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Review Article

Long Thoracic Nerve Palsy: When Is Decompression Indicated

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Scapular winging due to long thoracic nerve palsy can occur through traumatic injuries and non-traumatic events. The traditional view is that most patients will achieve spontaneous recovery within 2 years of winging onset. However, there is evidence that points to a less clear-cut natural history, with residual winging, muscle weakness, and fatigability being exhibited in a significant percentage of patients. Reports from proponents of a more proactive approach have shown that the surgical decompression of the long thoracic nerve beyond 12 months, through thoracic, supraclavicular, or combined approaches, can yield satisfactory results. This review examines our current understanding of long thoracic nerve palsy and explores the varying treatment strategies with their reported outcomes.

Normal scapular function is essential for optimal shoulder function. Precise scapular control allows the optimal positioning of the humerus in relation to the glenoid, thus transferring power from the core to the upper extremity. Disturbance of this function may manifest as scapular winging. It only refers to the abnormal prominence of the scapula and does not imply an etiological basis. The abnormality can be static—where the resting position is protracted compared with the contralateral side—or dynamic—where the normal smooth scapulothoracic motion is disrupted, which is known as scapular dyskinesis, scapula dysrhythmia, or scapulothoracic abnormal motion.[1–3] Scapular winging can be painful, functionally limiting, and cosmetically unsightly, which frequently requires consideration for treatment.

Scapular motion is controlled by the coordinated action of up to 17 periscapular muscles. Of these, the serratus anterior (SA) is the most commonly implicated muscle in scapular dyskinesis.[4] It is a large flat muscle, originating from the upper eighth to ninth ribs to insert on the costal surface of the medial border of the scapula. It is innervated by the long thoracic nerve (LTN). The SA works as a force couple in conjunction with the trapezius and rhomboid muscles to anchor the scapula to the chest wall stably.[2] Dysfunction of the SA causes failure of this force couple, resulting in medial winging. The dysfunction may be due to a myopathic or neurogenic process, with the latter accounting for the majority of cases.[5] Conversely, when there is a dysfunction of the trapezius or rhomboid muscles, the aberrant forces directed at the scapula are reversed, resulting in lateral winging.

Long thoracic nerve palsy may arise from direct injury to the nerve (such as thoracic trauma or surgery), traction injury, or neuralgic amyotrophy (NA). Historically, the expectation of LTN palsy as a result of closed trauma or neuritis has been of spontaneous resolution, with its natural history suggesting that symptoms do not last beyond 2 years.[6] However, more recent studies suggest that a significant proportion of patients may continue to suffer from pain and restriction beyond the expected time of recovery.[7] This group of patients may benefit from surgical treatment targeted at the LTN. Nonetheless, there is no consensus on the optimal strategy. This review examines our current understanding of LTN palsy and explores the varying treatment strategies with their reported outcomes. The authors also propose a treatment algorithm based on the literature and their clinical experience.

Anatomy and Biomechanics

The anatomy of the LTN is variable. It is a motor nerve that originates from the ventral rami of the fifth, sixth, and seventh

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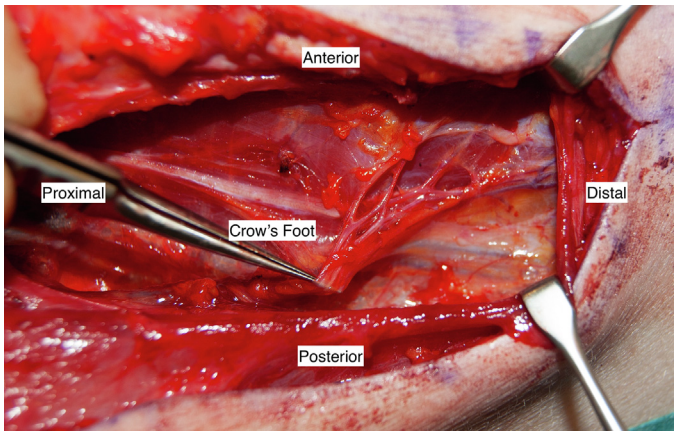

