

# Isolated Peripheral Nerve Lesions of the Brachial Plexus Affecting the Shoulder Joint

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**Abstract:** Shoulder pain, weakness, and instability are common findings of nerve entrapment and traumatic and inflammatory lesions of the brachial plexus. These disorders can be properly diagnosed with careful clinical examination and electrophysiologic testing. An understanding of the kinesiology of muscles acting on the scapula aids in the diagnosis of patients presenting with muscle atrophy and scapular winging.

This article reviews the kinesiology of the muscles acting on the scapula, the clinical manifestation of isolated peripheral nerve lesions of the brachial plexus, and the indications for clinical observation and rehabilitation as well as for surgical treatment of established lesions.

## Isolated Peripheral Nerve Injuries of the Brachial Plexus

As a multiarticulated joint system, the upper extremity is a delivery system to position the hand in space for its prehensile function (grasp, pinch, and hook) and as a tactile organ. To fulfill its function, this system requires both a stable and mobile base. The scapula is superbly positioned phylogenetically to serve this purpose. It is positioned dorsally and rotated laterally 35 to 40 degrees from the frontal plane to accommodate the upright position of the bipedal primate [17].

The glenohumeral joint behaves as a ball-and-socket joint. Although there are differences in size between the glenoid (average 25 mm in diameter) and the humeral head (average 48 mm in diameter), as a unit, they form a functional congruous joint. The radii of curvature of the cartilaginous surfaces approach 99% [33] and the center of rotation is functionally fixed to minimize translation of the humeral head [33]. The resultant forces are compressive rather than shearing in nature. These forces are dissipated in a ball-and-socket joint, like that of a hip [33]. Only imbalance created either by loss of the rotator cuff muscles (which act to dynamically compress and rotate the humeral head) or the scapular stabilizer muscles (which serve to dynamically position and stabilize the glenoid) can disrupt this fine, elegant balance of the scapular humeral rhythm [17,33]. As a result, shearing forces, which are so destructive to joint function, are minimized [33].

The nomenclature of scapular motion about the thorax is described as elevation, depression, adduction (retraction), abduction (protraction), medial rotation of the inferior angle of the scapula, lateral rotation of the inferior angle of the scapula, and anterior and posterior tilting [11,17]. Upward rotation of the scapula is most important for arm elevation. The upward rotator muscles of the scapula are the trapezius and the serratus anterior muscles. However, Inman et al. [17] have also included the levator scapula. Maximum scapular rotations can only occur when the trapezius and the serratus anterior muscles function normally.

To understand the effects of peripheral nerve injuries of the brachial plexus on shoulder girdle function, it is crucial to realize that muscles do not act independently. Each muscle is a component of a muscle force couple that executes normal shoulder motion [17]. This concept is reinforced and painfully realized when a single muscle transfer is attempted to compensate for a loss of multiple muscle units or when there is diffuse brachial plexus injury.

Isolated peripheral nerve injuries of the brachial plexus present as common clinical manifestations: pain, weakness, and instability. These complaints are common to all dysfunctions of the shoulder girdle and may be misdiagnosed and mistreated as disorders of the glenohumeral joint [30]. A detailed history and physical examination is imperative. Although a thorough clinical neurologic examination may have been performed, the true diagnoses can easily be missed. For example, the differential diagnosis of shoulder pain with atrophy of the supraspinatus and infraspinatus muscles may be due to either a compressive lesion secondary to a suprascapular nerve ganglion or to a chronic rotator cuff tear [14]. In addition, as part of the differential diagnosis, it must also be realized that these two lesions may also coexist [29]. The true diagnosis can only be ascertained by electrodiagnostic testing and shoulder magnetic resonance imaging. Unfortunately, delay and misdiagnosis is the norm in identifying peripheral nerve lesions of the shoulder. An understanding of the dynamic causes of winging of the scapulae in conjunction with timely electrophysiologic studies will direct physicians, in many instances, to the true nature of the illness.

## Long Thoracic Nerve of Bell

The long thoracic nerve of Bell is derived from ventral rami C5, C6, and C7. Its course runs downward and passes

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either in front of or behind the middle scalene muscle. Then, it reaches the upper slip of the serratus anterior muscle and descends along its anterior surface.

