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Neuropathies and Nerve Entrapments Around the Scapula and the Shoulder

Marcello Motta, MacDonald Tedah Djemetio, and Giuseppe Milano

Abstract

Nerve entrapments can occur in a number of locations throughout the body. Injury of peripheral nerves and entrapment neuropathies in the upper extremity are reported to be responsible for 2% of shoulder pain or painful instability. Nerve injury should be considered when a patient experiences pain, weakness, or paresthesia in the absence of a known bone, soft tissue, or vascular injury. The onset of symptoms may be acute or insidious. Our focus in this section will be on peripheral nerve entrapments of shoulder girdle: suprascapular nerve, the spinal accessory nerve, the long thoracic nerve, the axillary nerve, and the dorsal scapular nerve. For each nerve, we will describe the anatomy, with particular focus on the various sites of potential entrapment; then we will highlight the clinical symptoms and their associated physical examination findings; in the end we will outline the diagnostic pathway and the various possible treatment approaches.

Keywords

Shoulder neuropathy · Entrapment Syndrome Suprascapular nerve · Spinal accessory nerve Long thoracic nerve · Axillary nerve · Dorsal scapular nerve

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30.1 Introduction

Injury of peripheral nerves and entrapment neuropathies in the upper extremity are reported to be responsible for 2% of shoulder pain or painful instability [1, 2]; this frequency is likely to be underestimated, because these conditions have been overlooked in the past. The main reasons for this are an undefined clinical presentation and a lack of specific clinical signs, whatever the nerve involved.

The nerve injuries are divided into three categories: neuropraxia, axonometsis, and neurotmesis (Fig. 30.1).

- *Neuropraxia* involves a focal damage of the myelin fibers around the axons and is the least severe; in this case axon and the connective tissue sheath remain intact. This condition typically has a limited course (in most of cases, days to weeks).

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- *Axonotmesis* involves the injury of the axon itself and is more severe of the previous one; the regeneration of the nerve is possible, but for a recovery, that for most of the patients is not complete, months are needed [3].
- *Neurotmesis* involves the total disruption of the axon and the regrowth or clinical recovery has a little likelihood.

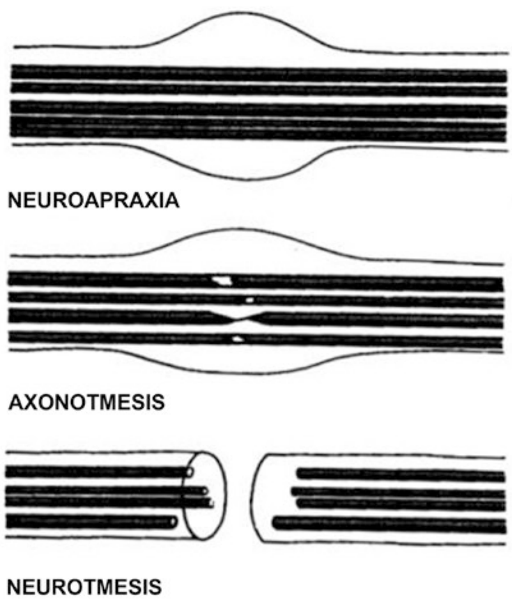

Fig. 30.1 Types of peripheral nerve injuries

The injuries that occur most often are neuropraxia or axonotmesis and the mechanism of the injury includes repetitive microtrauma, direct pressure and stretch or compression-induced ischemia.

Nerve injury should be considered when patient reports weakness, pain or paresthesia that are not related to a known soft tissue, vascular or bone injury. The onset of the symptom is often insidious or acute. The type, the location and any relationship between a symptom and specific activity should be investigated [4] (Fig. 30.2). Every evaluation of a patient with a suspect of an upper extremity nerve injury should start with looking for the presence of a radial pulse, and sensation and movement in the digits; in the case there are no obvious neurovascular compromise, the history of the patient

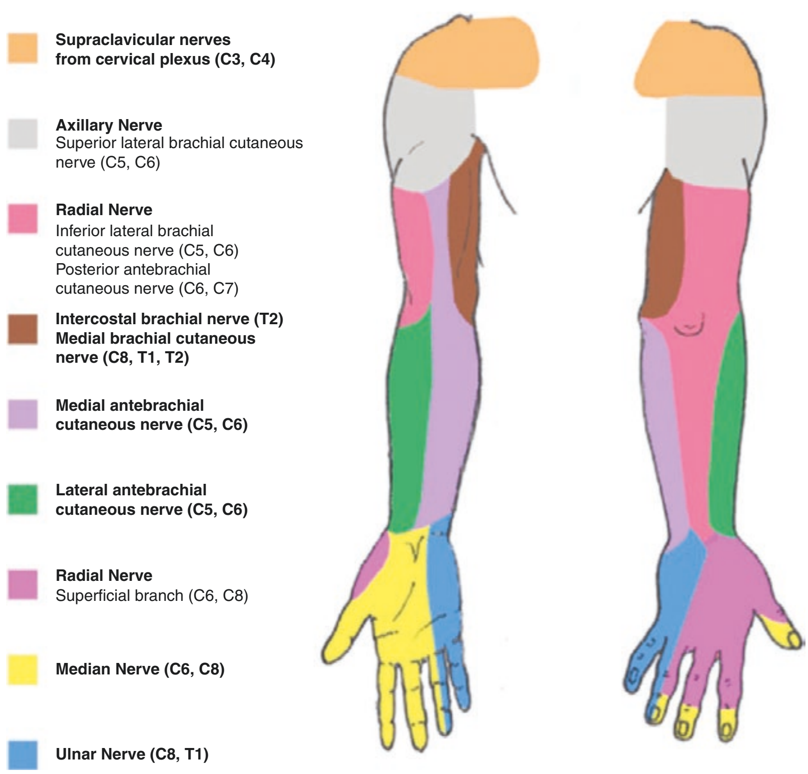

Fig. 30.2 Anterior and posterior view of the upper limb cutaneous innervation (From*: Casal D, Cunha T, Pais D, Iria I, Angélica-Almeida M, Millan G, Videira-Castro J, Goyri-O'Neill J. A stab wound to the axilla illustrating the importance of brachial plexus anatomy in an emergency context: a case report. J Med Case Rep. 2017 Jan 4;11(1):6.* https://doi.org/10.1186/s13256-016-1162-6. Distributed under the terms of the Creative Commons Attribution 4.0 International License (CC BY 4.0): http://creativecommons.org/licenses/by/4.0/)

is the key to make a diagnosis [5]. The examination follows the classical pattern of inspection, palpation, joint range of motion (ROM), muscle strength testing and sensory and neurologic examination. It is mandatory a full understanding of the nerve commonly involved, their function and the area of the body in which they have and increased risk of compression or entrapment. Radiographs are useful to identify other conditions that can mimic peripheral nerve injuries, like fractures or cervical spondyloarthropathy. Ultrasonography is a less expensive modality to define anatomic entrapment, but its use is limited by lack of standardization of technique and interpretation.

Chronic nerve injury can lead to denervation changes in muscle, and these could be visible on magnetic resonance imaging (MRI) as abnormal signal patterns; whatever normal MRI findings do not rule out nerve injuries [6].

Electrodiagnostic testing consists of nerve conduction studies and electromyography (EMG). Nerve conduction studies assess the integrity of sensory and motor nerves. Areas of nerve injury of demyelination appear as slowing of conduction velocity along the nerve segment affected [7]. EMG records the electrical activity of a muscle from a needle placed into the muscle, looking for signs of denervation. The combination of nerve conduction studies and EMG can help distinguish peripheral form central nerve injuries [8].

The initial management of most nerve injuries is non-surgical. The main components of treatment are relative rest and protection of the injured area. Anti-inflammatory medications are often added, although they are unknown if they aid healing. Mobility of related joint should be maintained, and effort should be made to increase the strength of any supporting or accessory muscle [9].

Operative treatment, that is more than often required when conservative treatment has been unsuccessful, consists of surgical decompression of the nerve injured that can be performed by an open or an arthroscopic approach.

