LongThor	C5-7	Nml	2+	2+	0	Nml	Nml	Nascents	Discrete	Nml	
Right	Deltoid (Mid)	Axillary	C5-6	Nml	2+	2+	0	Nml	Nml	Nascents	Discrete	Nml	
Right	FlexCarRad	Median	C6-7	Nml	1+	1+	0	Nml	Nml	Inc Polys	Dec	Nml	
Right	Deltoid (Ant)	Axillary	C5-6	Nml	2+	2+	0	Nml	Nml	Nascents	Discrete	Nml	
Right	Deltoid (Post)	Axillary	C5-6	Nml	2+	2+	0	Nml	Nml	Nascents	Discrete	Nml	

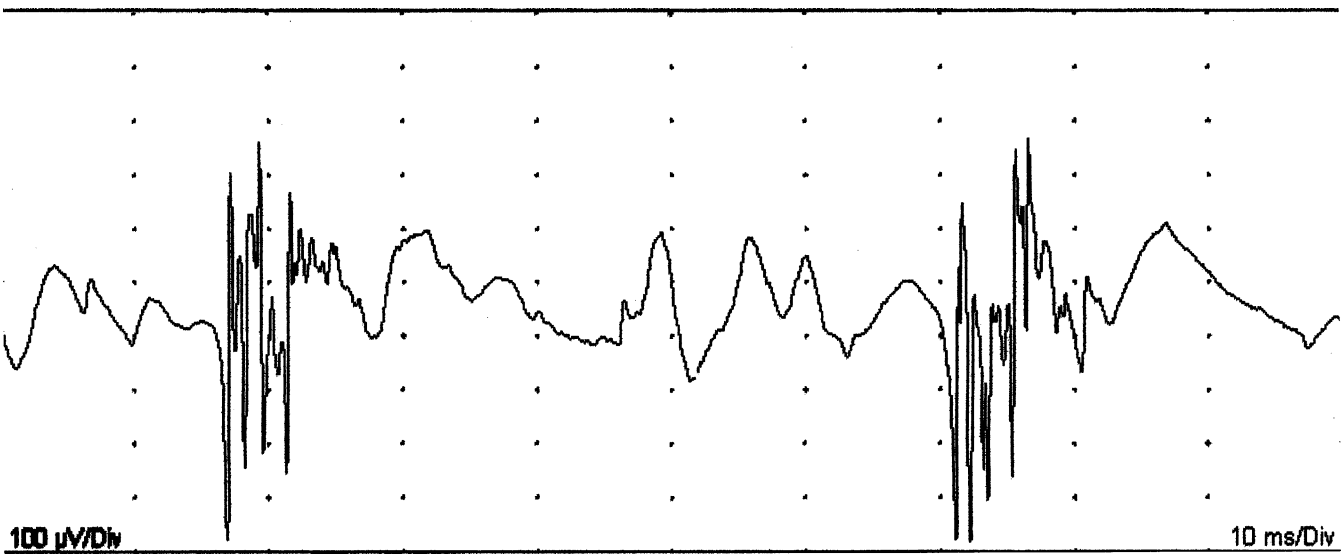

Fig. 12. Nascent potential on examination of the biceps

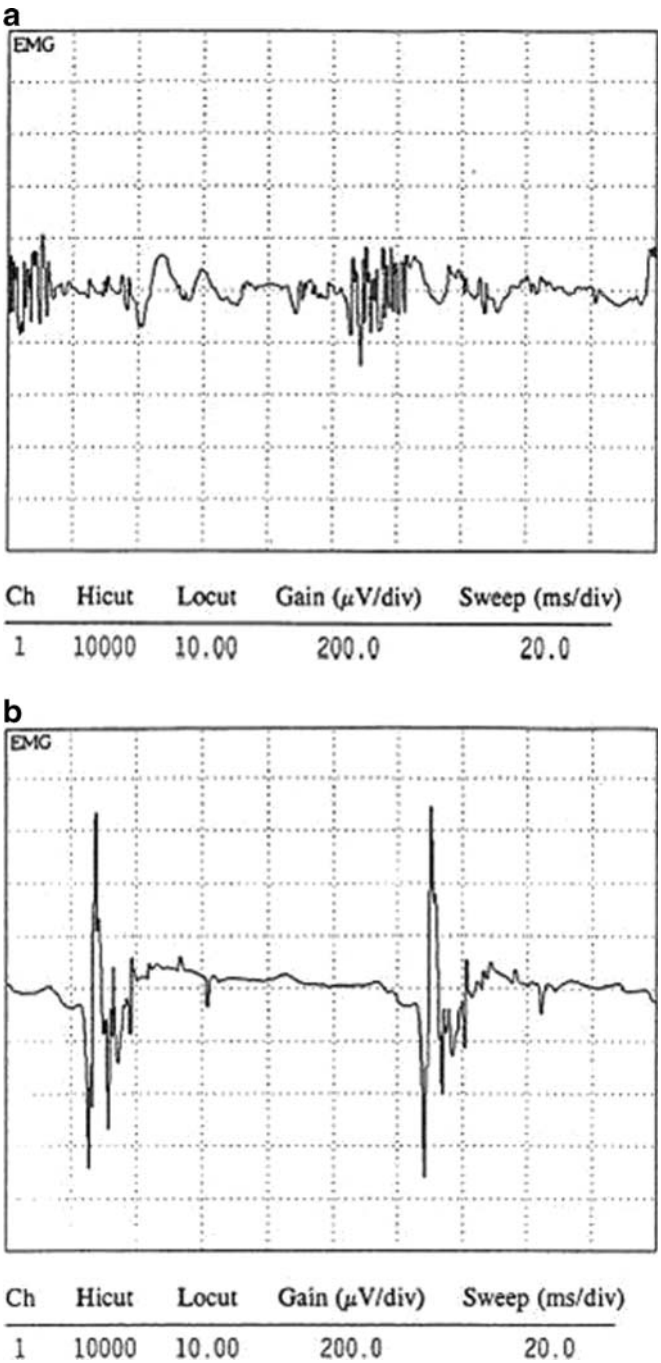

Fig. 13. **a**. A nascent potential from axonal regeneration. **b**. A long duration polyphasic potential from terminal collateral sprouting

localize the level of injury and determine if the lesion is pre- or postganglionic and complete or incomplete. When evaluating cervical roots and brachial plexus injuries, motor amplitudes may not add much to the diagnostic localization. However, sensory studies comparing the affected limb to the contralateral limb are helpful for anatomic localization, especially when determining root level as well as pre- and postganglionic injuries. After this type of nerve injury, determining the extent of injury also allows for prediction of prognosis. EMG testing can be used to demonstrate neurological recovery after nerve injury and nerve repair. One must keep in mind that nerve regeneration and reinnervation is a slow process, in the order of about 1–4 mm/day [9–11]. Thus, serial EMG testing may be helpful to monitor recovery.

Neurological recovery occurs in two forms: true axonal regeneration and the reinnervation of denervated muscle fibers by terminal collateral sprouting. True axonal regeneration occurs at the site where the nerve has been injured and where axonal degeneration begins [12]. The portion of the axon proximal to the level of