

Chronic Brachial Plexopathies and Upper Extremity Proprioception and Strength

C. Buz Swanik, MS, ATC; Tim J. Henry, MS, ATC; Scott M. Lephart, PhD, ATC

ABSTRACT: Brachial plexopathies, where traction or compressive forces disrupt motor and sensory nerve conduction, are the most common nerve injuries in collision sports. Athletes frequently do not report these episodes, however, predisposing the brachial plexus to recurrent trauma. The purpose of this study was to identify how multiple injuries to the brachial plexus affects shoulder strength and proprioception. Ten male intercollegiate football players with at least three unilateral episodes of brachial plexopathies were tested an average of 10 weeks after the most recent episode. The uninvolved shoulder was used as the control. Isometric peak torque was assessed for shoulder abduction, external

rotation, and elbow flexion. Proprioception was measured under two conditions: threshold to detection of passive motion and reproduction of passive positioning. Dependent *t* tests revealed significant mean differences ($p < .05$) between the involved and uninvolved extremity for abduction peak torque, overall mean peak torque, and one out of four conditions of threshold to detection of passive motion conditions. This was in the neutral position moving into external rotation. In addition, subjects with greater numbers of episodes exhibited larger strength deficits. The results of this study emphasize the need for timely re-evaluation of athletes with chronic brachial plexopathies.

Brachial plexopathies are injuries to the nerves of the brachial plexus.¹⁹ These lesions occur frequently in collision sports such as football, hockey, and wrestling.^{1,19,22,29,30} In collegiate football, studies have shown that approximately 50% of the players report at least one episode per season.^{23,26} Many of these athletes (87%) complain of recurring incidents.²⁶ The competitive nature of athletics and the “play with pain” philosophy may give rise to gross under-reporting of these injuries.^{26,30} Athletes often continue to participate without notifying medical personnel of their symptoms.

Symptoms of brachial plexopathies include: transient burning, stinging, and/or muscle weakness throughout the involved upper extremity.^{1,10,24,27} Several mechanisms for producing this injury have been suggested. The most prevalent is a force that causes lateral flexion of the cervical spine and concomitant shoulder depression to the contralateral side.^{1,10,26,30,31} The interval between the cervical spine and shoulder increases, placing traction on the brachial plexus.⁵ Likewise, a blow to the supraclavicular region may cause damage by compressing the underlying nerves. These lesions frequently reside in the upper trunk as identified by electromyography (EMG) studies and clinical evaluations.^{10,19,27} The anatomical location of the upper trunk, both superior and superficial, makes it more vulnerable to compression and traction forces.^{15,27}

Research has evaluated some of the effects of these lesions on the efferent (motor) pathways. It has been suggested that a chronic syndrome can develop from repeated brachial plexopathies.^{22,30} However, the consequences of this syndrome on the neuromuscular system have not been documented. Moreover,

the effects of brachial plexopathies on shoulder proprioception have not been studied.

The purpose of this study was twofold: 1) to investigate the effects of repeated brachial plexopathies on the proprioception pathways in the shoulder, and 2) to assess how multiple injuries to the brachial plexus influences muscular strength. It was hypothesized that proprioception and strength would be deficient in the affected shoulder when compared to the healthy shoulder. Furthermore, subjects with a greater number of episodes would exhibit larger proprioception and strength deficits in their affected shoulders.

METHODS

This was a retrospective study that used the subject's contralateral healthy limb as an internal control for comparison. A certified athletic trainer identified and evaluated subjects by excluding cervical radiculopathies, neuropathies, and orthopedic injuries. Before participation, all subjects read and signed a medical history questionnaire and consent form approved by the University of Pittsburgh Biomedical Institutional Review Board. The experimental group consisted of 10 male, division I football players (age = 20.4 ± 1.5 years) with unilateral grade 1 brachial plexopathies. Grade 1 injuries or neuropraxias, display symptoms for only a few minutes.^{1,5,10} All subjects experienced at least three episodes during the 1994 season with a mean of 15.3 ± 13.5 over the course of their career and were tested ($\bar{x} = 10.3 \pm 3.2$ weeks) after the most recent episode. The dependent variables assessed were shoulder proprioception and isometric strength. The same examiner conducted random order testing during a single session.

