Chapter

36 Ultrasound-Guided Botulinum Toxin Injections for Thoracic Outlet Syndrome

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Introduction

Thoracic outlet syndrome (TOS) is a rare, often perplexing disorder caused by compression of neurovascular structures, just above the first rib and behind the clavicle or under the pectoralis minor, leading to cervicogenic–brachial pain and other symptoms in neck and upper limb. Historically, several names including cervical rib syndrome, scalenus anticus syndrome, costoclavicular syndrome, hyperabduction syndrome and pectoralis minor syndrome have all been used to describe the group of disorders involving the thoracic outlet (Murphy, 1916; Ochsner, 1935; Falconer 1943; Wright, 1945; Sanders, 2017). Patients with TOS may present clinically with a wide variety of signs and symptoms that may confound clinicians unfamiliar with the condition (Sanders *et al.*, 2008; Ferrante, 2012; Foley *et al.*, 2012). There are no gold standard diagnostic tests to establish the diagnosis of thoracic outlet syndrome, as the term collectively encompasses the spectrum of clinical presentations related to neurovascular compromise in the region of the thoracic outlet (Peet 1956). This may lead to misdiagnosis, or overdiagnosis, of TOS in patients who present with symptoms suggesting TOS but who have other underlying causes for their symptoms.

One of the many emerging applications for BoNT therapy is for the treatment of musculoskeletal pain disorders, including TOS. The antinociceptive mechanism of BoNT for the treatment of cervical dystonia, chronic migraine, joint pain, myofascial pain and cerebral palsy has long been recognized and reported in the literature, but the mechanism of action remains incompletely understood (Barwood *et al.*, 2000; Mense, 2004; Aoki, 2005; Gobel *et al.*, 2006; Singh *et al.*, 2009). Although numerous reports have emerged within the literature citing the efficacy of BoNT for pain and other symptoms associated with TOS, additional research is needed to establish the exact role of BoNT in treatment of TOS and other pain disorders. (Jordan *et al.*, 2007; Danielson and Odderson, 2008; Torriani *et al.*, 2010; Foley *et al.*, 2012). The principal proposed mechanism of action in TOS includes reduced muscle spasm leading to decreased compression of neurovascular structures (Jordan *et al.*, 2007; Tsao, 2007; Danielson and Odderson, 2008). In addition, injection of BoNT into muscles also leads to denervation atrophy, which may theoretically reduce compression from hypertrophied musculature and decrease neurogenic or vasogenic symptoms. It has also been reported that BoNT reduces release of nociceptive neurotransmitters, which could also be a contributing factor in reduced pain in TOS.

When considering BoNT therapy for TOS, accurately identifying the problematic muscle, if there is one, and then targeting this muscle for injection is often challenging. Injections in this region have an intrinsic risk associated with inserting a needle and injecting structures located within a region traversed by large caliber vessels, nerves and the apex of the lung. In addition, injections of BoNT are associated with additional risks, including dysphagia and muscle weakness (Simpson *et al.*, 2008; Truong *et al.*, 2010; Hong *et al.*, 2012). Various targeting techniques have been reported to guide injection therapy for TOS, including with BoNT. This chapter briefly reviews TOS and the literature related to use of BoNT for TOS and also provides a detailed review of procedural techniques for ultrasound (US) guidance in BoNT therapy for TOS.

Thoracic Outlet Syndrome

The term "thoracic outlet syndrome" describes a group of disorders leading to cervicobrachial pain and neurovascular symptoms and caused by compression of elements of the brachial plexus, subclavian vessels or both by musculoskeletal structures in the region of the neck known as the thoracic outlet (Sanders *et al.*, 2008; Ferrante, 2012; Foley *et al.*, 2012; Sanders, 2017).

When the signs and symptoms of neurogenic TOS or vasogenic TOS are clear and diagnostic testing is supportive, a diagnosis of true TOS is undisputed. The controversy surrounding TOS may be partly attributed to mis- or overdiagnosis in patients with nonspecific cervicogenic pain miscategorized as "non-specific thoracic outlet syndrome" (Ferrante, 2012). A chapter describing a treatment technique for TOS, therefore, warrants a short review of the disorder to increase the accuracy of diagnosis and to reduce unnecessary and risky interventions in patients with an alternative diagnosis.