30.2 Suprascapular Nerve Entrapment

Suprascapular nerve (SSN) entrapment has become a recognized cause of posterolateral and superior shoulder pain [10, 11]. The condition has been generally considered as rare cause of shoulder weakness and pain [12, 13], but it has been diagnosed more frequently thanks to the recent advancements in the discover of anatomic variants and in the progress of imaging techniques and electrodiagnostic studies.

The real prevalence and incidence of this disease is unknown; some authors suggest that 1–2% of all shoulder pain are caused by an SSN entrapment [14]. Nowadays the condition is being increasingly reported in athletes in which overhead and throwing activities are made repetitively (swimmers, volleyballers, weight lifters, baseballers, and tennis players) [15, 16].

30.2.1 Anatomy

The SSN is a mixed motor and sensory nerve that has origins from the upper trunk of the brachial plexus. The SSN arises from the fibers of the fifth, sixth cervical nerve roots and in 15–22% of the cases from the fourth cervical nerve root [17]. It spreads from the upper trunk of the brachial plexus and runs across the posterior cervical triangle, then under the coracoclavicular ligament and trapezius muscle, and then posteriorly to pass through the suprascapular notch or incisura scapulae. The superior portion of the suprascapular notch is delimited by the transverse scapular ligament that has variable thickness and can become completely or partially ossified [18–20], factor that could lead to entrapment neuropathy. The suprascapular notch has a highly variable shape and has been classified into six types [18]. After the suprascapular notch, the nerve travels underneath the supraspinatus muscle and provides its motor innervation. The SSN is responsible, up to 70%, of the sensory innervation of the shoulder [21]. The SSN then passes inferolaterally toward

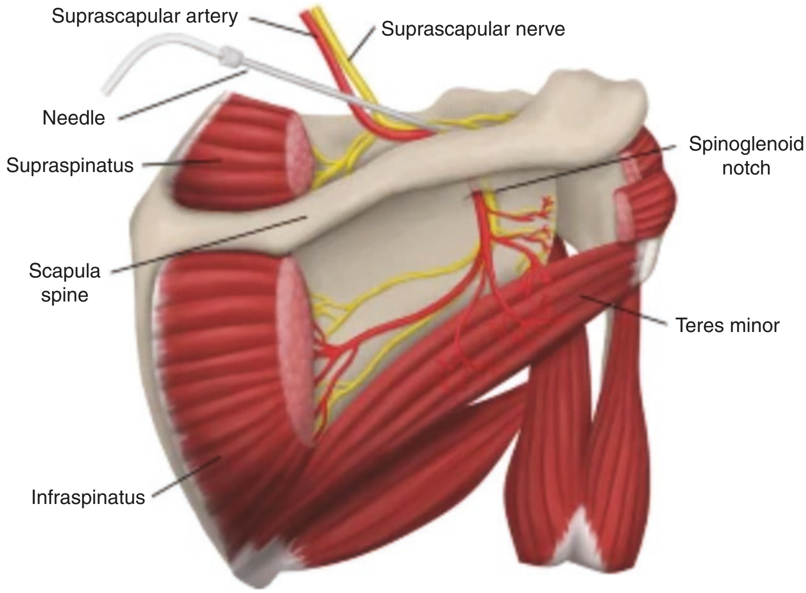

Fig. 30.3 Anatomy of suprascapular nerve (Reproduced with permission from*: Shah, J.M., Pellis, Z., Provenzano, D.A. (2020). Suprascapular Nerve. In: Peng, P., Finlayson, R., Lee, S., Bhatia, A. (eds) Ultrasound for Interventional Pain Management. Springer, Cham.* https://doi.org/10.1007/978-3-030-18371-4_4*)*

the rim of the glenoid fossa; there it turns around the lateral edge of the scapular spine, under the spinoglenoid ligament that forms a fibro-osseous tunnel, or the spinoglenoid notch, through which the nerve arrives in the infraspinatus fossa; before the spinoglenoid ligament there is the origin of the deep sensory fibers direct to the shoulder joint [22]. In the infraspinatus fossa the nerve supplies the motor innervation to the infraspinatus muscle (Fig. 30.3).

30.2.2 Etiology and Pathomechanics

There are two distinct potential sites of entrapment along the course of SSN: suprascapular notch and spinoglenoid notch. The most common site of its entrapment is at the suprascapular notch [23].

The entrapment at the suprascapular notch causes poorly localized posterolateral shoulder pain and weakness. The entrapment at the spinoglenoid notch is related to atrophy and weakness of infraspinatus muscle [24]. The possible causes for SSN entrapment can be divided into:

- *Primary causes:* primary or idiopathic causes are related to the anatomy and composition of the suprascapular notch [18] (height of foramen, configuration of the superior transverse ligament and partial or complete ossification of this ligament [25]) and the spinoglenoid notch.
- *Secondary causes:* space-occupying lesions [26], paralabral cyst [25, 27] associated with paralabral pathology, direct trauma with glenohumeral dislocation, fractures, penetrating injuries, repetitive overhead sports or activities [15, 16] and viral neuritis resulting in Parsonage-Turner Syndrome [28].

30.2.3 Clinical Diagnosis

Symptoms and signs are related to the location of nerve entrapment. The entrapment at the suprascapular notch results in relevant shoulder pain provoked by compression of the deep sensory fibers which innervate the glenohumeral and acromioclavicular joints [22, 23]; the pain is localized in the posterolateral area of the shoulder and scapula and described as a dull ache that can radiate into the ipsilateral part of neck and arm; this clinical presentation is often seen in people that perform repetitive overhead motions (especially in athletes like tennis players, baseballers, weight lifters and swimmers or carpenters). The onset of this pain is insidious and develops in the dominant arm. Shoulder weakness and fatigue are symptoms often seen in patients, especially in abduction and external rotation of the arm.

The entrapment at the spinoglenoid notch is associated with weakness and atrophy of the infraspinatus muscle; pain is absent, because the deep sensory fibers that are direct to the shoulder joint exit proximally to this entrapment site [22, 24].

The diagnosis of neuropathy of the SSN is still an exclusion diagnosis, but the increasing number of reports in the literature is helping the clinicians to find it out sooner and to rule out other possible causes [29]. The history of patients may unveil more than 6 months of posterolateral or superior shoulder pain treated unsuccessfully and/or painless muscle wasting with external rotation weakness.

The physical examination of the patient with suspected SSN entrapment should begin by checking the supraspinatus or infraspinatus muscle for atrophy; this finding is most easily seen in the infraspinatus muscle, because the supraspinatus is normally obscured by the trapezius muscle; the combined muscle wasting of the supraspinatus and/or infraspinatus is typical of the entrapment at the suprascapular notch, while isolated muscle wasting of infraspinatus is caused by entrapment at the spinoglenoid notch (Fig. 30.4).

Fig. 30.4 Atrophy of the infraspinatus muscle (arrow)

At first should be evaluated the active ROM of the shoulder, by comparing it to the contralateral side and searching for limitation in external rotation with the arm held in 90° of abduction or in adduction. Then the suprascapular notch has to be palpated to elicit pain [22]; in order to find the exact position of the notch the clinician needs to put his hand on the patient's affected shoulder with the fingertips on the clavicle, then presses the thumb along the distal third of the scapular spine.

The pain at the suprascapular notch can be shown with the *suprascapular nerve stretch test* [30]: the examiner is placed behind the patient and with one hand he turns the head of the patient to the contralateral side of the affected shoulder in a gentle motion while retracting the involved shoulder backwards with other hand; if the test elicit or exacerbate pain is positive.

The position of the spinoglenoid notch may be found, following the inferior border of the scapular spine, 4 cm medially from the posterolateral corner of the acromion. The test for the spinoglenoid notch is the c*ross-body adduction test* [31], which consists of forcibly adduction of the arm across the chest, so that the spinoglenoid ligament can be tightened in order to compress the nerve and to reproduce the patient's symptoms (Fig. 30.5).

30.2.4 Instrumental Diagnosis

In the patients that show history and/or symptoms and signs suggestive of suprascapular neuropathy further diagnostic testing should be performed.