The mechanisms of injury to this nerve may be as a result of viral illnesses, repetitive trauma or stretching, general anesthesia, or surgical procedures [10,20,33]. The clinical result of this injury is winging of the scapulae without any muscular atrophy in the shoulder girdle. Most patients will have a great deal of disability due to this nerve injury. Functional loss will include the inability to lift and pull heavy objects, to play sports such as tennis or golf, and to perform tasks involving reaching above the shoulder level. This functional impairment results from the limited upward rotation of the scapulae and anterolateral positioning around the chest wall. These movements are essential to perform functions that demand elongation of the arm, such as pushing or pulling. With the loss of these three scapulae functions (i.e., fixation to the chest wall, rotation, and protraction), the upper limb will appear to hang from the scapulae, accentuating pain and cosmetic deformity.

In the absence of a normal muscle force coupling with the trapezius, the scapula will medially rotate, wing-out, and its spine will be elevated [11,16–18]. The deformity will be accentuated on forward elevation, because the trapezius alone will function as the sole rotator. With complete paralysis, there is marked weakness and inability to elevate the arm above 130 degrees.

In the majority of cases of closed injuries, recovery will be spontaneous although function may not return for up to a year. If there is no clinical or electrophysiologic evidence of recovery within 6 months to a year, surgery should be considered. However, exploration of the nerve in closed injuries is almost never warranted.

In 1979, Gregg et al. [11] reported winging of the scapula in a series of 10 young patients with traction injury to the long thoracic nerve of Bell. The average recovery time was 9 months and functional recovery was good. Foo and Swann [9] reported on 20 patients with winging of the scapula. The majority of the subjects experienced spontaneous onset of pain followed by deformity and associated loss of function. History of trauma was elicited in only three patients. Their functional recovery occurred within 2 years without any specific treatment. However, patients in both series displayed a mild degree of residual weakness [11].

Iannotti et al. [15] reported on 15 patients with injuries to the long thoracic nerve of Bell. Nine patients underwent reconstructive procedures utilizing a pectoralis major muscle transfer with a fascia lata tubular graft attached to the inferior border of the scapula. These patients were reported to have good results [9,16,18,21,29]. Icton and Harris [16] reported on 15 patients with winged scapulae who were followed for 1–16 years after surgical treatment with pectoralis major muscle transfers. The results were good in nine patients, fair in two, and poor in four. These four patients were reoperated on for avulsed fascia lata (one patient) and for scarring of the fascia lata graft (three patients). Furthermore, in this group, two patients had other

muscle paralysis. No cause could be ascertained in the other two patients.

In 1995, Post [29] reported on eight patients with 1–5-year follow-up after pectoralis major transfer for winging of the scapula. The results for these patients were excellent. They returned to work with full, pain-free, motion, but did experience slight weakness on elevation of the arm. However, it is of interest to note that in this small series, three patients were previously misdiagnosed and had four prior operations including cervical spine fusion, teres minor release for quadrilateral space syndrome, first rib resection, and an acromioplasty with lateral clavicle resection.

### Spinal Accessory Nerve (Cranial Nerve XI)

The spinal accessory nerve is a cranial nerve whose spinal portion is derived from four to five rootlets of the medulla oblongata and motor cells in the anterior gray column as low as the fifth cervical segment. The spinal portion of the nerve enters the foramen magnum and exits the jugular foramen where it travels beneath the sternomastoid muscle. This nerve proceeds superficially over the levator scapula muscle, innervating this muscle in the posterior triangle of the neck. It remains quite superficial and innervates the trapezius muscle [2,35].

Injury to the spinal accessory nerve (cranial nerve XI) is commonly due to a stab wound, surgery in the posterior triangle of the neck such as a lymph node biopsy, blunt trauma to the top of the shoulder, or to prolonged pressure on the shoulder [31,35]. The patient presents with pain and is often misdiagnosed as having a rotator cuff injury. With delay in diagnosis, secondary signs will appear such as drooping of the shoulder, asymmetry of the neckline, and winging of the scapulae. This type of scapula winging is not as prominent as that seen with long thoracic nerve injuries. Scapulae winging is accentuated with abduction, especially with elevation beyond 90 degrees [2].

