Severe Brachial Plexus Injuries in American Football

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Abstract

This article reports a series of severe permanent brachial plexus injuries in American football players. The authors describe the mechanisms of injury and outcomes from a more contemporary treatment approach in the form of nerve transfer tailored to the specific injuries sustained. Three cases of nerve transfer for brachial plexus injury in American football players are discussed in detail. Two of these patients regained functional use of the extremity, but 1 patient with a particularly severe injury did not regain significant function. Brachial plexus injuries are found along a spectrum of brachial plexus stretch or contusion that includes the injuries known as “stingers.” Early identification of these severe brachial plexus injuries allows for optimal outcomes with timely treatment. Diagnosis of the place of a given injury along this spectrum is difficult and requires a combination of imaging studies, nerve conduction studies, and close monitoring of physical examination findings over time. Although certain patients may be at higher risk for stingers, there is no evidence to suggest that this correlates with a higher risk of severe brachial plexus injury. Unfortunately, no equipment or strengthening program has been shown to provide a protective effect against these severe injuries. Patients with more severe injuries likely have less likelihood of functional recovery. In these patients, nerve transfer for brachial plexus injury offers the best possibility of meaningful recovery without significant morbidity. [Orthopedics. 2016; 39(6):e1188-e1192.]

The terms “stinger” and “burner” are used by players, trainers, and coaches to describe a constellation of pain, burning, and paresthesias that radiate down the arm after contact to the head or upper extremity. Most of these injuries are caused by brachial plexus stretching or direct injury and contusion. Patients typically have sensory symptoms involving the C5 and C6 distributions that resolve within seconds to minutes. More severe injuries can be associated with objective muscular weakness that is usually limited in duration and, unless severe or prolonged, does not typically alter the prognosis.

In the United States, football is the leading cause of sports-related injury, and recent changes to rules and playing methods have been made to limit the risk of head and neck injury. A stinger, or brachial plexus traction injury, is the most common injury of the cervical spine and peripheral nerves in football. Brachial plexus traction injury with prolonged neurologic symptoms is the most common reason for high school and college football players to be referred to the emergency department or orthopedic clinic for evaluation of the cervical spine. It is estimated that stingers occur at least once in the career of 50% of football players and at an incidence of approximately 7.7% per year, with recurrence rates of as high as 57%. The recent emphasis on shoulder tackling to prevent head and neck injuries also may have incidentally increased the prevalence of brachial plexus stretch injuries.

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Three distinct mechanisms have been postulated to be the anatomic causative factors of stingers: nerve root compression, brachial plexus stretch, and direct blow to the superficial fibers of the brachial plexus. Nerve root compression is related to extension and compression of the cervical spine, resulting in nerve compression within the neuroforamen. Extension-compression injuries are more common in older collegiate and professional athletes, are more likely to be recurrent, and are associated with pre-existing neuroforaminal and cervical stenosis. This extension-compression type represents a large proportion of stingers, in many athletes, cervical stenosis does not appear to affect the initial occurrence of a stinger. Traction injuries to the brachial plexus often occur in younger high school athletes and are believed to result from a forceful blow to the head from the side or from head extension concomitant with shoulder depression. No association has been found between brachial plexus stretch and cervical and neuroforaminal stenosis, but these injuries may be associated with a small neck that dissipates the force of a tackle less readily or with poor blocking and tackling techniques. These traction-type stingers are believed to result from a lesion of the plexus at the division level. Patients with traction-type stingers may have a positive result on a brachial plexus stretch test as well as tenderness to palpation in the supraclavicular fossa; in contrast, extension-type stingers often cause coexistent neck pain. The third mechanism, direct blow to the brachial plexus, may be caused by compression at Erb’s point between the shoulder pad and the superior medial scapula. Although this mechanism may be a factor in a small number of injuries, it is not believed to be a major contributor to stingers.

This article reports a series of severe permanent brachial plexus injuries that occurred in American football players and describes the mechanisms of injury and outcomes from a more contemporary treatment approach. The Table summarizes the patients in this case series. One

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Axillary Nerve</th>
<th>Suprascapular Nerve</th>
<th>Musculocutaneous Nerve</th>
<th>Interval to Surgery, mo</th>
<th>Procedure</th>
<th>Outcome British Medical Research Council Grade</th>
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<td>0/5</td>
<td>0/5</td>
<td>0/5</td>
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<td>Cranial nerve XI, Hemi-contralateral C7 nerve, Intercostals 4, 5, 6, Musculocutaneous nerve</td>
<td>0/5 0/5 0/5</td>
</tr>
</tbody>
</table>

*Motor function defined in terms of British Medical Research Council grade, a standard 1-5 muscle strength scoring system.*
patient had complete neural disruption at the time of injury. The authors also identify a more severe form of this injury that should be included in an expanded grading system.