Standard radiographs of the scapula and shoulder are important to assess the presence of fracture, osseous dysplasia, osseous variants of the suprascapular notch and bone tumors. In addition to standard X-ray of the shoulder, a suprascapular notch view permits evaluation of notch variants, while a stryker notch view will show the suprascapular notch.

Computed tomography (CT) scans may better assess fractures and anatomy, but it is not so useful for patients that suffer from suprascapular neuropathy; the only case in which CT may be of

Fig. 30.5 (**a**) Suprascapular nerve stretch test: the examiner is placed behind the patient and with one hand he turns the head of the patient to the contralateral side of the affected shoulder in a gentle motion while retracting the involved shoulder backwards with other hand. (**b**) Cross body adduction test: forcibly adduction of the arm across the chest

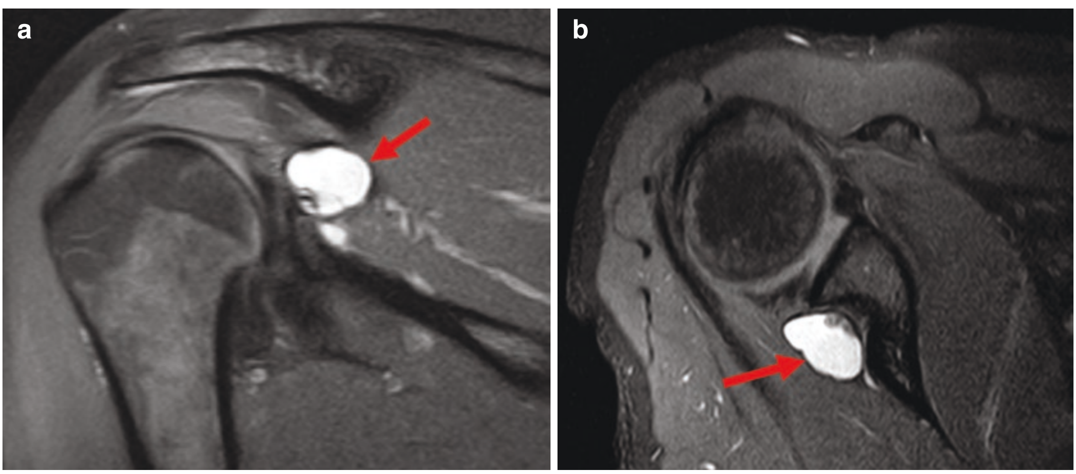

Fig. 30.6 MRI of a right shoulder showing a spinoglenoid notch ganglion cyst (arrow). (**a**) Coronal view, PD-weighted sequence. (**b**) Axial view, PD-weighted sequence

high diagnostic value is when the transverse scapular ligament is ossified.

MRI allows optimal evaluation of the labrum, rotator cuff tendons and fatty infiltration and atrophy of muscles [32] (Fig. 30.6).

Electrodiagnostic studies, including EMG and nerve conduction velocity studies, are the gold standard for the diagnosis and confirmation for suprascapular neuropathy. The indications for this test include unexplained persistent shoulder pain, weakness, and atrophy not associated with a rotator cuff tear, or MRI that shows fatty infiltration and/or edema of rotator cuff muscle without a rotator cuff tear [33, 34]. EMG may show denervation of the infraspinatus or supraspinatus muscle with resultant sharp waves and fibrillations; conduction velocity studies asses a latency value from the Erb's point to the supraspinatus and infraspinatus muscle and a latency between the muscle. Diagnostic accuracy of EMG for this condition is 91%, while for nerve conduction velocity is between 72% and 79% [35].

30.2.5 Differential Diagnosis

The differential diagnosis of neuropathy of the SSN includes the following condition: cervical disk disease, posterior cord lesion of brachial plexus, thoracic outlet syndrome, biceps tendinitis, degenerative arthritis of the acromioclavicular joint, glenohumeral instability, labrum pathology, degenerative arthritis of the glenohumeral join, quadrilateral space syndrome, brachial plexitis and rotator cuff tendinitis or tear.

30.2.6 Treatment

The initial approach for most isolated SSN lesions, not due to a space-occupying lesion or a rotator cuff tear, is a nonoperative treatment that includes activity modification, physiotherapy, and non-steroidal anti-inflammatory drugs (NSAIDs). Overhead movements and activities should be avoided, and the physiotherapy should focus on motion of the affected shoulder and strengthening of muscles. Most of the authors agree that an initial nonoperative treatment, in the absence of a space-occupying lesion, is prudent and can result in the reduction or resolution of weakness and pain, albeit the success rate is still unclear. In fact, some authors suggested that operative treatment should be adopted as soon as suprascapular neuropathy is diagnosed in order to prevent the increase of muscle damage [36].

When the cause of neuropathy is the compression on the nerve by mass or a cyst operative treatment should be considered. The choice of operative treatment is patient-based and depends on various aspects: signs and symptoms, location of the lesion and electrodiagnostic findings. Surgical decompression of the SSN has to be considered in the patients in which there is a relevant muscle wasting, weakness or severe pain and there was a failure of conservative therapy.

When compression is at the level of the suprascapular notch, operative treatment often consists in the release of the transverse scapular ligament with decompression or excision of any associated mass; this procedure may be undertaken through an arthroscopic or open technique. The open technique starts usually with a transverse incision cephalad to scapular spine or through a vertical incision of 5 cm from the posterolateral edge of acromion; then the trapezius is elevated, the supraspinatus is reflected and dissection is carried down to suprascapular notch in the posterior part; so the transverse scapular ligament can be identified and released, paying attention not to damage the overlying vascular structures. The arthroscopic technique described by Lafosse et al. [36], consists in the use of a SSN portal, which is located between the scapular spine and the clavicle, approximately 7 cm medial to the lateral border of the acromion; the transverse scapular ligament is identified as the medial continuity of the conoid ligament above the suprascapular notch. Correct placement of the SSN portal is guided by the use of a spinal needle introduced orthogonal to the suprascapular fossa and anterior toward the transverse scapular ligament; then a trocar is placed through the suprascapular nerve portal and positioned lateral to the SSN within the notch for protection. The second portal is placed 2 cm lateral to the SSN portal and permits the introduction of arthroscopic scissors for the release of the transverse scapular ligament.

At the level of spinoglenoid notch, operative treatment is in most of cases associated with a space-occupying lesion: the majority of lesions includes cysts, lipomas or other benign tumors of this region. The management of lipoma or benign tumors requires a resection of the lesion with an open approach. The treatment for cysts is controversial and may be done through an open or athroscopic approach: in the arthroscopic approach the posteromedial and posterolaterals portals in the infraspinatus fossa or subacromial approach are used, while for the open approach the posterior approach is used.

30.3 Spinal Accessory Nerve Entrapment

The spinal accessory nerve (SAN) is a mixed sensory-motor nerve [37] that can be trapped or damaged due to its superficial course in the neck. The causes that can determine these conditions are varied. SAN paralysis is manifested by an isolated weakness and atrophy of the sternocleidomastoid (SCM) muscle and trapezius muscle ("dropped shoulder syndrome"), as well as pain in the shoulder and neck [38]. In fact, the trapezius muscle represents one of the main stabilizing muscles of the shoulder girdle. Its dysfunction causes a loss of stability of the scapula with a functional deficit in the remaining stabilizing muscles. In the diagnosis the help provided by electrodiagnostic and imaging techniques is fundamental. Treatment of this condition can be either conservative or surgical.

30.3.1 Anatomy

The SAN is the 11th cranial nerve and it consists of two parts: spinal and cranial. The cranial root begins in the nucleus ambiguous in the medulla. The spinal root originates from a cluster of motor neuron cell bodies in the accessory nucleus, located in the lateral part of the anterior horn of the first five segments of the spinal cord. Therefore, these fibers head cranially through the foramen magnum, where they join the cranial fibers [38]. After leaving the skull via the jugular foramen, the spinal accessory nerve penetrates to the deep surface of the SCM muscle, entering the posterior triangle of the neck. The spinal accessory nerve is superficial throughout this course and is associated with a chain of five to ten lymph nodes [39]. The superficial location of this nerve, along with its close association with lymph nodes place it at risk during surgical dissection of the posterior triangle of the neck during procedures such as lymph node biopsies [40, 41] (Fig. 30.7).