Proprioception Assessment

Proprioception was measured with a proprioception testing device (Fig 1). Previous studies on the proprioception testing device revealed a test-retest reliability of $r = .92$.¹⁸ The subject

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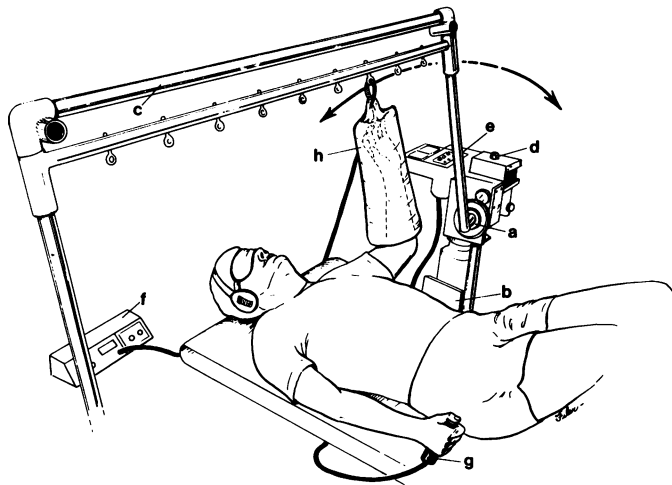


Fig 1. Proprioception testing device: a, rotational transducer; b, motor; c, moving adapter; d, control panel; e, digital microprocessor; f, pneumatic compression device; g, handheld on/off switch; and h, pneumatic compression sleeve. (From: Lephart SM, Kocher MS. The role of exercise in the prevention of shoulder disorders. In: Matsen FA, Fu FH, Hawkins RJ. *The Shoulder: A Balance of Mobility and Stability*. Rosemont, IL: American Academy of Orthopaedic Surgeons; 1993:611. Reproduced with permission from the American Academy of Orthopaedic Surgeons.)

was positioned supine with the shoulder abducted and elbow flexed to 90°. The forearm was placed in a pneumatic sleeve, which was attached to the drive shaft on the proprioception testing device. A head set and blindfold were fitted to eliminate auditory and visual cues.¹⁶ Proprioception was measured as the threshold to detection of passive motion and reproduction of passive positioning.^{4,16,17,18} Threshold to detection of passive motion is believed to selectively activate the quick-adapting mechanoreceptors responsible for sensation of joint motion or kinesthesia.^{4,9,17,18} Reproduction of passive positioning stimulates the slow-adapting mechanoreceptors that mediate joint position sense.^{4,9,17,18} The starting position, direction of movement, and shoulder side were randomized.

Threshold to detection of passive motion was initiated after three practice attempts. The subject signaled he was ready, and within the next 10 seconds the proprioception testing device passively rotated his arm at a velocity of 0.5°/s. Upon perceiving motion, the subject disengaged the device by pressing a handheld switch and the degree of rotation was recorded. Three trials were performed, moving into both external and internal rotation.^{4,16,17}

Reproduction of passive positioning was also tested after three practice attempts. The subject's shoulder was rotated from two reference positions to a randomized angle in both external and internal rotation. The velocity of rotation was varied to nullify time cues. Subjects were allotted 10 seconds to concentrate on the presented angle; the arm was then passively moved back to the reference position. The subject used an on/off switch to passively reproduce the presented angle.^{4,16,17} The difference between the presented angle and the reproduced angle was recorded in degrees.

Strength Assessment

Strength was measured using the Cybex II Isokinetic Dynamometer (Lumex, Inc, Ronkonkoma, NY), which has proven to be reliable and was calibrated before beginning the study.²¹ Muscular assessments of isometric peak torque were recorded for shoulder abduction, external rotation, and elbow flexion. Each test position included a warm-up, followed by three isometric trials. All trials consisted of a 3-second maximum voluntary contraction and a 10-second rest between trials, during which the force curves were scrutinized for the sincerity of effort.^{7,8,14} The same procedure for each of the three strength conditions was repeated for the opposite upper extremity.