injury regenerates distally through the endoneurial tube toward the denervated muscle. In the second form of neurological recovery, terminal collateral sprouting occurs by reinnervation of denervated muscle fibers by small nerve sprouts that arise from nearby uninjured and intact axons [13]. The most terminal portion of these axons generates sprouts that grow until they innervate the denervated muscle fibers. This obviously occurs over a much shorter distance.

EMG testing can identify and distinguish between these two processes [14]. Electrophysiologically, a normal motor unit has a di- or triphasic configuration. Polyphasic potentials are abnormal electrical configurations of a motor unit and can be identified after axonal injury. Two types of polyphasic potentials can form after axonal degeneration: (1) nascent potentials (Fig. 13a), and (2) motor units formed from terminal collateral sprouting (Fig. 13b). True axonal regeneration leads to the formation of nascent potentials, which are usually low in amplitude, polyphasic in configuration, and can have a short or normal and sometimes even long duration. The maturity of the myelin and the distribution and quantity of the muscle fibers being reinnervated will determine the size, the number of phases, and the duration of these nascent potentials. Terminal collateral sprouting leads to the formation of long-duration polyphasics, which have a very different configuration. These motor units always have a di- or tri-phasic motor unit followed by a polyphasic tail (Fig. 13b). The polyphasic tail represents the immature terminal collateral sprouts. Although both are polyphasic, their configuration is distinctly different, and it is important to remember that only the nascent potentials represent true axonal regeneration.