The SAN innervates the trapezius and SCM muscles, even if some studies have documented innervation of the upper trapezius directly from

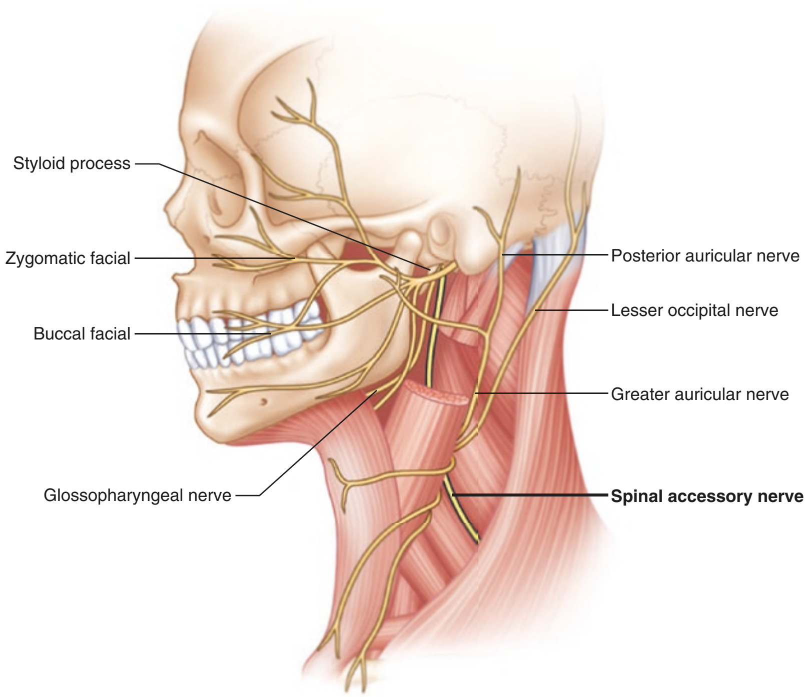

Fig. 30.7 Anatomy of spinal accessory nerve (SAN). The nerve penetrates to the deep surface of the SCM muscle, entering the posterior triangle of the neck, and then innervates the trapezius muscle. The SAN is superficial throughout this course (Reproduced with permission from *Stogicza, A.R. (2016). Spinal Accessory Nerve Entrapment. In: Trescot, A.M. (eds) Peripheral Nerve Entrapments. Springer, Cham.* https://doi.org/10.1007/978-3-319-27482-9_27)

the cervical plexus [42]. The SAN has connections to other nerves (hypoglossal nerve, the stellate ganglion, the mandibular branch of the facial nerve, accessory phrenic nerve, great or posterior auricular nerve and the brachial plexus), and this could be the cause of the variable symptoms and signs related to this injury [38].

The trapezius is a very large muscle that has an especially broad origin spanning the external occipital protuberance, the medial nuchal line, and the spinous processes of vertebrae C7 through T12. The superior fibers of this muscle insert onto the posterior aspect of the clavicle, the medial fibers insert onto the medial aspect of the acromion, and the inferior fibers converge onto an aponeurosis that inserts onto the spine of the scapula. The superior and inferior fibers of the trapezius elevate and rotate the scapula laterally. This action moves the glenoid upward to accommodate shoulder abduction. The middle fibers stabilize the scapula during its range of movement (ROM). In general, this muscle acts to elevate, retract, rotate, and depress the scapula, depending on which portion of the muscle is activated.

30.3.2 Etiology and Pathomechanics

There can be several causes that lead to an injury or entrapment of the SAN. The most frequent cause is usually an iatrogenic lesion secondary to surgical procedures involving the posterior triangle of the neck, an ideal access point for many surgeons to perform a lymph node biopsy, tumor resection, or selective neck dissection [43–45]. Even postoperative scarring can lead to entrapment symptoms. Many traumatic mechanisms of injury have been reported, including flexion-extension injuries, falls, sport injuries like during water-skiing or even deep tissue massage [46–48]. Moreover, neuritis provoked by varicella infection, motoneuron disorders or other neurologic pathology can lead to a SAN palsy [49].

SAN injury causes paresis of both SCM and trapezius muscles on the ipsilateral side and unopposed contraction of the muscles on the contralateral side, leading to torticollis [38].

30.3.3 Clinical Diagnosis

Patients with a SAN lesion complain of ipsilateral neck and shoulder pain sometimes irradiated to the ipsilateral arm, with limitation of ROM to the elevation and abduction of the affected limb at the level of the shoulder (“droopy shoulder”) and torticollis [50, 51]. A common finding is a subclavicular/pectoral asymmetry [52], and asymmetric neckline, with trapezius muscle atrophy and disruption of the normal scapulothoracic rhythm and medial scapular winging, especially when the shoulder is actively abducted or externally rotated [53]. As reported by some authors, the active elevation lag sign and the triangle sign (Fig. 30.8) can detect a trapezius dysfunction and can be useful to distinguish between scapular winging from SAN injury, and the more common cause of winging, like long thoracic nerve injury [53, 54].

The SAN lesion may be accompanied by a concomitant lesion of the great auricular nerve and this would explain the sensory alteration sometimes reported by patients over the angle of the jaw, the ear, the shoulder and the chest. Sometimes patients do not complain of shoulder pain. This would appear to be related to the compensatory hypertrophy of the levator scapulae muscle on the ipsilateral side. Conversely, sometimes the weakness of the SCM and the trapezius can cause traction on the brachial plexus, resulting in a painful shoulder syndrome with an eventual frozen shoulder (adhesive capsulitis) [39].

30.3.4 Instrumental Diagnosis

Diagnostic exams such as CT or MRI can help to identify causes of entrapment (i.e., neoforma-

Fig. 30.8 (**a**) Left trapezius palsy (arrow) trapezius palsy. (**b**) Elevation lag sign. The patient is standing and asked to forward elevate the normal shoulder to its maximum then the affected one. The difference in forward elevation between the two sides, known as the active elevation lag, is characteristic of trapezius dysfunction because the acromion, in this condition, cannot elevate. (**c**) Triangle sign. The test is performed with the patient lying prone with both arms elevated to approximately 120°. In the presence of trapezius weakness, the patient is unable to elevate the arm in this plane, producing a lag or a "triangle sign"

tions or postoperative scarring) or to evaluate the degree of muscle atrophy of the SCM and trapezius. They also can exclude the absence of medullary and root disorders, absence of basilar impression or any shoulder disorders. Nerve conduction like needle EMG studies may also show fibrillation potentials and/or positive sharp waves, typical signs of axonal injury to the nerve, with reduced or absent volitional recruitment of motor unit action potentials (MUAPs) within the trapezius and/or SCM. However, reduced compound motor action potential (CMAP) can be present, due to the possible innervation by the cervical plexus of the upper portion of the trapezius [54].

30.3.5 Differential Diagnosis

The differential diagnosis includes paralysis of the rhomboid and serratus anterior muscles, suprascapular nerve injury, herniated nucleus pulposus of cervical disks, progressive neuromuscular disease, scapular osteochondroma, herpes zoster or other infections, thoracic outlet syndrome and Parsonage-Turner syndrome.

30.3.6 Treatment

Establishing the etiology of the lesion is essential for correct treatment. In most cases the SAN experiences a neuroapraxic injury, such as in blunt or stretching trauma. This condition resolves from 6 to 9 months. An EMG of the damaged nerve should be performed 6 weeks after index injury, then repeating the EMG every 3–6 months to evaluate nerve function and recovery. Conservative treatment consists of a rehabilitation program for the maintenance of shoulder ROM, together with the strengthening of the medial stabilizing muscles of the scapula, such as rhomboids [55]. An adequate exercise allows a good functional recovery, although nonoperative treatment options have low success rate [55, 56]. Also, analgesic therapy plays a fundamental role in SAN lesions given the frequent presence of neuropathic pain to slow down the rehabilitative recovery.