**Case Reports**

**Patient 1**

A 21-year-old male right-hand–dominant Division I college football player presented 3 months after an extension and lateral flexion injury that occurred during practice. He had immediate loss of upper extremity function involving the C5 distribution. Initial examination showed no active shoulder abduction, forward flexion, or external rotation. He had British Medical Research Council grade 2 internal rotation of the shoulder and no active elbow flexion. Strength was normal in the triceps and wrist. He also had a concussion that resolved with expectant management.

Computed tomography (CT) of the cervical spine showed right transverse process fractures at C6 to T1 (Figure 1). Electromyography obtained at 7 and 13 weeks after injury showed no axillary or musculocutaneous nerve function. Approximately 3 months after injury, the patient underwent multiple nerve transfers for upper extremity reconstruction. Cranial nerve XI was used for transfer to the suprascapular nerve. A branch of the radial nerve was used for axillary nerve transfer. Ulnar nerve and median nerve fascicles were transferred to the musculocutaneous nerve branches to the biceps and brachialis, respectively. After surgery, the patient began an early program of electrical stimulation. Range of motion was evaluated frequently in the training room. When evaluated 4 years after surgery, the patient had British Medical Research Council grade 3 deltoid function but no active shoulder external rotation. Elbow flexion improved significantly to grade 4 (Figure 2).

**Patient 2**

A 23-year-old male right-hand–dominant Division I college football player presented 1 month after he sustained a blow from the left side during a game. The blow caused shoulder depression with lateral deviation of the head and neck. Initially, symptoms were consistent with a short period of transient quadriplegia, and the patient was transported to the emergency department for further evaluation. On follow-up clinical examination, he had no elevation or internal or external rotation of the shoulder and no elbow flexion. Triceps strength was graded as M3. Two-point discrimination was elevated to 8 mm for the thumb and index finger. After the initial injury, CT of the cervical spine showed a small amount of material, consistent with a small subdural hemorrhage, around the cord. Electromyography performed 8 weeks after the injury showed changes consistent with C5 and C6 root level injury. Magnetic resonance imaging of the cervical spine showed mild congenital stenosis and mild degenerative disk disease at C5 and C6. Dedicated magnetic resonance imaging of the brachial plexus identified edema in the C5 and C6 nerve roots within the foramina. Multiple nerve transfers designed to restore shoulder function and stability as well as elbow flexion were performed approximately 4.5 months after the injury. For reconstruction, distal cranial nerve XI was transferred to the suprascapular nerve. A branch of the radial nerve to the medial head of the triceps was transferred to the axillary nerve. To restore elbow flexion, a branch of the ulnar nerve to the flexor carpi ulnaris was used to reinnervate the brachialis, and a branch of the median nerve to the flexor digitorum to the long finger was identified with nerve stimulation and transferred to the biceps branch of the musculocutaneous nerve. At 6 years after injury, shoulder flexion strength was grade 4, elbow flexion strength was grade 5, internal rotation was grade 5, and external rotation was grade 3 (Figure 3). In addition, the patient was able to forward flex the shoulder to 105°.

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*Figure 1: Axial computed tomography scan of the cervical spine showing characteristic transverse process fractures associated with brachial plexus injury.*

*Figure 2: Postoperative photograph showing improved elbow flexion and biceps tone after nerve transfer.*

*Figure 3: Postoperative photograph showing improved biceps and deltoid tone after nerve transfer.*
Patient 3
A 20-year-old right-hand–dominant Division 1 college football player who played the position of safety had a severe injury during game play. The injury was characterized by lateral neck flexion and shoulder depression. Sideline evaluation showed a completely flaccid left upper extremity and no peripheral pulse. Emergency surgical evaluation and exploration showed disruption of the entire brachial plexus as well as subclavian arterial injury. The patient underwent emergency vascular reconstruction and secondary upper extremity nerve reconstruction with multiple nerve transfers. These transfers included hemi-contralateral C7 with interposed vascularized reversed ulnar nerve graft to the median nerve; transfer of intercostal nerves 4, 5, and 6 to the musculocutaneous nerve; and transfer of cranial nerve XI to the suprascapular nerve. Unfortunately, the patient did not recover meaningful upper extremity function and is considering amputation.