Figure 1. Clinical photograph demonstrating the crow's foot landmark.

cervical roots in the majority, but can have contributions from C4 or C8.[8] Branches can also be absent from C5 or C7 in up to 15% and 3% of cases, respectively. It averages 27 cm in length and remains superficial for most of its course.[9] In 50% of patients, the fifth and sixth roots tend to pass between the middle and posterior scalene muscles, with the remainder traveling either through the middle scalene or anterior to this muscle.[9] The seventh root always passes anterior to the middle scalene muscle and joins the C5 and C6 contributions at the level of the second rib. All contributions travel deep to the brachial plexus before passing over the first rib and descending along the lateral aspect of the chest wall as inferiorly as the ninth rib, superficial to the SA. Its sole innervation is into the SA.

Its small diameter, superficial location, and lengthy course make it vulnerable to injury. Although a few potential compression sites exist, the exact location of where the nerve is constricted, and thus, the location of surgical decompression, remains controversial. It is thought to be prone to compression as it pierces through the scalenus medius and crosses the second rib. It may also be compressed extrinsically by the complex arrangement of traversing branches of the thoracodorsal artery, known as the "crow's foot" (Fig. 1).[10] A recent clinical study has shown that 2 variations exist in the anatomy of the thoracic part of the LTN.[10] A type I LTN is more common and its distribution is classically described in the literature as a single major nerve trunk that could be observed running superficial to the SA while giving out multiple minor muscular branches along its length. A previously unrecognized type II LTN was observed in 20% of patients with LTN palsy, where the nerve splits into 2 equal major branches, at or just proximal to the crow's foot landmark, with each branch then giving off multiple minor ramified muscular branches. The authors postulated that patients with LTN palsy might be more likely to have variation in their neural anatomy than those without.

Clinical Assessment

The most common cause of scapular winging reported in the literature is medial winging due to SA dysfunction following LTN palsy.[11] When the LTN is injured, the classically described clinical sign is a scapula that assumes a position of medial rotation and superior translation of the inferior angle.[11] The medial border of the scapula becomes more prominent as the dysfunctional SA is no longer able to hold the scapula against the thoracic cage, and the degree of elevation becomes more pronounced during active movement as the muscle becomes more fatigued.

Lateral winging of the scapula is less common and attributed to trapezius dysfunction, mostly due to spinal accessory nerve palsy or rhomboid muscle paralysis due to injury of the dorsal scapular nerve. In lateral winging, the affected shoulder droops with an inferior translation of the scapula, and the inferior angle rotates laterally. This can be a subtle finding, and abducting the arm can accentuate the lateral winging.[11]

In our experience, scapular winging represents a continuum of severities. Patients at the mild end of the spectrum may only present with a slight scapular asymmetry on shoulder elevation. In contrast, those at the severe end can display noticeable winging at rest. Currently, despite there being a number of published methods examining scapular winging, there is no reliable clinical grading system to document its severity.[2] It is known that dynamic motion of the shoulder can be more sensitive in revealing scapular winging compared with static postures[12] and that elevation of the arm past the horizontal plane may be limited or impossible in patients with dyskinesis, as the scapula cannot be stabilized against the thorax.[13] Statically, winging due to SA dysfunction is expected to occur during the wall-press test as a result of loss of scapular protraction[13] and may also be present when the arms are at rest by the patient's side when it is severe.[1] Further evaluation of SA dysfunction includes the scapular assistance test, scapular reposition test, and the shoulder flexion resistance test. In the scapular assistance test, the examiner pushes superiorly and laterally on the inferomedial border of the scapula, thereby simulating the serratus anterior muscle.[1] Improvement of pain through the arc of shoulder motion and/or increased range of motion is a positive test result indicating muscle dysfunction. The scapular reposition test is performed by manually stabilizing the scapula in a retracted position on the thorax.[14] This position confers a stable base of origin for the rotator cuff, and a positive test result is indicated by an improvement in power and/or a reduction in pain reporting. The shoulder flexion resistance test is performed by positioning the shoulder passively at 30°, 60°, and 100° relative to the horizontal, then applying resistance on the forearm with the elbow in full extension. A positive test result at each interval is determined by the presence of scapular dyskinesis.[15]