Typical neuropathic pain medications are gabapentin, pregabalin, tricyclic antidepressants, and duloxetine. The success of nerve blocks and use of stronger narcotics is not well studied in the literature [57]. Nevertheless, nonoperative treatment options for trapezius palsy have lower success rates compared with serratus anterior palsy [55, 56].

Surgical procedures can be divided into early procedures (within days to months of the nerve injury) and late procedures [58, 59]. If there is a well-founded suspicion of traumatic nerve injury or compression, early operative exploration of the nerve with potential graft or neurolysis is indicated, with a favorable outcome if performed within 20 months of index injury [60, 61]. Late surgical procedures focus on restoring shoulder function. Dynamic transfer of the elevator scapulae and rhomboid minor with the Eden-Lange procedure is the preferred operative treatment for trapezius palsy also being able to correct the scapular winging [39], with satisfactory results [39, 59, 60].

30.4 Long Thoracic Nerve Entrapment

The long thoracic nerve (LTN) entrapment is a condition that can occur in several sites of its course and for many causes. It determines a "medial scapular winging," a dysfunction that causes a loss of stability of the scapula with a functional deficit in the remaining stabilizing muscles. An injury to this nerve causes pain and weakness with severe disability for the patient. Electrodiagnostic and imaging techniques are fundamental in making a diagnosis. Treatment of this condition can be either conservative or surgical.

30.4.1 Anatomy

The LTN is a long and superficial nervous structure. It comes from the ventral branches of the nerve roots C5–C7 and occasionally C8. Portions of C5–C6 usually cross the scalene muscle, while the contribution of C7 passes between the anterior and middle scalenes. Distally the nerves join to form the LTN, which descends along the chest wall. The LTN innervates the serratus anterior [62].

The serratus anterior is a large fan-shaped muscle that can be divided into three parts [63], each of them with a peculiar thoracic and scapular insertion. The upper part originates from the first and second ribs and fits on the upper corner of the scapula; the central part instead originates from the second and third ribs and is inserted on the medial edge of the scapula; the lower part originates from the lower ribs to the third rib and fits on the lower corner of the scapula. The lower portion allows the abduction and external rotation of the scapula, while the upper part antagonizes this action. The central part protracts the shoulder blade. Globally, these muscle bellies stabilize the medial edge of the scapula to the chest wall to allow optimizing the position of the glenoid for upper extremity function [64] (Fig. 30.9).

30.4.2 Etiology and Pathomechanics

The LTN can be trapped in several places along its course. It is vulnerable to damage at different levels. In fact, the superficial course along the lateral thoracic wall places the long thoracic nerve at risk of compression or contusion [65, 66]. The nature of this entrapment is generally mechanical

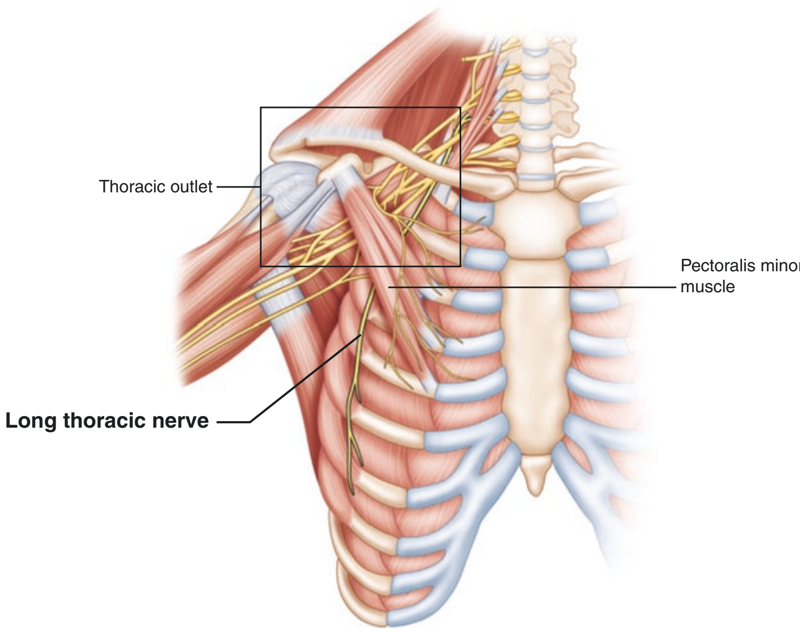

Fig. 30.9 Anatomy of long thoracic nerve and serratus anterior muscle (Reproduced with permission from *Olamikan, S., Karl, H.W. (2016). Long Thoracic Nerve Entrapment. In: Trescot, A.M. (eds) Peripheral Nerve Entrapments. Springer, Cham.* https://doi.org/10.1007/978-3-319-27482-9_30)

(direct compression or excessive traction). This may occur following motor vehicle accidents, falls from a height, and sports accidents involving sudden shoulder depression and twisting of the neck. Repetitive activities with the head tilted away from the nerve and the arm overhead—as occurs in baseball pitchers, javelin throwers, and tennis servers—may place the long thoracic nerve on stretch. Similarly, upper extremity overuse in industrial laborers and homemakers may also contribute to serratus anterior muscle palsy [57, 67].

The areas of possible entrapment are: (1) within the middle scalene muscle or between the middle and the posterior scalene [62, 68–71]; (2) between the clavicle and the second rib [57]; (3) between the coracoid and the second rib [71]; (4) between the lower surface of the scapula and the second rib [72]; (5) at the emergence of the LTN from the axillary sheath or fascial sling [73, 74]; (6) by a thoracodorsal artery branch where it crosses the LTN [73, 75]; (7) in the half-axillary line [76]; (8) anterior to the lower corner of the scapula [77, 78].

The nerve can also be injured due to a penetrating wound or following medical or surgical procedures. In fact, it has been described how this nerve can be anesthetized or damaged during brachial plexus blockages [79], or accidentally injured during surgery (i.e., mastectomy or axillary lymph node dissection).

The LTN is also the most common nerve involved with brachial plexitis. Conditions like Guillain-Barré syndrome, Arnold-Chiari malformation, systemic lupus erythematosus, viral illness, and Lyme's disease have been implicated in serratus anterior palsy [67, 80]. LTN palsy has also been reported following abnormal arm positioning during sleep [81] as well as post-partum or following infections [82]. In other cases, it is not possible to trace the real cause. Cases where there was an associated paralysis of LTN and SAN have rarely been described [83–86].

30.4.3 Clinical Diagnosis

In the initial evaluation of the patient, it is important to have a clear description of hand dominance, occupation, medical history, and surgical history of the ipsilateral shoulder, cervical spine, and breast [56, 57, 72]. Often patients complain about shoulder pain associated with stiffness and weakness. This symptomatology

Fig. 30.10 Serratus anterior palsy due to injury of the long thoracic nerve. (**a**) Medial winged scapula occurs for upper translation and medial rotation of the lower angle of the scapula. (**b**) Scapular winging is best viewed by having the patient push his or her hands against the wall (wall push-up test)

can radiate distally to the arm or proximally to the paraspinous cervical area [72, 87]. The symptoms are exacerbated with overhead activity. In some patients, prolonged driving or sitting against a hard surface can cause some discomfort to the posterior area of the shoulder [39]. As previously reported, this symptomatology is linked to an injury or entrapment of the LTN with subsequent weakness of the anterior serratus. In advanced cases this can lead to complete paralysis of the muscle. This may result in a medial-winged scapula for upper translation and medial rotation of the lower angle of the scapula [67, 88]. The scapula becomes unstable, and a chronic weakness of the stabilizing muscles can cause pain, muscle disorders or decreased shoulder abduction, neck and upper back pain [80]. Moreover, glenohumeral pathology can mimic symptoms of scapular winging, but its symptoms are exacerbated with forward elevation and abduction. The wall push-up test can be used to detect serratus anterior muscle dysfunction [88] (Fig. 30.10).