Classification of Thoracic Outlet Syndromes

One accepted classification system for TOS defines specific subtypes based on affected/compromised structures and/or

clinical features (Sanders *et al.*, 2008; Ferrante, 2012). The defined subtypes of TOS and their symptoms include:

* *Neurogenic*: compression of elements of the brachial plexus (roots, cords) leads to numbness, tingling, pain, atrophy, weakness; involvement of the lower trunk is the most often described form of TOS
* *Arterial vasogenic*: compression of the subclavian or axillary artery leads to claudication symptoms with a cool pale extremity, decreased pulses, pain and Raynaud's symptoms
* *Veno-vasogenic*: dusky discoloration of the limb with diffuse limb swelling
* *Combined neurovascular*: combination of above symptoms
* *Nonspecific*: cervicogenic pain, numbness or paresthesia that do not clearly fit into the above categories; non-specific TOS is a controversial diagnosis and use of this diagnosis is discouraged (Ferrante, 2012).

Anatomy of the Thoracic Outlet

The thoracic outlet refers to the region of the body extending from the supraclavicular fossa to the axilla. From anterior to posterior, this encompasses the area located between the clavicle and first rib. The thoracic outlet is further divided into three specific regions: the interscalene triangle, the retroclavicular region and the subcoracoid region or retropectoral (retropectoralis minor) space (Fig. 36.1a,b).

Structures of interest contained within or traversing the region of the thoracic outlet include;

* *Bone*: C7 vertebra, cervical rib (if present), fibrous extensions from C7 (if present), clavicle, coracoid process
* *Muscles*: anterior, middle scalenes, subclavius, pectoralis minor muscle and tendon
* *Nerves*: brachial plexus including the trunks (supraclavicular), divisions (retro-clavicular) and cords (infraclavicular); axillary and musculocutaneous nerves (infraclavicular)
* *Vessels*: subclavian artery and vein, axillary artery and vein.

Sites of Entrapment of Nerves

Reported sites of entrapment of nerves or vessels include compression between:

* *Anterior and middle scalene muscles*: neurogenic symptoms in the distribution of C8 to T1 nerve roots. Compression is attributed to an enlarged anterior or middle scalene, muscle spasm or a fibrous band within the anterior scalene or aberrant location of the trunk(s) of the brachial plexus
* *Anterior scalene and cervical rib or anterior scalene and a fibrous band extending from C7*: symptoms may be neurogenic or arterial/vasogenic, including decreased pulse, cool, pale extremity or Raynaud's symptoms
* *Clavicle and first rib*: largely post-traumatic (most often at the level of the cords of the brachial plexus or axillary vessels) following a mid-shaft clavicle fracture with robust callus formation; the callus may compress neurovascular elements and patients most often present with combined neurovascular TOS symptoms
* *Beneath the pectoralis minor muscle or tendon*: compression may be neurogenic (medial or lateral cord), arterial (axillary) or venous (axillary).

Epidemiology

Thoracic outlet syndrome is a rare disorder. The reported incidence of true neurogenic TOS approaches 1 per million. Given that neurogenic TOS is diagnosed in 95% of those with TOS, the vasogenic causes will be exceedingly rare (Torriani *et al.*, 2010). Given the vague symptoms and lack of anatomical correlates, there can is no reliable information related to the epidemiology of nonspecific TOS.

Work-Up and Differential Diagnosis

Establishing the diagnosis of TOS begins with the clinical examination. This includes information obtained from the pertinent history, identification of clinical symptoms combined with findings from a detailed physical examination and provocative orthopedic maneuvers. When anatomical findings consistent with TOS are not obvious in patients presenting with pain in the arm, with or without numbness, cervicogenic pain or radiculopathy must be ruled prior to selecting a treatment. Physical exam maneuvers for TOS place the shoulder in horizontal flexion or extension, abduction and external rotation in order to place stress on the neurovascular structures within the thoracic outlet and accurately reproduce reported symptoms. Common orthopedic tests include maneuvers such as Adson's, Wright's, Elevated Arm Stress Test and Roos. The greatest limitation of the provocative orthopedic maneuvers is that they have been shown to produce the signs and symptoms of vascular compromise in both symptomatic as well as asymptomatic individuals (Plewa and Delinger, 1998). For this reason, the authors cannot recommend the use of any single or series of orthopedic maneuvers as the definitive screening tool for TOS. Rather, it is recommended that patients with suspected TOS undergo conservative treatment for mildly symptomatic cases, or further diagnostic testing for more severe cases, before proceeding to more definitive treatments such as BoNT injections or surgery.