DISCUSSION
Brachial plexus traction injury in football is underreported in the literature. Severe brachial plexus traction injury occurs in football players and may be related to player size, velocity, and technique. Although preventing serious injuries is clearly better than treating them, no mechanisms for preventing these injuries have been identified. Conditioning and strengthening programs focusing on the neck and core musculature have been proposed to decrease the incidence and recurrence of stingers, but these programs have not been systematically validated, and current equipment does not provide adequate protection. Although refinements in tackling techniques are believed to have decreased the incidence of stingers, this has not been proven, and recent emphasis on leading with the shoulder rather than the helmet actually may increase the incidence of brachial plexus traction injuries.

Nerve transfer is not a new idea, but the interest in perfecting and improving this procedure has changed the landscape for patients with brachial plexus traction injuries. For injuries at C5 and C6, the primary goal of treatment is restoration of elbow flexion, followed closely by stability and restoration of external rotation and abduction of the glenohumeral joint. Nerve transfers have become an important tool in treatment. When possible, the transfer should occur as close as possible to the target muscle to minimize time to functional recovery, and direct end-to-end repair in nerve transfer is preferred over the use of a nerve graft or alternative techniques.

A meta-analysis of the literature on nerve transfers for restoration of shoulder abduction showed that spinal accessory nerve transfers to the suprascapular nerve seem to provide the best outcomes in shoulder abduction strength. More distal dissection of the spinal accessory nerve allows preservation of trapezial muscle function with sufficient length to repair the suprascapular nerve without the need for interposition graft. Dual nerve transfer (cranial nerve XI to the suprascapular nerve combined with radial nerve transfer to the axillary nerve) is useful in increasing the general strength of the shoulder and has been proven as a viable technique.

Meta-analysis of the literature on nerve transfer for restoration of elbow function compared transfer to the biceps branch of the musculocutaneous nerve with intercostal nerves as well as the spinal accessory nerve, showing that intercostal nerve transfers were superior; however, the study analyzed only single-nerve transfers and a small number of ulnar nerve fascicle transfers. More recent reports on dual-nerve transfers for restoration of elbow flexion seem promising because the authors reanimated the biceps and the brachialis, which serves as the primary elbow flexor. Oberlin’s ulnar nerve flexor carpi ulnaris fascicle transfer, combined with transfer of an expendable fascicle from the median nerve to the biceps and brachialis, resulted in recovery of elbow flexion strength without significant donor nerve morbidity. In the current study, these nerve transfer methods provided significant improvement in arm function in patients with C5 and C6 root injury. In addition, 1 patient had a difficult case of complete plexus disruption that did not respond to comprehensive aggressive nerve reconstruction.

Stingers are often unreported, but they are believed to occur in 59% to 70% of all football players. Patients are often unaware of the potential for permanent injury and inability to return to play. Sporadic reporting may limit the ability of trainers and coaches to intervene to decrease their prevalence and the possibility of more serious brachial plexus injuries. A systematized reporting method and a database of injuries would be useful for trainers, coaches, and physicians.

Current algorithms for the treatment of brachial plexus traction injury are based largely on findings on physical examination and temporal changes in symptoms and strength. Patients with severe brachial plexus injuries require close follow-up to identify poor recovery. Reconditioning programs for severe brachial plexus injuries are unproven. If symptoms persist, evaluation to identify underlying injuries may be needed.

Electrodiagnostics may help to prognosticate and determine a role for surgical intervention, although definitive localization of the injury in the upper trunk or nerve root may be difficult. These diagnostic tests may have a role in monitoring recovery and guiding rehabilitation. Magnetic resonance imaging may allow visualization of the entire plexus, and CT myelography can help to distinguish injury of the nerve roots, rootlets, and trunk. Determination of the pre- or postganglionic location of brachial plexus injury is critical to determine the potential for spontaneous recovery. Depending on
their severity, postganglionic lesions have the potential for recovery and should be monitored closely with physical examination and electromyography.³¹ Preganglionic lesions and root avulsions have no potential for spontaneous recovery, and early surgical intervention with nerve transfer is indicated to maximize functional recovery.³⁰,³¹

**CONCLUSION**

Surgery is usually considered when the likelihood of nerve recovery is low without repair or transfer. Classically, nerve root avulsions are most likely to require reconstruction. Early nerve transfer or reimplantation improves pain relief because of reinnervation of the muscle.³² Suprascapular nerve neurotization is a high priority in upper limb reanimation for restoration of glenohumeral joint stability, shoulder abduction, and improved external rotation, whereas concomitant neurotization of the axillary nerve has led to improved outcomes in shoulder abduction.³³ The best results occur with early neurotization of the suprascapular nerve less than 6 months after injury.³³ Further study with advanced imaging and electrophysiology is needed to identify this injury at an earlier stage and stratify patients who have complete nerve transection and will benefit from early surgical intervention.

**REFERENCES**