Shoulder external rotation was tested in a standing position. The subject's arm was at his side with 90° of elbow flexion. A hand grip and VELCRO® straps secured the forearm to the dynamometer, while permitting shoulder rotation (Fig 2).^{6,7,14} Shoulder abduction was measured in a seated position, with the trunk reclined 40°. The torso was secured by VELCRO straps while the subject grasped an adapter connected the dynamometer. This arrangement restricted motion to shoulder abduction (Fig 3).^{7,20,21} Elbow flexion strength was tested with the

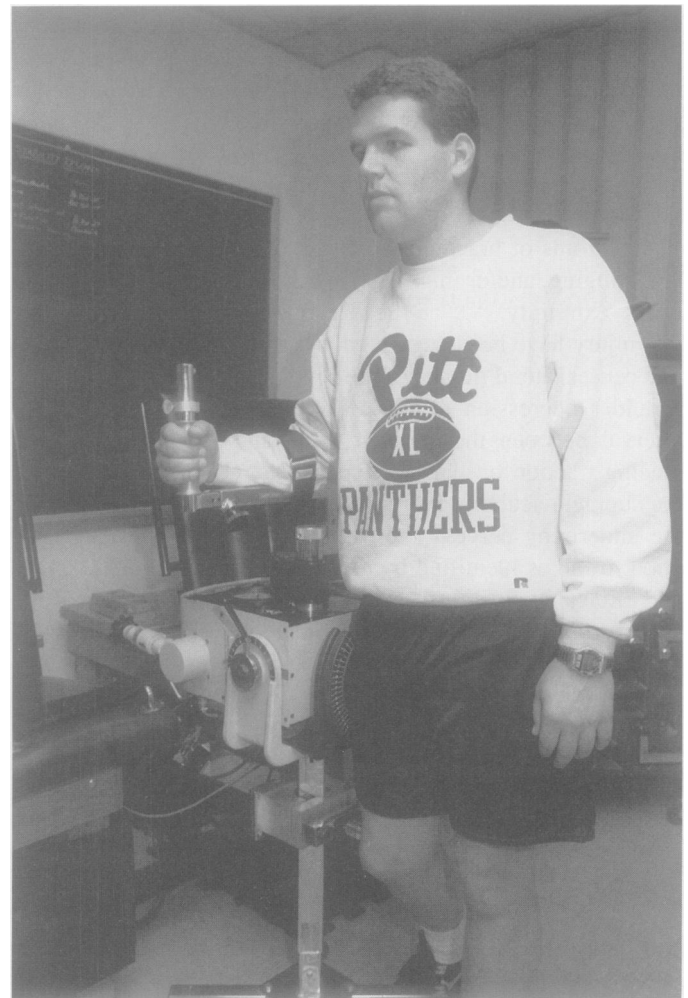


Fig 2. Isometric shoulder external rotation test position on the Cybex II dynamometer.

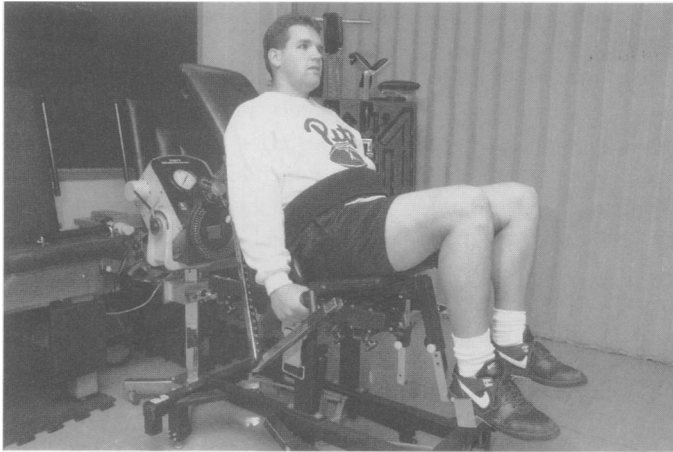


Fig 3. Isometric shoulder abduction test position on the Cybex II dynamometer.