In our practice, we use the Wrightington Winging Score to grade the static and dynamic components of scapular winging in a single recordable score (Table 1).[5] Both scapulae are adequately exposed to allow comparison and unhindered observation of the upper thorax. Initially, the patients are asked to stand with their arms at rest by their sides. One point is scored if scapular winging is visible at rest. Next, the patients are asked to elevate their arms forward to the horizontal plane and push against the wall with their elbows extended (wall-press test), first with their forearm in pronation and second with supination. One point is scored if winging is present for each position. Finally, the patients are asked to actively perform bilateral shoulder forward flexion, followed by shoulder abduction (in the coronal plane). One point is scored if the patient fails to achieve full forward flexion with the affected arm when compared with the contralateral side, and 1 point is scored if the patient fails to achieve full abduction with the affected arm. The total score ranges from 0 to 5, with a higher score indicating a worse winging severity. We did not include the scapular assistance, scapular reposition, or shoulder flexion resistance tests in this score to enable the Wrightington Winging Score to be applicable in both face-to-face as well as remote video consultations.

Investigations

In addition to a supportive history and clinical signs, we refer all patients for needle EMG of the SA for a confirmatory diagnosis of LTN palsy. Although there could be false-negative operator-dependent sampling errors with this test, EMG results have been documented as being supportive in up to 96% of cases with this condition.[16,17] The importance of neurophysiologic testing has also

Table 1
Wrightington Winging Score

Clinical Assessment		Criteria	Score
Static	At rest (arms by side of body)	Abnormal prominence of scapula	1
		Normal scapular position	0
	Wall-press test (shoulders at 90° forward flexion, elbows at 0° extension, and wrists at 90° extension)	Winging evident in pronation of the forearm	1
		Winging evident in supination of the forearm	1
		No scapular winging in either positions	0
Dynamic	Active shoulder forward flexion	Limited forward flexion	1
		Full forward elevation	0
	Active shoulder abduction	Limited abduction	1
		Full abduction	0
	Total (0–5)		—

been highlighted in a study by Clarke et al,[18] who demonstrated that clinical impression alone is unreliable in assessing scapular winging. Additionally, an EMG is a useful screening tool to identify potential myopathy, such as fascioscapulohumeral dystrophy. In a study of 96 patients with SA dysfunction, Ng and Wu[5] demonstrated that myopathy accounted for 12% of cases. In such cases, we would recommend referral to a neurologist for consideration of genetic testing and possible muscle biopsy.

Although ultrasound evaluation is well described in the literature for carpal tunnel and cubital tunnel syndromes, very little has been published on its application in the diagnosis of LTN lesions. A combined cadaveric and healthy human volunteer study has demonstrated that the LTN is easily visualized by high-frequency ultrasonography and has a mean diameter of 1.6 mm.[19] However, its efficacy in diagnosing LTN palsy is unknown, with only a single published letter in the literature describing an enlargement of the cross-sectional area of the nerve compared with the contralateral unaffected side.[20]

The advent of high-resolution magnetic resonance imaging has also brought hope that this could be used in the radiologic diagnosis of peripheral nerve lesions. Yet this has not been proven to be successful in LTN palsy. A retrospective study of 20 subjects investigated in a 3 Tesla scanner concluded that the LTN could not be confidently identified for any patient.[21] However, the authors noticed secondary findings of skeletal muscle denervation with decreased bulk of the serratus anterior in 20% of the subjects and stated that magnetic resonance imaging could be useful in the diagnosis of alternative causes of winging, such as concomitant brachial plexus pathology and an incidental finding of an osteochondroma of the scapula. A further published letter has supported its use in the investigation of secondary findings of LTN palsy, such as muscle edema, atrophy, and fatty degeneration.[22]