This type of scapulae winging will reflect the loss of muscle force coupling with the serratus anterior [11,17]. Paralysis of the upper portion of the trapezius will cause the loss of the ability to shrug and suspend the scapula as well as rotation upward. Loss of the middle portion will cause weakness of medial adduction and rotation of the inferior angle of the scapula. Loss of the lower segment will result in an inability to maintain the scapula to the chest wall and will also compromise rotation. As a result, the scapula is depressed, droops, and is laterally rotated, but winging is minimal. The functional deficit is weakness in the forward elevation and abduction of the shoulder. The drooping of the shoulder may cause brachial plexus nerve irritation, subacromial impingement, and may result in a thoracic outlet syndrome.

The disability and disfigurement from an accessory nerve injury are not as debilitating when the nerve is severed at its high proximal location such as during a lymph node resection, as when the incision is placed distally along the lateral border of the sternocleidomastoid muscle. Some suspensory trapezius function from accessory nerve branches proximal to the injury site may be preserved.

Because the diagnosis of accessory nerve injury is difficult and often missed, electrophysiologic testing is essential [2]. Non-operative treatment is advised in closed injuries. With open injuries, however, exploration, neurolysis, and grafting may be required [31]. Ogino et al. [27] reported on a series of 10 patients with accessory nerve injury: three had transection treated by primary end-to-end repair and five had segmental loss that required cable nerve grafting. The elapsed time period from injury to repair was longer than 6 months in five of the patients. Delay in diagnosis of accessory nerve injury as a common problem was reported by Hudson [13] in 60 patients who presented with pain and shoulder dysfunction after posterior cervical lymph node biopsy. The average delay in diagnosis was 14 months. Reconstructive surgical treatment should be offered to patients who suffer from pain and disfigurement secondary to brachial plexus traction, subacromial impingement, and thoracic outlet syndrome.

Bigliani et al. [2] reported on treatment of 18 patients with trapezius paralysis, nine of which were misdiagnosed. One patient was treated successfully surgically and 10 underwent the Eden-Lange surgical procedure. The seven patients followed for 10 years had the following results: five had excellent results, one had a good result, and one had an unsatisfactory result. All patients had the deformity corrected with good function and six patients had their pain relieved.

In a recent retrospective article on seven patients with accessory nerve injuries, Nakamichi and Tachibana [24] reported on six patients who sustained lacerations of the spinal accessory nerve following cervical node biopsy, which were subsequently treated by primary repair. Four patients fully recovered and two had residual stiffness of the shoulder. The seventh patient's mechanism of injury was a gunshot wound. He was treated with neurolysis and sustained some residual stiffness of the shoulder.

### **Dorsal Scapular Nerve**

The origin of the dorsal scapular nerve is the fifth cervical nerve root. This nerve is found just proximal to the upper trunk of the brachial plexus. After piercing the scalenus medius muscle, it passes posteriorly beneath the levator scapula, which it innervates, and begins its descent to innervate the rhomboid major and minor muscles.

Injury to the dorsal scapular nerve is rare. In the differential diagnosis of medial scapulae border pain, one must consider not only C5 root cervical radiculopathy, but also dorsal scapular nerve injury. Examination of the back will demonstrate atrophy of the rhomboid major and minor muscles. Scapulae winging will be minimal at rest and may appear like winging associated with paralysis of the trapezius on static examination. With dynamic testing, the scapulae will translate laterally with minimal inferior angle rotation. Winging is more noticeable when the arm is lowered from the forward elevated position. Weakness and atrophy can further be noticed when patients place their hands on their hips and attempt to push backward against resistance.

Treatment should only be considered when patients suffer from chronic debilitating pain. Then, the Dickson fascial sling operation can be performed [6].

### **Suprascapular Nerve Palsy**

The suprascapular nerve is derived from the upper trunk of the brachial plexus and receives its fibers from C5 and C6 [1]. It contains both the sensory and motor components, but does not innervate skin. Sensory branches innervate the glenohumeral and acromioclavicular joint [1]. This nerve courses downward and laterally, deep to the omohyoid and trapezius muscles, as it runs posteriorly beneath the trapezius. It reaches the suprascapular notch along with the suprascapular artery and vein. The artery is located above the transverse scapular ligament, the nerve beneath it.

The suprascapular nerve may be entrapped at the suprascapular notch as it enters beneath the transverse scapular ligament. At this site, entrapment can be caused by a ganglionic cyst, the transverse scapular ligament, or by repetitive use of the upper extremity. Entrapment at this site will cause painful paralysis of the supraspinatus and infraspinatus muscles because the sensory branches to the posterior capsule of the glenohumeral joint arise at this site. The other site of entrapment is at the spinoglenoid notch, where the infraspinatus muscle may be singularly paralyzed. Entrapment of the spinoglenoid site can be due to ganglionic cysts [14] or by the spinoglenoid ligament. Cummins et al. [5] found that the spinoglenoid ligament occurred in 80% of their dissections of 112 shoulders in 76 cadavers.