Selection of additional diagnostic testing should be directed by the patient's history, symptoms and exam findings as above. Electrodiagnostic testing including nerve conduction and electromyography (EMG) should be considered in patients presenting with neurogenic TOS symptoms, and the results of testing should be definitive to establish a diagnosis of neurogenic TOS. While a normal electrodiagnostic test result does not exclude other forms of TOS, the test may also reveal an alternative cause of the patient's symptoms such as other

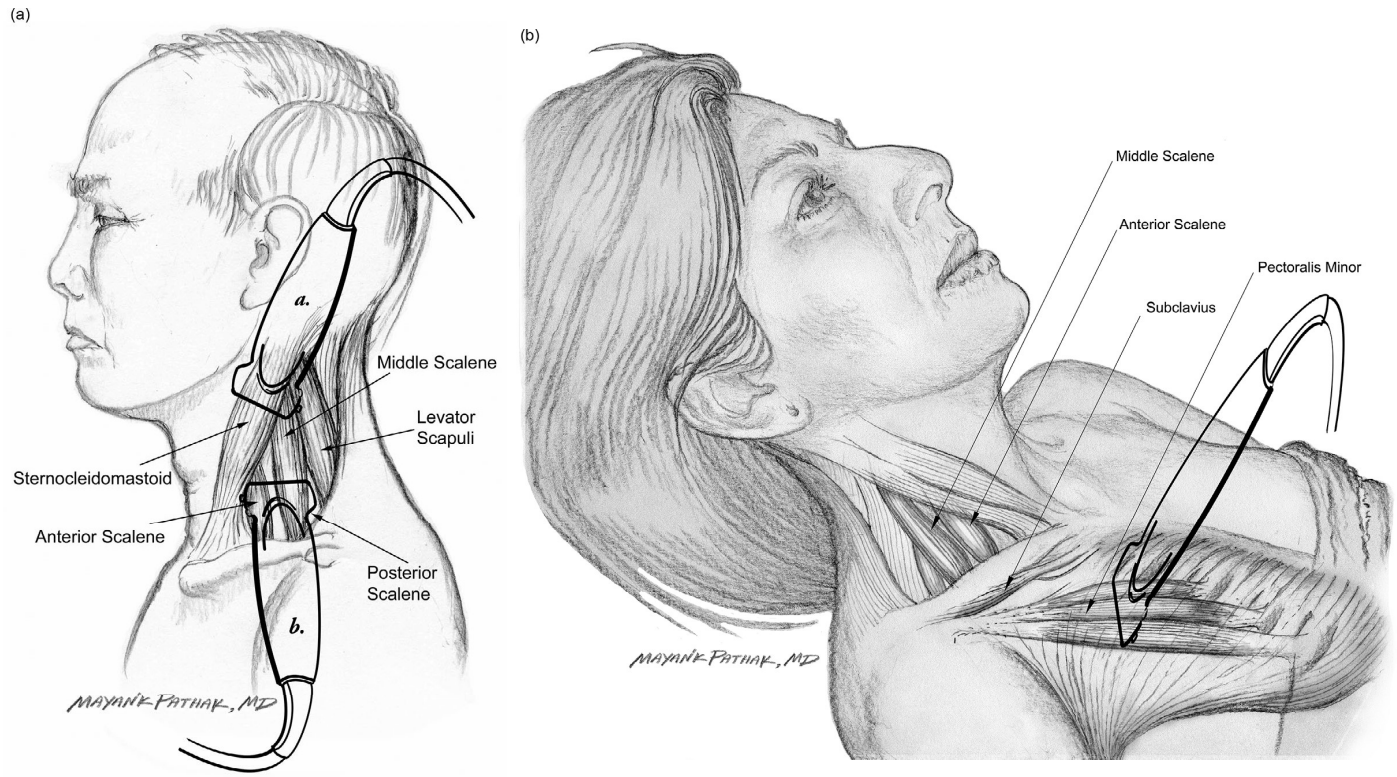

Fig. 36.1 (a) Anterolateral neck with transducer location, orientation SCM and scalenes (from Dr. Pathak). (b) Transducer location, orientation for short-axis pectoralis minor scan.