Clinicians should always remain vigilant of alternative causes mimicking scapular winging, such as labral injuries, rotator cuff tears, impingement, acromioclavicular joint injuries, cervical spine pathology, and missed fractures.[2]

Etiology of Injury

Injury to the LTN has been classically described to occur through traumatic injuries and nontraumatic events. Traumatic injuries are typically caused by a blunt force event, such as a blow to the thorax or sudden depression of the shoulder girdle resulting from a fall.[5,23] It can also occur through traction of the nerve, such as from a sudden jerk of the arm or neck.[5] Sharp trauma is normally due to iatrogenic injuries, such as radical mastectomy, first rib resection, and transaxillary sympathectomy, which accounted for 2% to 11% of cases in the larger published series.[5,23]

Nontraumatic LTN palsy is generally attributed to NA, also known as Parsonage-Turner syndrome and brachial neuritis. It is a clinical syndrome characterized by acute onset of pain followed by patchy paresis in the upper extremity.[17] It is often precipitated by an infection but may occur spontaneously. It has also been associated with exertion and exercise. A familial form, termed hereditary NA, is inherited through an autosomal dominant trait.[5]

The exact etiologic mechanism of NA remains poorly understood, with the available evidence suggesting a complex pathophysiology that includes an underlying predisposition, a susceptibility to dysfunction of the peripheral nervous system, and an autoimmune trigger for the attacks.[24] In our practice, we deemed patients to have had a traumatic LTN palsy if there was a clear, single traumatic episode before the onset of winging. Patients who developed winging spontaneously after physical exertion, viral illness, or without any precipitating events were diagnosed as having NA.[5] However, in reality, the distinction between trauma and neuritis is likely to be less clear, and they may overlap as well. A blunt force event is an eminently logical cause of trauma to the LTN, particularly in view of its thin caliber and superficial location. Nevertheless, competitive sports, vigorous exercise, and repetitive motions have all been described as instigators of NA. These activities may subject the LTN to microtrauma before precipitating an episode of neuritis, which implies a traumatic basis as a potential cause of NA.[17,24–28]

Conservative Management

On account of the rarity of LTN palsy, there is a lack of standardization in the management of this condition. The traditional view is that most patients will achieve spontaneous recovery within 2 years of winging onset.[11,29] As a result, most descriptions of winging management focus on the maintenance of shoulder motion, pain control, and activity modification with avoidance of precipitating factors. Physical therapy is generally prescribed for range of motion, scapular stabilization, rotator cuff stretching, and periscapular muscle strengthening. Although a number of specific strengthening exercises of the SA have been documented, such as the forward-punch, dynamic-hug, weight-resisted humerus elevation, and push-up plus exercises[30], no specific physiotherapy regimen for LTN palsy has been published. There are also concerns that standard shoulder therapy can potentially aggravate patient symptoms[28] or overstretch the paralyzed muscle,[29] which may lead to inferior outcomes.

Despite the historically positive outlook on this condition, evidence now points to a less clear-cut natural history of isolated serratus palsy, with residual winging, muscle weakness, and fatigue reported in 25% to 57% of patients.[6,31–33] In a study of 37 patients with milder disease who were followed up over a period of 2–31 years, Pikkarainen et al[7] found that the natural history of this condition was not as favorable as initially expected. They observed that one-fifth of the patients had long-lasting scapular winging with decreased flexion strength, one-fifth with reduced shoulder range of motion, and half had persistent pain on exertion. However, they did not identify factors that would enable clinicians to choose

patients appropriately for observation only. Foo and Swan[29] noticed a similar disease course in a series of 20 patients, with 30% experiencing residual winging at follow-up that ranged from 6 months to 12 years. However, they also did not offer advice on which factors are associated with a negative outcome. More recently, van Alfen et al[17] reported medium-term outcomes of 89 patients with winging due to NA. At a mean follow-up of 2.5 years, up to a third of the patients still experienced long-term pain and fatigue, and up to two-thirds experienced impairments in daily life. There was no correlation of pain or fatigue with the level of paresis on a Medical Research Council scale. However, patients with a comorbid condition fared worse than those without comorbidity. A follow-up study of another large cohort demonstrated similar findings, with 60% of patients with NA still complaining of pain after 6–24 months, with a similar proportion still having impaired shoulder movements, and 80% reporting difficulty in performing overhead tasks.[34] This group found that standard physical therapy consisting of rotator cuff muscle strength training was ineffective or aggravated symptoms in more than 50% of the patients. However, they also did not offer clinical advice on the associated factors that may lead to a poorer or protracted outcome.