Pain and weakness in external rotation and abduction of the glenohumeral joint accompanied by atrophy of the supraspinatus and infraspinatus muscles may misdirect one to the incorrect diagnosis of a complete rotator cuff tear [9,14]. Electrophysiologic testing will affirm the diagnosis as well as define the site of entrapment. MRI should be performed for further anatomic evaluation. When a mass lesion is identified, surgery should be performed within 6 months.

### **The Axillary Nerve**

The axillary and radial nerves originate from the posterior cord of the brachial plexus arising from the C5 and C6 nerve roots. The axillary nerve can be found immediately posterior to the coracoid process and conjoint tendon where it runs along the anterior surface of the subscapularis muscle and courses along the inferior border of the shoulder capsule. It enters the quadrilateral space with the posterior humeral circumflex artery. The axillary nerve then passes around the cervical neck of the humerus in a spiral fashion. It innervates the teres minor and then divides into anterior and posterior branches to innervate the posterior, middle, and anterior deltoid muscle. In addition, it provides sensation to the glenohumeral joint and the skin overlying the deltoid muscle.

Injury to the axillary nerve can occur close to the inferior capsule of the glenohumeral joint, where it is susceptible to direct trauma secondary to glenohumeral dislocation, lac-

eration during surgery, as well as to direct compression such as during the misuse of crutches [1,25]. Entrapment of this nerve can also occur at the quadrilateral space [4,10,28]. Injuries to the axillary nerve at the quadrilateral space can occur as a result of stretching or crushing. Although compression of the posterior humeral circumflex artery has been implicated by Cahill and Palmar [4], this remains to be proven. This diagnosis is usually made on clinical grounds. Originally, they described this syndrome in a subset of patients whose symptoms were originally attributed to thoracic outlet syndrome. These patients described poorly localized shoulder pain, paresthesias in a nondermatomal distribution, point tenderness in the quadrilateral space, and had an arteriogram demonstrating compression of the posterior circumflex humeral artery when the shoulder was abducted.

The axillary nerve palsy causing dysfunction of the deltoid muscle is a catastrophic event to the shoulder girdle. Forward elevation can be limited to 33 degrees (range of 0 to 75 degrees). With this degree of loss of motion, attempts at elevation and abduction will result in superior subluxation of the humeral head. The patient, however, may use "trick movements" to achieve full elevation of the arm by rotating the scapula, contracting the rotator cuff, long head of the biceps, and clavicular portion of the pectoralis major muscle [34]. There is no good surgical treatment to compensate for the loss of function of the deltoid muscle. Treatment of axillary nerve injuries either by closed nerve graft or neurolysis has been encouraging [3,4,10,28].

Cahill and Palmar [4] reported on 18 patients with axillary nerve entrapment, 16 of whom underwent operative decompression of the nerve at the quadrilateral space. Eight patients improved dramatically, eight patients improved markedly, and two patients showed no improvement (an 89% improvement). Francel et al. [10] presented a series of five patients with axillary nerve entrapment at the quadrilateral space treated by operative decompression. All patients resolved their sensory deficit and showed some improvement in active motion of the shoulder. In three patients, however, functional return was minimal. Petrucci et al. [28] reported on 21 cases of axillary nerve injuries treated by nerve grafting and neurolysis. Fifteen patients with transection of the axillary nerve required sural nerve grafting and six patients were treated with neurolysis alone. These patients were operated on within 6 months. The most frequent site of injury was the quadrilateral space. Of the 12 patients examined 1 year or more after surgery, 5 recovered normal function, 6 showed good recovery, and 1 displayed fair return of shoulder abduction due to axillary nerve function.

### Summary

The clinical evaluation of shoulder pain is often complicated by the wide array of possible differential diagnoses. The overlapping symptoms of multiple possible conditions make diagnosing injuries to the peripheral nerves of the brachial plexus challenging. Electrophysiologic testing is

indispensable in the diagnosis of these conditions. As a result, predictable surgical results can only be ascertained by the accuracy of the diagnosis.

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