30.4.4 Instrumental Diagnosis

Standard radiographs of the chest, cervical spine, shoulder and scapula can help to identify osseous anatomic abnormalities such as fractures, accessory ribs, mass lesions, or even osteochondromas [39]. These pathologies can be further investigated by CT or MRI. Moreover, both CT and MRI can help to rule out other causes of shoulder pain. MRI can also show signs of acute or chronic denervation of the serratus anterior, such as muscle edema with homogeneous T1- hypointense and T2 STIR- hyperintense muscle signals, muscular atrophy, and fatty degeneration with fasciculated muscle areas in T1- hyperintense and T2 STIR- hypointense signal [89]. EMG can confirm the diagnosis and is helpful in distinguishing any neuromuscular cause of winged scapula [67]. Repeating this test at 3- and 6-month intervals can be useful to evaluate patient improvement.

30.4.5 Treatment

Establishing the etiology of the lesion is essential for correct treatment. If there is a well-founded suspicion of traumatic nerve injury or compression, operative exploration of the nerve with potential graft or neurolysis is indicated, with a favorable outcome if performed within 20 months from index injury. There are three main approaches to the long thoracic nerve: the distal axillary/thoracic approach, proximal or supraclavicular approach, and the two-level proximal and distal approach [90].

In most cases the scapular winging is the result of neuroapraxic injury, such as in blunt or stretching trauma. This condition resolves from 6 to 9 months. An EMG of the damaged nerve should be performed 6 weeks after index injury, then repeating the EMG every three to 6 months to evaluate nerve function and recovery. Furthermore, once the diagnosis is made, it is necessary to start non-surgical therapies to maintain shoulder movement and prevent stiffness. A scapular brace may also be used, as it can improve scapulothoracic kinematics [39]. Some authors recommend caution when using EMG findings to predict prognosis and to guide the timing for surgical management [91], given the difficulty of evaluating electromyographically this muscle.

Noland et al. [90] proposed an algorithm for the treatment of isolated LTN paralysis based on intraoperative nerve stimulation. If stimulation of the nerve causes a contraction of the serratus anterior, a serial examination is recommended for at least 3 months for recovery of serratus anterior function. If no recovery happens, subsequent decompression of the LTN from an anterior axillary line incision with intraoperative stimulation and thoracodorsal end-to-side to LTN transfer is performed. Where no improvement in serratus anterior function is observed with supraclavicular decompression and intraoperative nerve stimulation, they recommend immediate decompression through an anterior axillary line incision with intraoperative stimulation. If adequate activation of the serratus anterior is achieved, an end to-side thoracodorsal to LTN transfer is performed to supercharge the reinnervation of the serratus anterior. If no serratus anterior function is observed, they recommend two nerve transfers to be performed simultaneously: the medial pectoral to LTN transfer to reinnervate the superior muscle slips, and a second thoracodorsal to LTN end-to-end transfer to reinnervate the inferior muscle slips [90].

After failure of 12–24 months of conservative management and no substantial improvement as demonstrated by the EMG, dynamic muscle transfer surgery should be considered. Dynamic transfer of the pectoralis major tendon to the inferior angle of the scapula is the preferred operation for treating LTN and serratus anterior deficiency, with a success rate range from 74% to 100% [39, 72, 88, 91–94].

30.5 Axillary Nerve Entrapment

Axillary nerve entrapment is also known as quadrilateral space syndrome (QSS), which is a neurovascular syndrome characterized by the compression or the mechanical injury of the axillary nerve or one of his major branches and/or the posterior circumflex humeral artery (PCHA) in the quadrilateral space [95]. It was first described by Cahill and Palmer in 1983, who were investigating the poor results of surgical decompression for thoracic outlet syndrome [95, 96]. "This rare syndrome led to poorly localized shoulder pain, discrete tenderness to palpation over the QS, and possible teres minor and deltoid denervation. These symptoms are exacerbated by abduction and external rotation or forward flexion of the shoulder" [97]. The diagnosis is difficult and requires an accurate assessment.

30.5.1 Anatomy

The quadrilateral space or Velpeau's space is located over the posterior scapular and subdeltoid region. It is bounded superiorly by the teres minor or the scapulohumeral capsule, inferiorly by the teres major and latissimus dorsi muscles, medially by the long head of the triceps and laterally by the humeral shaft. This space contains two major structures: the PCHA and the axillary nerve (Fig. 30.11). The axillary nerve (a motor-

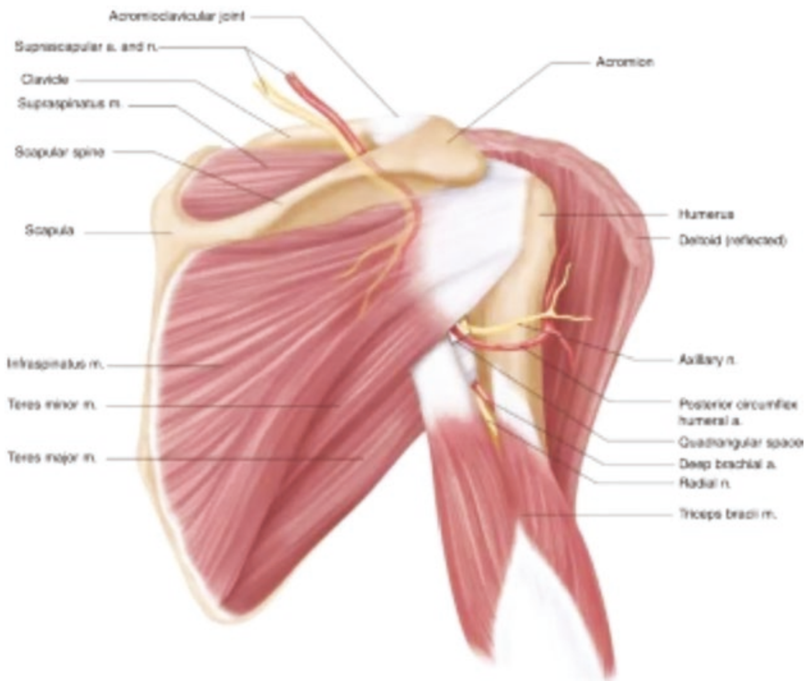

Fig. 30.11 Anatomy of the quadrilateral space (Reproduced with permission from *Pearl, G.J., Hansen, S.K. (2021). Quadrilateral Space Syndrome and Management of the Posterior Circumflex Humeral Artery. In:Illig, K.A. et al. (eds) Thoracic Outlet Syndrome. Springer, Cham.* https://doi.org/10.1007/978-3-030-55073-8_94*)*

sensitive nerve) is one of the terminal branches of the posterior cord of the brachial plexus (C5–C6). In the shoulder, it crosses the antero-inferior portion of the subscapularis muscle, then courses posteriorly through the quadrilateral space wrapping around the surgical neck of humerus posteriorly with the posterior humeral circumflex vessels. Then it divides into an anterior and posterior trunk. The posterior trunk gives branches to the teres minor muscle and the posterior deltoid muscle before terminating as the superior lateral brachial cutaneous nerve. The anterior trunk continues giving branches to supply the middle and anterior deltoid muscle while travelling on the deep subfascial surface and within the deltoid muscle [98]. The PCHA also divides after entering the QS into branches similar to the anterior and posterior branches of the axillary nerve [97]. In order to simplify and give a better understanding of the course of the axillary nerve, Duparc et al. have divided the anatomic course of the axillary nerve into five segments: one from its origin to the inferior border of the subscapularis [96]; from the subscapularis to the long head of the triceps brachii [97]; from the triceps to the surgical neck of the humerus [98]; from the humerus to the entry into the deltoid; and [99] its intramuscular course into the deltoid. This subdivision allows a better radiological and surgical approach to the axillary nerve [99, 100].