subject lying supine, arm abducted 45° and resting on a pad. The subject's torso was secured with VELCRO straps while he grasped an adapter connected to the dynamometer (Fig 4).^{7,13}

RESULTS

Dependent *t* tests were used to determine the mean differences between the involved and uninvolved shoulders. All values are reported as means and standard errors. Analysis revealed that shoulder abduction strength of the involved shoulders ($\bar{x} = 87.6 \pm 7.6$ ft/lb) was significantly less ($p < .05$) than the uninvolved shoulders ($\bar{x} = 101.4 \pm 7.4$ ft/lb). In addition, the mean of the three combined strength scores was significantly less ($p < .05$) for the involved side ($\bar{x} = 59.1 \pm 4.0$ ft/lb) when compared to the uninvolved limb ($\bar{x} = 66.3 \pm 3.5$ ft/lb). The mean peak torque values for external rotation and elbow flexion were not significant ($p < .05$; Fig 5).

The involved shoulders also demonstrated a significantly longer ($p < .05$) threshold to detection of passive motion in the neutral position moving into external rotation for the involved arm ($\bar{x} = 1.9^\circ \pm 0.26^\circ$) as opposed to the uninvolved shoulder ($\bar{x} = 1.45^\circ \pm 0.22^\circ$). All other conditions for proprioception were not significant ($p > .05$).

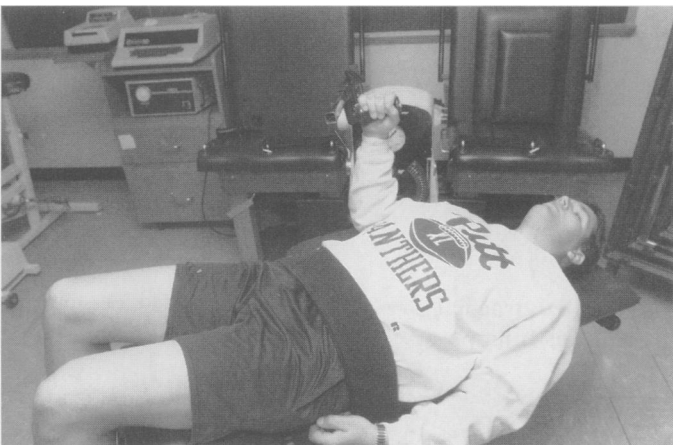


Fig 4. Isometric elbow flexion test position on the Cybex II dynamometer.

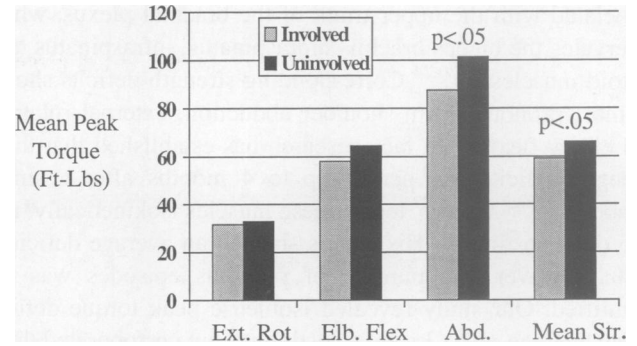


Fig 5. Mean peak torque (ft/lb) of the involved vs uninvolved shoulders for external rotation (Ext Rot), elbow flexion (Elb Flex), abduction (Abd), and mean of the three strength conditions (Mean Str).

Pearson's product moment correlation was used to determine the relationship between the number of episodes and peak torque deficits in the involved and uninvolved upper extremities. There was a strong relationship ($r = .80$, $p < .003$) between the strength deficits of the involved limbs, and the number of career episodes reported (Fig 6). The threshold to detection of passive motion and reproduction of passive positioning conditions were not significant in relation to the number of episodes.