peripheral nerve entrapments, peripheral neuropathy, plexopathy, radiculopathy or focal anterior horn cell loss (Tsao, 2007; Urschel and Kourlis, 2007; Ferrante, 2012). In neurogenic TOS, abnormal results have been reported in the median, ulnar or medial cutaneous nerve of the forearm sensory nerve action potentials. Abnormalities of ulnar potentials when recording from the fifth finger is virtually mandatory for diagnosis of neurogenic TOS. Denervation in muscles innervated by C8–T1 is also characteristic, and atrophy in these same muscles may also be seen in long-standing neurogenic TOS (Ferrante, 2012; Foley *et al.*, 2012).

Diagnostic imaging in patients with TOS generally includes plain radiographs to evaluate for a cervical rib, clavicular callus, other bony abnormalities or mass effect. Fibrous bands will not be visible on plain films but are detected by magnetic resonance imaging (MRI). Imaging with MRI or computed tomography (CT) may also be useful when an apical lung mass, tumor or malignancy is suspected (Ferrante, 2012).

When venovascular symptoms predominate, venography is the gold standard. For patients with suspected arterial vasogenic TOS, MRI, CT angiography or duplex ultrasonography should be considered (Stapleton *et al.*, 2009). High-frequency US is useful for both procedural guidance and as a diagnostic tool for arterial vasogenic TOS and has the advantage of portability, lower cost and no exposure to ionizing radiation. Ultrasound in B-mode and Doppler modes can also provide a dynamic assessment of vascular flow and can be used during provocative clinical maneuvers to assess the effect of a maneuver or neck/limb position on flow within a vessel (Jordan *et al.*, 2007; Danielson and Odderson, 2008; Odderson *et al.*, 2009; Torriani *et al.*, 2010). It is important to recognize that arterial or venous compression with various maneuvers is also seen in asymptomatic individuals, so thoughtful clinical correlation is needed.

In patients with symptoms of neurogenic or combined neurovascular TOS, where hypertrophied scalene muscles are thought to cause compression, diagnostic muscular blocks with lidocaine may aid in establishing the diagnosis (Brooke and Freischlag, 2010). A number of reports in the literature have investigated the role of anesthetic injections with lidocaine and BoNT as diagnostic tools both to establish the diagnosis of TOS and, prior to surgery, to guide the surgeon's choice of procedure for TOS (Odderson *et al.*, 2009; Benzon *et al.*, 2012). Recently reported standards of the Society for Vascular Surgery recommend the use of scalene blocks with local anesthetic in their criteria to define neurogenic TOS before proceeding with surgical intervention (Illig *et al.*, 2016). One possible limitation when comparing muscular blocks with BoNT is the therapeutic limitations of lidocaine when compared with BoNT. As mentioned above, one therapeutic advantage of BoNT injection is the resulting denervation atrophy that occurs over time and may decrease the size of the target musculature and reduce the compressive forces on the neurovascular structures. For this reason, the authors recommend that additional studies are required comparing the use of BoNT preceded by diagnostic scalene blocks before conclusive recommendations can be made. However, for clinicians unsure if the scalenes are contributing to symptomatic TOS, despite diagnostic testing above, we recommend that temporary muscular blocks with local anesthetic agents such as lidocaine should be considered before proceeding with BoNT injection.

Treatment

Various treatments for TOS have been proposed, including postural exercises or physical therapy, manipulation, acupuncture, BoNT injections and various surgical procedures. The focus of this text is on BoNT therapy as a diagnostic and or therapeutic intervention for TOS. There are several excellent reviews that contain a full discussion of the various proposed treatments for TOS (Jordan *et al.*, 2007; Ferrante, 2012; Foley *et al.*, 2012). Most importantly, certain therapies are only rational for specific causes or subtypes of TOS. For example, if the brachial plexus is compressed by a first rib, there is no role for BoNT therapy and surgical referral must be considered. Currently the use of BoNT is supported only by anecdotal case reports (class IV evidence, class C recommendations), and there is certainly no logic in using BoNT therapy if entrapment or compression by a muscle is not the cause of TOS symptomatology (Gross and Johnston, 2009).