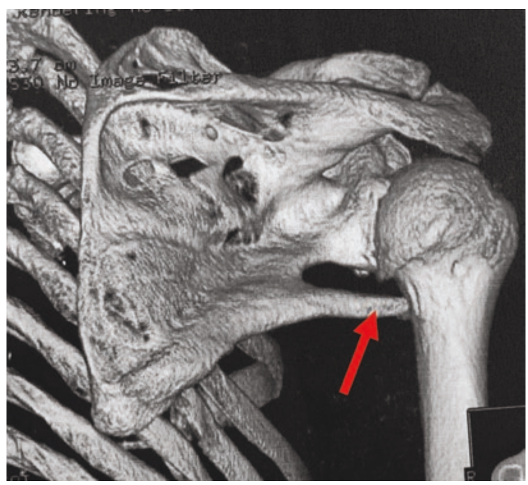

Fig. 30.12 3-D CT scan of a right shoulder (posterior view). A large post-traumatic ossification (arrow) causes compression of the axillary nerve

30.5.2 Etiology and Pathomechanics

This syndrome can be caused by any condition (acute or chronic) that decreases space and causes compression or stretching of the QS contents. The true frequency of QSS is unknown given the difficult and often incorrect diagnosis [97].

At the base of acute forms there is usually a traumatic mechanism such as an anterior glenohumeral dislocation or, rarely, an acute compression given by a scapula bone splinter [101]. Other causes are hematomas, traction injuries to the upper extremity, and deep posterior shoulder intramuscular injections [96].

In the chronic forms, the entrapment syndrome is due to anatomical, tumoral or degenerative locoregional causes (Fig. 30.12). The most frequent cause of entrapment is abnormal fibrous bands [95, 102–104]. These structures, as noted by McClelland et al. in a cadaveric study [108], originate from the thick fascial layer of the long head of the triceps and attach to the teres major, near to the axillary nerve. Moreover, they cause a decreased cross-sectional area at the QS when they tightened during shoulder abduction [105] and external or internal rotation, so the authors postulated that this may cause an entrapment of the QS contents [103].

As described by Flynn et al. [97], the axillary nerve can also be compressed by space-occupying lesions like dilated veins [106], glenoid labral cyst [114] or paralabral cysts [107–110] or benign tumors such as lipomas, humeral osteochondromas [111] and axillary schwannomas [112]. Even a hypertrophied portion of the subscapularis muscle can lead to a QSS, especially in competitive overhead athletes [95, 113] or an atypical nerve course [114].

It is important to underline how the compression mechanism can also be dynamic and positional, making the diagnosis of QSS more complicated.

QSS mainly affects the dominant shoulder of active patients aged 20–40 years, with a male predominance [97, 108]. QSS usually occurs in athletes involved in overhead sports where is common abduction and external rotation such as volleyball, swimming, and baseball [102, 103, 108].

The exact pathophysiology of QSS is unclear. As hypothesized by Brown et al. [108] two forms of QSS can be identified: a vascular QSS (vQSS) form, where a continuous mechanical trauma to the PCHA causes a reduced perfusion itself resulting in thrombosis or aneurism with distal emboli (due to repetitive abduction-external rotation movements as seen in elite overhead athletes); and a neurogenic QSS (nQSS) form, where the presence of fixed structural entities act like space-occupying lesions compressing the axillary nerve (and PCHA) without thrombosis. As suggested by Brown et al. [108], it is important to underlie how the axillary nerve does not appear to sustain injury from the mechanical strain that leads to vQSS. This seems to be related to the intrinsic properties of excursion and strain response to stretch of the nerve itself [115]. As seen later, these different mechanisms reflect two different phenotypes of QSS, with either neurogenic or vascular symptoms.

30.5.3 Clinical Diagnosis

As previously reported, the QSS can manifest according to two particular phenotypic forms, a vascular and a neurogenic one [108].

In the nQSS, the clinical manifestations include poorly localized shoulder pain, paresthesia in the affected extremity in a non-dermatomal distribution, numbness and discrete point tenderness over the quadrilateral space [95, 97]. Entrapment symptoms can be reproduced by flexion, abduction, and external rotation of the affected shoulder [116].

Muscle strength testing reveals weakness of arm abduction and external rotation. Deltoid atrophy or weakness, or both, can be seen in chronic cases but is not a common finding [117].

In the vQSS, the clinical manifestations include coolness, cold intolerance, pallor, cyanosis of the upper extremity digits, with or without splinter hemorrhages, and loss of pulses (radial or ulnar). These clinical findings can reflect a thrombosis of the PCHA. In fact, PCHA arterial thrombosis can go up to the axillary artery and then embolize distally [108, 118].

30.5.4 Instrumental Diagnosis

The diagnosis of QSS cannot disregard the correlation between the clinic exam and instrumental diagnostic tools.

Of all the available imaging techniques, simple radiographs are a fundamental step in the diagnostic approach to any shoulder disease. In fact, they allow not only to evaluate acute traumatic pathologies (e.g., fractures) but also to highlight possible radiopaque occupying-space lesions [111]. Both MRI and CT can be used to evaluate the contents of the quadrilateral space or to search for some indirect signs of QSS, such as neurogenic edema (T2 sequences on MRI) or muscle atrophy of the deltoid or teres minor [109, 116, 119]. In fact, a selective atrophy of the teres minor can arise due to a compression of the corresponding branch of the axillary nerve or PHCA. MRI and CT are also useful to exclude other causes of shoulder pain that can enter into differential diagnosis with QSS, or to evaluate any nerve compressions deriving from scapular bone fragments from the lateral pillar [96, 101].

EMG can detect denervation of the deltoid and teres minor [101, 108, 109, 116]. However, EMG can be falsely negative due to its dynamic or positional nature [106]. Nonetheless, this method can help in the differential diagnosis of other causes of neurological pain such as cervical radiculopathies or brachial plexitis.

Axillary nerve compressive neuropathy most often occurs in association with fibrous bands in the quadrilateral space or other space-occupying lesions, such as paralabral cysts or lipomas. Despite this, an isolated denervation of the teres minor can be associated with other pathologies that cause an alteration of the biomechanics of the shoulder such as rotator cuff injuries, prior surgery and traction injuries with glenohumeral instability [102, 103, 115].

A subclavicular arteriography with the shoulder placed in abduction and maximum external rotation in all those patients who presented symptoms attributable to vQSS [95]. This was intended to evaluate a possible compression or thrombosis of the PCHA. Methods such as ultrasound, MR-angiography, and CT-angiography are currently preferred, although their accuracy in confirming the diagnosis of QSS [120] is not very clear. As reported by Brestas et al. [119], an isolated and widespread increase in muscle echogenicity of the minor muscle teres, in the absence of tendon tears, trauma, or a history of inflammatory myositis, can strongly suggest the existence of an axillary nerve injury [26].

30.5.5 Differential Diagnosis

The differential diagnosis of the quadrilateral space syndrome should include cervical disc disease, brachial plexitis (Parsonage-Turner syndrome), lesions of posterior cord of brachial plexus, rotator cuff tendonitis or tear, biceps tendon tendonitis, adhesive capsulitis, degenerative arthritis of the glenohumeral joint, degenerative arthritis of the acromioclavicular joint, glenohumeral instability, thoracic outlet syndrome, hypothenar hammer hand syndrome, and suprascapular nerve injury [116, 121, 122].

30.5.6 Treatment

There are few references in the literature regarding the treatment and management of QSS. Certainly, this syndrome might significantly limit the sports activity of overhead athletes [106]. The possible therapeutic approaches were analyzed in a recent systematic literature review by Flynn et al. [97]. They range from conservative treatment, always recommended as a first option, to surgical treatment.

Conservative treatment consists of NSAIDs and physical therapy. Techniques such as transverse friction massage, active release soft tissue massage techniques, shoulder ROM exercises, posterior rotator cuff strengthening, and scapular stabilization exercises can lead to good results [3]. No less important for the patient is to avoid all those activities that place the shoulder in abduction and external rotation. Surgical treatment may be considered if the symptoms have not improved within 6 months, or if the compression is linked to an acute traumatic event (i.e., scapular fracture) or a space-occupying injury.