DISCUSSION

The results of proprioception testing suggest that repeated, grade 1 plexopathies have little effect on threshold to detection of passive motion and reproduction of passive positioning. The mean isometric strength values appear to indicate deficits under all three conditions tested, but only abduction strength reached a significant level. The most remarkable finding in this data was that subjects with increased numbers of brachial plexopathies also demonstrated less isometric peak torque in their involved shoulder. Evidence of this effect until now has been anecdotal.

Isometric Strength

Complaints of transient muscle weakness are common among athletes with brachial plexus lesions. These deficits are

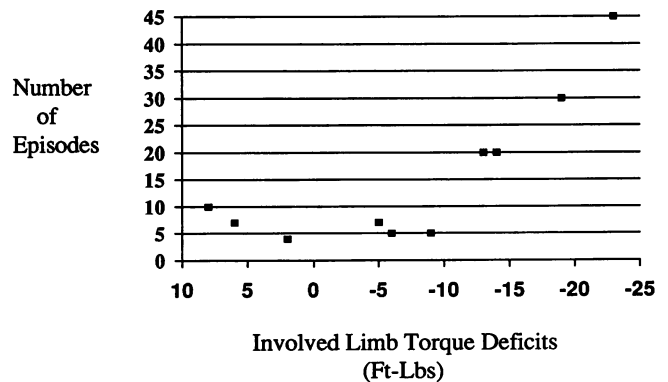


Fig 6. Strong relationship ($r = .80$) between the strength deficits of the involved limbs and the frequency of brachial plexopathies reported by subjects.

associated with the upper trunk of the brachial plexus, which innervates the biceps brachii, supraspinatus, infraspinatus, and deltoid muscles.^{17,20,24} Corresponding strength deficits should be most pronounced in shoulder abduction, external rotation, and elbow flexion. In fact, research has established that these strength deficits can persist up to 4 months after a single episode.^{10,23,27} Speer²⁷ tested these muscles isokinetically, at 3 to 5 days postinjury. His results showed an average deficit of 14%; however, the number of previous episodes was not quantified. Our study revealed isometric peak torque deficits for an average of 10.3 weeks postinjury, but corroborated those reported by Speer.²⁷ The involved upper extremities were 13% weaker for elbow flexion, and abduction peak torques were 14% less than the uninvolved shoulders. These results were expected and concur with Archambault's¹ statement that the deltoid is often the last to recover full strength, although the mechanism is not understood.

There was no apparent strength deficit in external rotation torque values. This discrepancy with previous literature may be due to methodology. Cahalan⁶ found that peak torque for external rotation is generated at 90° of abduction, whereas our testing protocol adhered to those suggested by the manufacturer⁷ and employed by Murray,²⁰ who tested external rotation in the neutral position. Moreover, Kuhlman¹⁴ reported the rotator cuff muscles account for only 50% to 75% of external rotation strength; consequently, subjects with strength deficits in their rotator cuff could compensate by recruiting additional muscles. Although individual subjects did present with deficits in external rotation, the procedure used in this study may not have accurately isolated and measured the strength of the supraspinatus and infraspinatus muscles.

Upon reviewing the data, a trend was found within the strength deficits. Each subject appeared to exhibit strength deficits in two of the three conditions. This can be explained by the variability of mechanisms and locations of each injury within and between subjects. For this reason, the three mean peak torque values for each strength condition was averaged and also found to be significantly different ($p < .05$) between the involved and uninvolved shoulders. This value by no means represents cumulative shoulder strength, but rather a general assessment of the three conditions tested in this study.