Electrodiagnostic studies are especially useful after surgical procedures involving nerve transfers. In these patients, muscles like the supraspinatus, deltoid, and biceps are now innervated by different nerves. Our patient now had the supraspinatus and infraspinatus innervated by a branch of the spinal accessory nerve, the deltoid innervated by a branch of the radial nerve, the brachialis innervated by a branch of the median nerve, the nerve and the biceps innervated by a branch of the ulnar nerve. Testing these muscles for EMG evidence of axonal regeneration can be difficult. The individual will usually have cognitive difficulty activating these muscles, and muscle reeducation with the use of biofeedback is often necessary.

Frequently, an individual may show no improvement in strength several months after a nerve injury or nerve transfer surgery. The clinical impression may be that no recovery has occurred. However, when axonal regeneration occurs, the initial immature nascent potentials are small and

may be incapable of generating a significant or a detectable force. EMG testing at this stage may identify these early immature motor units. This not only indicates neurological recovery when clinically there appeared to be none, but also prognosticates that additional functional recovery should occur in time.

Conversely, after a neurological injury an individual may show signs of improved strength and function. This could represent a form of neurological recovery, but adaptive biomechanical changes, functional compensation, and muscle substitution patterns can all lead to improved strength and function. These may give the appearance of neurological recovery when in fact there may be none. EMG needle testing can definitively answer this question. If there are no active motor units in a given muscle, then neurological recovery has not yet occurred.

Neurological recovery is also dependent on healthy viable muscle tissue. This is necessary for the release of nerve growth factors from denervated muscle. These factors act as a catalyst to stimulate the axon to regenerate. If the denervated muscle becomes fibrotic, these factors may no longer be released. Muscle tissue must also remain viable and electrically active if a regenerating axon is going to establish a connection with a functional neuromuscular junction. Chronically denervated muscle will eventually become fibrotic and electrically inactive. This usually happens somewhere between 18 and 24 months. In these cases, the use of electrical stimulation is controversial. There have been no studies to date proving that electrical stimulation improves functional outcome. However, electrical stimulation may be useful in maintaining muscle blood flow and viability as well as maintaining a receptive environment for axonal reinnervation

Conclusions

Burner syndromes are brachial plexus or nerve root injuries that commonly occur in football and are usually self-limiting with minimal morbidity. We have presented a case of a burner with catastrophic sequelae. Nerve transfers can help restore essential function in these types of injuries as demonstrated in this patient. Electrodiagnostics can play an important role in the anatomic localization of the injury, prognosticating outcome, determining role for and type of surgical intervention, monitoring recovery, and guiding rehabilitation.

References

1. Sallis RE, Jones K, Knopp W (1992) Burners: offensive strategy for an underreported injury. Physician Sportsmed 20:47–55
2. Archembault JL (1983) Brachial plexus stretch injury. J Am Coll Health 31:256–260
3. Speer KP, Bassett FJ III (1990) The prolonged burner syndrome. Am J Sports Med 18:591–594
4. Levitz CL, Reilly PJ, Torg JS (1997) The pathomechanics of chronic, recurrent cervical nerve root neurapraxia. The chronic burner syndrome. Am J Sports Med 25:73–76
5. Barnes R (1949) Traction injuries of the brachial plexus in adults. J Bone Jt Surg Br 31:10
6. DiBennedetto M, Markey K (1984) Electrodiagnostic localization of traumatic upper trunk brachial plexopathy. Arch Phys Med Rehabil 65:15
7. Warren RF (1989) Neurologic injuries in football. In: Jordan BD, Tsairis P, Warren RF. Sports neurology. Aspen, Rockville, MD, Aspen pp 235–237
8. Hershman EB (1990) Brachial plexus injuries. Clin Sports Med 9:311–329
9. Sunderland S, McArthur RA, Nam DA (1993) Repair of a transected sciatic nerve. A study of nerve regeneration and functional recovery: report of a case. J Bone Jt Surg Am 756:911–914
10. Berenberg RA, Forman DS, Wood DK et al (1977) Recovery of peripheral nerve function after axonotomy. Effect of triiodothyronine Exp Neurol 57:349–363
11. Forman BS, Berenberg RA (1978) Regeneration of motor axons in the rat sciatic nerve studied by labeling with axonally radioactive protein. Brain Res 156:387–436
12. Herbison GJ (1984) Neuropathic Needle Examination. AAEM Course A: fundamentals of EMG. American Association of Electrodiagnostic Medicine, Rochester, MN, pp 21–25
13. Wohlfart G (1958) Collateral regeneration in partially denervated muscles. Neurology 8:175–180
14. Feinberg JH (2006) EMG myths and facts. Hospital for Special Surgery Journal 2:19–21