Surgical decompression allows neurolysis and removal of fibrous bands or other possible space-occupying lesions responsible for the pathology [95, 104, 106, 120, 123, 124]. Arthroscopic debridement can be indicated [108] if is present an intra-articular pathology (i.e., paralabral or labral cysts) that can determine the compression of the QS content. In case QSS is due to an acute thrombosis or aneurysm of the PCHA, a thrombolytic treatment is possible, although this produces limited results [124, 125]. More frequently, thrombosis is chronic at the time of diagnosis and does not respond to this treatment. In these cases, surgical treatment with ligation of the PCHA and thrombectomy in case of distal embolization, or removal of the aneurysm [108] may be more appropriate. As hypothesized by Flynn et al. [97] this treatment could be supplemented by the administration of anticoagulants for 3–6 months. There is no clear indication for the use of antiplatelet agents.

30.6 Dorsal Scapular Nerve Entrapment

There may be several reasons for periscapular and interscapular pain. Among these, a possible cause often overlooked is the entrapment of the dorsal scapular nerve (DSN) [126]. Many causes can lead to this syndrome. Electrodiagnostic and imaging studies are fundamental to clarify and interpret the clinical symptoms. Treatment of this condition can be either conservative or surgical.

30.6.1 Anatomy

DSN is a primarily motor nerve originating from C5–C6. After leaving the scalene region (crossing the middle scalene muscle), it heads first below the brachial plexus and the levator scapulae [127], and then runs along the deep surface of the rhomboid muscles. It is responsible for the motor innervation of the levator scapulae muscle itself, which elevates the scapula, and of both the rhomboid major and minor muscles, which pull the scapula medially [128] (Fig. 30.13).

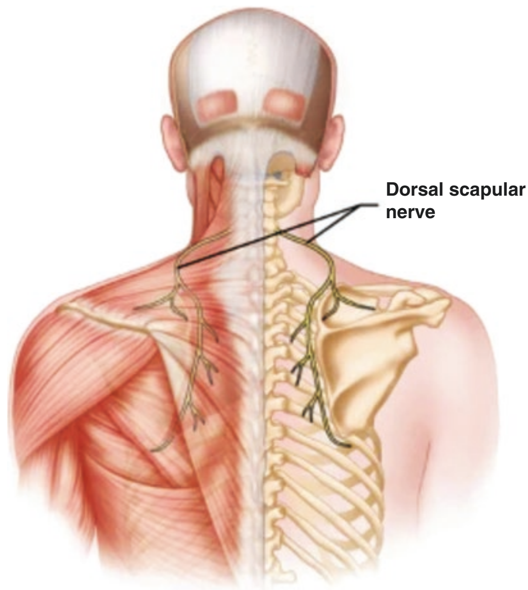

Fig. 30.13 Anatomy of dorsal scapular nerve (Reproduced with permission from *Trescot, A.M. (2016). Dorsal Scapular Nerve Entrapment. In: Trescot, A.M. (eds) Peripheral Nerve Entrapments. Springer, Cham.* https://doi.org/10.1007/978-3-319-27482-9_32*)*

30.6.2 Etiology and Pathomechanics

This nerve can be entrapped anywhere along its course, albeit entrapment is often located at the middle scalene muscle, because the nerve often pierces this muscle [1]. It also can be entrapped at the rhomboids. In general, DSN entrapments are attributed to the hypertrophy of the middle scalene muscle [129, 130]. This impingement determines a well-defined chronic pain syndrome, often referred to as DSN syndrome [131]. It may result not only from repetitive overhead activities (in some cases with heavy lifting), but also after chronic postural strain, trauma (like stretching of scalene during "whiplash" or anterior shoulder dislocation), or by abnormally large C7 transverse process [132]. The cause of injury can also be iatrogenic (usually post-surgical or post-bracing) [38, 133]. In fact, it can be injured during interscalene brachial plexus injections [134]. As pointed out by Williams et al. [131], procedures such as nerve blocks, scalenectomy, or removal of the first rib (all common treatments for thoracic outlet syndrome) could place a variably positioned DSN at greater risk of iatrogenic damage, recommending an ultrasonography to accurately assess the position and pathway of the DSN before a planned surgical interventions in the scalene region. In addition, the frequency of anatomical DSN impingement in the general population is unknown. However, anomalies in the brachial plexus may affect the course of the DSN and its predisposition to impingement [131]. Moreover, the impingement of the DSN can affect the interscalene space contributing to an "atypical" thoracic outlet syndrome (TOS) [135, 136].

30.6.3 Clinical Diagnosis

The entrapment of the DSN is often accompanied by a compromised shoulder girdle function, characterized by stiffness, pain, dysesthesia and dysfunction in midscapular region, irradiating through the back, upper extremity, and/or cervical region [137]. Furthermore, these disorders are often accompanied by a compromised shoulder girdle function, characterized by atrophy of the rhomboid muscles and the elevator scapula which can lead up to a lateral winged scapula [138], although that condition is subtle and usually attributed to pathology of the LTN or the SAN. Atrophy of rhomboids and of the elevator scapula muscle can be shown by lowering the patient's affected arm slowly from forward elevation.

Being primarily a motor nerve, it could be strange how its entrapment can determine a painful interscapular symptomatology. However, this occurs if the syndrome has progressed to the point in which scapular winging occurs. In fact, the cutaneous branches of the thoracic posterior rami adjacent to the scapula may become stretched out, thus causing pain. This is also possible if the nerve is impinged by taut bands within the scalene or rhomboid musculature [126]. For these reasons, the diagnosis of this entrapment syndrome is difficult, and it may be confused or included with LTN entrapment.

30.6.4 Instrumental Diagnosis

Imaging modalities such as MRI or ultrasonography can be used in the diagnosis of this entrapment syndrome, as well as electrodiagnostic study (EMG).

Plain radiographs of the shoulder, cervical spine, and chest are recommended in the initial evaluation of the winged scapula without an apparent etiology [130, 139]. A thoracic MRI may show edema, atrophy or signal changes affecting the rhomboids. In fact, an increased rhomboid signal on MRI corresponds closely with spontaneous activity on EMG, with a relative sensitivity of 84% and specificity of 100% for detecting denervation [38, 130]. Ultrasonography can be useful in evaluating the pathways of the DSN and as an aid in the diagnosis in patients with generalized shoulder pain or dysfunction.

Needle stimulation of the rhomboid and the levator scapulae shows delayed potentials exceeding the cut-off value of the controls or side-to-side latency difference exceeding the cut-off point obtained from the controls. It also may show EMG abnormalities from these muscles, such as long duration and polyphasic MUP with spontaneous activity. Generally, amplitude measurements are not taken into account for the evaluation of the conduction anomaly, since the recording does not allow the reliable measurement of the potential amplitude of the compound muscle action. Moreover, it is not always possible to detect EMG abnormalities from the levator scapulae as such muscle can be directly innervated by branches from C3, C4, and C5 anterior roots, or because entrapment can occur lower in the rhomboids [126].

30.6.5 Differential Diagnosis

The differential diagnosis includes rotator cuff disease, glenohumeral instability, acromioclavicular pathology, adhesive capsulitis, shoulder impingement syndrome, cervical radiculopathy, brachial plexopathy, thoracic facet pathology and thoracic disk pathology.

30.6.6 Treatment

As with other entrapment syndromes, the first treatment choice is typically conservative and based on NSAIDs and specific rehabilitation programs. Once the pain has improved, the patient can start a physical therapy and rehabilitation program consisting of active and passive ROM exercises for the shoulder and the neck, muscle strengthening exercises, mainly focused on scapula stabilizing muscles, rhomboid and levator scapulae [130]. This exercise program can also be associated with a myofascial release of the scalene region, trigger points injections or postural reeducation. Haim and Urban [140] described the use of botulinum toxin for the treatment of rhomboid muscle dystonia associated with DSN entrapment.

Surgery might be appropriate in cases that remain unresponsive to conservative treatment or in cases where a traumatic damage to this nerve is evident. Chen et al. [135] reported good results in terms of relief of the symptoms after the section of the middle scalene muscle with the complete decompression of the DSN. However, most of the DSN compressions were categorized as TOS. They also found isolated lesions and described them as atypical TOS [135].

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