The Effect of Repeated Episodes on Strength

Robertson²³ and Vereschagin³⁰ have previously suggested that a chronic syndrome may develop from repeated acute brachial plexopathies. These events are characterized by an increase in the frequency and severity of episodes, resulting in larger strength deficits. However, this tendency has not been objectively documented. We explored the relationship between frequency and strength by comparing the overall mean peak torque value to the number of episodes each subject recalled throughout his career. Results established a strong relationship between the strength deficits and the number of episodes. Multiple episodes of grade 1 plexopathies may cause scarring of the epineurium and adjacent tissue. Because this scar tissue is less elastic, it is more susceptible to repetitious trauma. For

each injury, the athlete's symptoms are transient, but strength deficits remain between episodes.

The impact of these strength patterns on shoulder stability and coordination are not completely understood.¹⁷ It is believed that these muscles (the posterior rotator cuff, deltoid, and biceps) create force couples, which have an integral role in the dynamic stabilization of the glenohumeral complex.^{4,16,17} Lesions to the upper trunk of the brachial plexus may disrupt this mechanism by impairing efferent motor control. This creates a muscle imbalance in the shoulder, increasing the susceptibility to acute and chronic musculoskeletal injuries of the shoulder (Fig 7).^{4,16,17}

Proprioception

Despite the pronounced effect of these lesions on motor neurons, sensory fibers appear relatively unaffected.¹⁰ Athletes typically experience only a few minutes of paresthesia with a grade 1 plexopathy.^{10,12} The reason efferent fibers sustain more damage than afferent fibers is not understood. Leffert¹⁵ suggests that large, myelinated afferent fibers can diffuse traction and compression forces more so than smaller efferent fibers. However, proprioception depends on both the afferent and efferent pathways.^{9,17} Mechanoreceptors in cutaneous, muscular, and articular structures transduce mechanical deformation of tissue into electrical signals.⁵ The brachial plexus transmits this proprioceptive (afferent) information to the central nervous system.^{4,9,17} The appropriate response is then transmitted back through the plexus along the efferent (motor) pathway. Deafferentation of the proprioception receptors can disrupt motor coordination and/or joint stabilization.^{2,4,9,17} However, studies have demonstrated a strong compensatory mechanism for joint motion and position sense.^{2,3,17} We hypothesized that lesions to the brachial plexus would disrupt the afferent-efferent proprioception loop.

Sainburg et al²⁵ studied patients with proprioceptive deafferentation resulting from sensory neuropathies. They found a

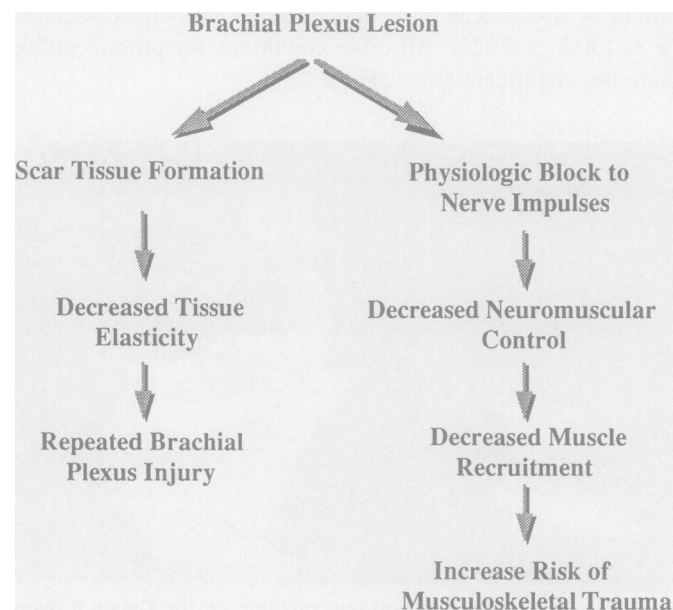


Fig 7. Chronic brachial plexopathy injury paradigm.

large variability in the timing of agonist/antagonist muscle activation, which resulted in the loss of motor coordination and joint position sense. In contrast, our study revealed minimal deficits in proprioception. The transmission of joint motion and position information does not appear to be affected by grade 1 lesions to the upper trunk of the brachial plexus. Although threshold to detection of passive motion in the neutral position with external rotation was significantly less in the involved shoulder, no distinct trends were observed. Several arguments can explain these findings.