Surgical Management

Ferry[35] argued that conservative management of scapular winging was an option based on the inadequate availability of well-reported data. This view appears to have been supported by more recent publications, with encouraging results reported by proponents of a more proactive approach involving surgery rather than observation alone.[25–27,36–39] Three main approaches to the LTN are described as follows: thoracic, supraclavicular, and combined, which are described in the following sections.

Thoracic decompression

Le Nail et al[25] postulated that when winging is caused by SA palsy alone, the LTN is compressed distally by fascial or vascular structures, whereas patients with proximal constriction of the LTN would also experience dysfunction of the rhomboids. In their study of 52 patients with isolated SA paralysis due to trauma or repetitive exertion confirmed on EMG (which included patients published in an earlier series by Laulan et al[38]), they decompressed the thoracic LTN at a median duration of 1 year from the onset of symptoms. They graded the paralysis as “complete” in 27 cases and partial in 25. Using their self-derived outcome criteria based on a combination of subjective motor and pain recovery, the results were “excellent” in 27, “good” in 18, “fair” in 4, and “poor” in 3 cases.

Maire et al[26] reported on 8 patients with LTN palsy caused by trauma, exertion, and iatrogenic injury in a single-surgeon series who underwent thoracic neurolysis. It is unclear how long the patients had paralysis before surgery, and the mean follow-up was 8 months. They found that all patients had recovered from winging by 6 months, with a mean Constant score improvement of 20. In another study of 6 patients with insidious onsets of scapular winging, all patients recovered motor function and achieved resolution of visible deformity between 6 and 12 months after surgery, although no functional outcomes were recorded.[39]

Supraclavicular decompression

Nath et al[27] reported on a study of 47 patients with LTN palsy due to trauma and NA, where they performed 50 supraclavicular decompressions of the LTN at variable durations of winging, from less than 3 months to more than 10 years. They found that 88% of their cohort “significantly improved” within 3 months but did not state the criteria used to judge the improvement. In the study population, 38 patients also had paralysis of other periscapular muscles, including the deltoid, spinati, and biceps. This suggests that the patients between the studies by Nath et al[27] and Le Nail et al[25] may be different. Nath et al’s group would appear to involve a more diffused brachial plexus lesion, which might have influenced their preference for supraclavicular decompression.

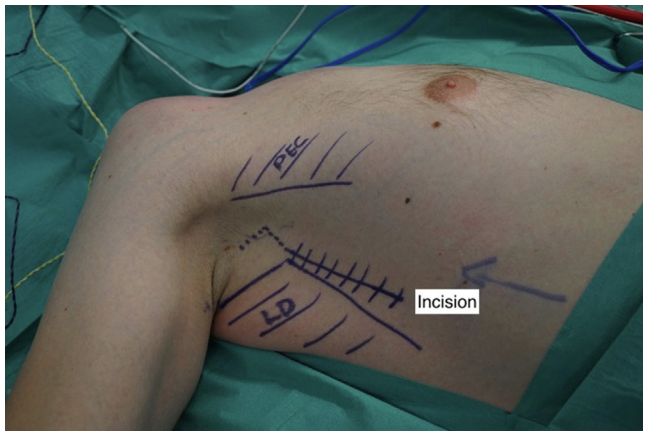

Figure 2. Clinical photograph demonstrating our surgical incision for thoracic LTN decompression. LD, latissimus dorsi; PEC, pectoralis major.