It is possible that only the efferent pathway is disrupted, as supported by the strength deficits. However, conduction velocity throughout the proprioception loop remains sufficient to compensate for any loss. Likewise, the quantity of proprioceptive information obstructed by a lesion may be negligible in comparison to the amount transmitted to the central nervous system.

Training has also been shown to enhance proprioception.^{2-4,11} Subjects in this study were participating in either rehabilitation, weight training, or full practice sessions and were tested on average 8.6 weeks after the most recent episode. Proprioceptive deficits may be present during the initial phase, but resolve with time and continued activity. Therefore, proprioception testing at 72 hours postinjury may provide more insight on the effect of lesions to the afferent-efferent loop.

Clinical Implications

Clinical evaluation is crucial for locating and classifying lesions within the brachial plexus. Athletes with lesions to the upper trunk present unilateral, circumferential burning and stinging sensations, which do not correspond to dermatomes.^{10,21,29} Complaints of point tenderness in the supraclavicular fossa (Erb's point) are common.¹⁰ However, limitations in cervical strength and range of motion are not present, unlike injuries to the nerve root.^{10,19,23} Transient weakness in the upper extremity may be immediate or delayed 72 hours.^{1,23,27} Subjects in this study all complained of immediate but transient muscle weakness lasting 3 to 5 minutes. Muscle weakness and abnormal EMGs cannot persist longer than 4 weeks if a lesion is to be classified as grade 1.^{19,27} EMG studies and nerve conduction velocity tests are more accurate assessments of brachial plexus lesions, but do not appear abnormal until demyelination occurs approximately 2 weeks postinjury.^{27,28} In addition, these tests do not correlate with strength or functional tests that clinicians frequently rely on to determine if an athlete is able to participate.^{27,28}

The chronic syndrome develops from repeated episodes and is the responsibility of both the athlete and medical personnel. Subjects in this study reported only 52% of their episodes to athletic trainers or coaches, confirming research by Sallis et al²⁶ who observed that 50% of these chronic injuries go unreported. This statistic is particularly disturbing considering the persistent deficits in strength. Speer²⁷ also suggested that athletes participating on the collegiate level sustain more brachial plexopathies than athletes in lower levels of competition. In this study, 63% of these episodes occurred during the athlete's most recent collegiate season. The concentration of

these injuries and low reporting rate can most likely be attributed to the intensity of competition and motivation of athletes to continue participating. Clinicians are also at fault for returning the athletes to competition without considering the delay in strength deficits and EMG abnormalities. Because EMG testing is not feasible for all of these injuries, clinicians must classify injuries based on physical exams. The current classification does not consider multiple episodes or the associated strength deficits revealed in this study. This evidence demonstrates the need for a classification strategy which includes the duration of symptoms and number of episodes. However, without examining athletes between each episode, clinicians cannot differentiate the pathoetiology responsible for strength deficits.

CONCLUSION

A trend in strength deficits was identified in subjects with repeated episodes of grade 1 brachial plexopathies, but only one was statistically significant. It has been suggested that repeated episodes cause an increase in the frequency and severity of these lesions. Our results support this allegation. Greater strength deficits were observed in subjects with more episodes. Four tests of reproduction of passive positioning were not different between groups; however, threshold to detection of passive motion was significantly different in neutral position moving into external rotation. Three other conditions of threshold to detection of passive motion were within normal limits.

The results of this study stress the importance of timely re-evaluations for athletes with chronic brachial plexopathies. Clinicians should place emphasis on the athlete's previous history, because strength deficits may not appear for 72 hours and EMG analysis is not effective until 2 weeks after a grade 1 brachial plexopathy.^{1,10,26} Information from this study can assist in determining the participation status of athletes with chronic episodes. Although athletes do not always report episodes or display functional deficits, continued participation with these lesions predisposes the upper extremity to recurrent brachial plexopathies and/or musculoskeletal trauma.

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