Disa et al[36] and Schippert et al[37] both described the outcomes of supraclavicular decompression of the LTN in 4 and 6 patients, respectively, for posttraumatic winging. The duration of symptoms in the 2 studies varied between 8 weeks to 2.5 years before surgery. All patients recovered by 6 months after surgery, with only 1 recurrence in the Schippert et al[37] cohort due to a later second injury to the nerve.

Combined approach

Noland et al[40] described a strategy using both the supraclavicular and thoracic approaches. If there was no contraction of the SA, a nerve transfer was also performed using the thoracodorsal or medial pectoral nerves as a donor for the LTN. Despite a detailed description of the surgical technique in 19 patients, no outcome data were reported.

The Authors’ Practice

On account of the paucity of the currently published literature on the surgical management of LTN palsy, there is a lack of standard protocols for diagnosis and postoperative outcome reporting, making the findings of these studies difficult to generalize and apply clinically. In our practice, we have observed that most patients who recover spontaneously do so in 9–12 months from the onset of winging. Therefore, we recommend an initial period of conservative management for 9–12 months before considering surgery. For those with persistent winging beyond that period, we offer LTN neurolysis. The published articles suggest that thoracic neurolysis alone is able to improve scapular winging.[25,26,37] Given its relative ease and low morbidity, our practice is to decompress the thoracic LTN initially.

Thoracic LTN neurolysis

The procedure is performed with the patient in a supine position under general anesthesia without muscle paralysis. The ipsilateral hemithorax is elevated with a sandbag under the affected

scapula to allow easy access to the axilla. The arm is abducted to 90° and placed free on an arm board. A 5–7 cm linear longitudinal incision is made over the anterior border of the latissimus dorsi (LD) muscle at the level of the fifth rib (Fig. 2). After dissecting through the subcutaneous fat, the anterior edge of the LD muscle is retracted laterally to develop the interval between the LD and SA muscles. At this point, attention is paid to identify the thoracodorsal neurovascular bundle found on the inner surface of the LD muscle. A tributary from the thoracodorsal vessel is followed to the chest wall, forming the crow's foot lesion. Underneath this leash of vessels, the silvery white LTN can be seen underneath a fascial layer. The vessel should be carefully mobilized away from the LTN, taking care to not cause any collateral damage. A combination of surgical clips and bipolar diathermy applied to the vessel may be necessary to facilitate safe mobilization. This is then followed by blunt dissection and release of the fascial layer superficial to the LTN. Proximally, at the depth of the axilla, one has to be aware of the lateral thoracic artery that can travel either superficial or deep to the LTN. A Macdonald dissector may be used to check the extent of the proximal decompression. Distally, the LTN is neurolyzed into the individual muscle slips.

After surgery, the patient begins mobilization immediately through a therapy-guided protocol. Gentle range of motion exercises without resistance are commenced in the first 3 weeks, including postural awareness and active or active-assisted shoulder movements. Between 3 to 6 weeks, we aim to strengthen scapular muscle strength, improve kinetic chain stability, and maintain proper shoulder alignment. From 6 weeks onward, the patient is encouraged to regain power and endurance of the scapular musculature and gradually initiate sporting activity.

By using the above approach, we have observed 60% complete or near-complete resolution of scapular winging, with significant improvement in their Wrightington Winging Score (unpublished data). Our results of supraclavicular LTN decompression have been mixed and less predictable, as this was only offered to those patients who failed to achieve an improved outcome following thoracic decompression as a second-stage procedure. We reserve pectoralis major tendon transfer as a later salvage option.

In conclusion, scapular winging can be debilitating, and an accurate diagnosis is key to optimal treatment. Although some patients may spontaneously recover, LTN palsy is not always self-limiting. Currently, no clinical parameter can guide us in identifying those who may not recover, and thus, may benefit from earlier intervention. The published evidence has shown that surgical decompression of the LTN beyond 12 months can still yield satisfactory results. Therefore, an initial period of conservative management for 9–12 months appears reasonable and logical. For those patients who are unresponsive to conservative measures, thoracic LTN neurolysis can then be considered.

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