Botulinum Neurotoxin Therapy under US Guidance

As noted above, BoNT therapy for TOS is associated with risks attributed to needle insertion/injection procedure itself as well as the inherent risks associated with BoNT pharmacology. It is incumbent on the clinician to establish a diagnosis of true TOS, the true site of compression and involved structures prior to proceeding with BoNT therapy. Clearly, BoNT has no place in treatment of TOS caused by boney callus formation from a clavicle fracture or compression from a true cervical rib. Additionally, BoNT has no place in treatment of nonspecific TOS where no objective compression can be identified. However, BoNT may be useful for patients with compression of neurovascular structures caused by the effects of muscle hypertrophy or muscle action on bones (anterior scalene on first rib, pectoralis minor-coracoid process), leading to TOS.

Since the mid-2000s, several reviews of US-guided BoNT therapy have been published that provide detailed descriptions of these procedures, which is detailed below (Jordan *et al.*, 2007; Danielson and Odderson, 2008; Le *et al.*, 2010; Torriani *et al.*, 2010; Foley *et al.*, 2012).

Techniques

Ultrasound guidance for TOS has been described for BoNT injections in the anterior and middle scalene, pectoralis minor and subclavius muscles. Accurately localizing any of these

muscles may be challenging without imaging-based guidance, such as US or fluoroscopy.

A general review of US and US guidance techniques useful for BoNT injections is provided elsewhere in this text (e.g., Chapter 7). For a more detailed review of US-guided procedural techniques, see Smith and Finnoff (2009), Alter (2010) and Alter *et al.* (2020).

Both in-plane and out-of-plane injection techniques can be useful when performing US-guided BoNT injections for TOS, and clinicians who perform these procedures should be skilled using either technique and also be familiar with their advantages and disadvantages. Because of anatomical variations, patient-related issues with positioning and other factors, one specific technique may be required for a given procedure. Therefore, a brief discussion, specific to the topic of these techniques for TOS, is provided below (Smith and Finnoff, 2009; Alter, 2010).

Out-of-Plane

When using an out-of-plane injection technique, the needle is inserted across the short axis of the transducer (Fig. 36.2a), resulting in a cross-sectional view of the needle, which is visualized as a bright (i.e., hyperechoic) dot (Fig. 36.2b). In some situations, an out-of-plane technique may provide a more direct path to the target. However, a disadvantage of this technique is that the entire length of the needle on its path is not visualized. Therefore, during needle insertion using this technique, care must be used to keep the *tip of the needle* within the US beam because if the tip is advanced beyond the US transducer, it may be in an untargeted structure. Rather than using the oft-described shallow angle of insertion (Fig. 36.3a), the authors use a steep angle of needle insertion relative to the transducer (Fig. 36.3b), which assists in keeping the needle within the ultrasound beam and may reduce the likelihood that the needle tip passes beyond the beam (Farrell *et al.*, 2020). When using the out-of-plane technique, to track the path of the needle to the target, a walk-down technique is universally recommended. In this approach, the needle is vibrated or jiggled during insertion. Jiggling the needle creates movement in the tissue through which the needle is passing. The clinician tracks the position of the needle by observing movement of tissue as the needle passes from the skin and superficial structures into deeper structures on its path to the target. When the needle tip reaches the target, a small quantity of the injectate is injected, confirming the correct location.

In-Plane

Many, if not most, clinicians prefer using an in-plane technique when performing US-guided procedures, including BoNT injections. When using this technique, the needle is inserted along the length of the transducer (perpendicular to the ultrasound beam) (Fig. 36.4a). An advantage of the in-plane approach is that this technique provides a long-axis view of the needle and, therefore, the entire length of the needle and its path to the target may be visualized (Fig. 36.4b). However, in some circumstances, this technique (keeping the needle perpendicular to the ultrasound beam) may be technically challenging to perform, and it also often requires an indirect path of the needle to the target. This may be difficult in the anterior neck where an indirect needle path may traverse regions with various large vessels and nerves. Another important consideration is that the angle of needle insertion will affect needle visualization. As noted above, inserting the needle at a shallow angle of insertion (Figure 36.5a) positions the needle perpendicular to the ultrasound beam, thereby decreasing anisotropy and enhancing needle visualization. In contrast, when the needle is inserted with a steep angle of insertion (Fig. 36.5b), it is oblique to the ultrasound beam, resulting in

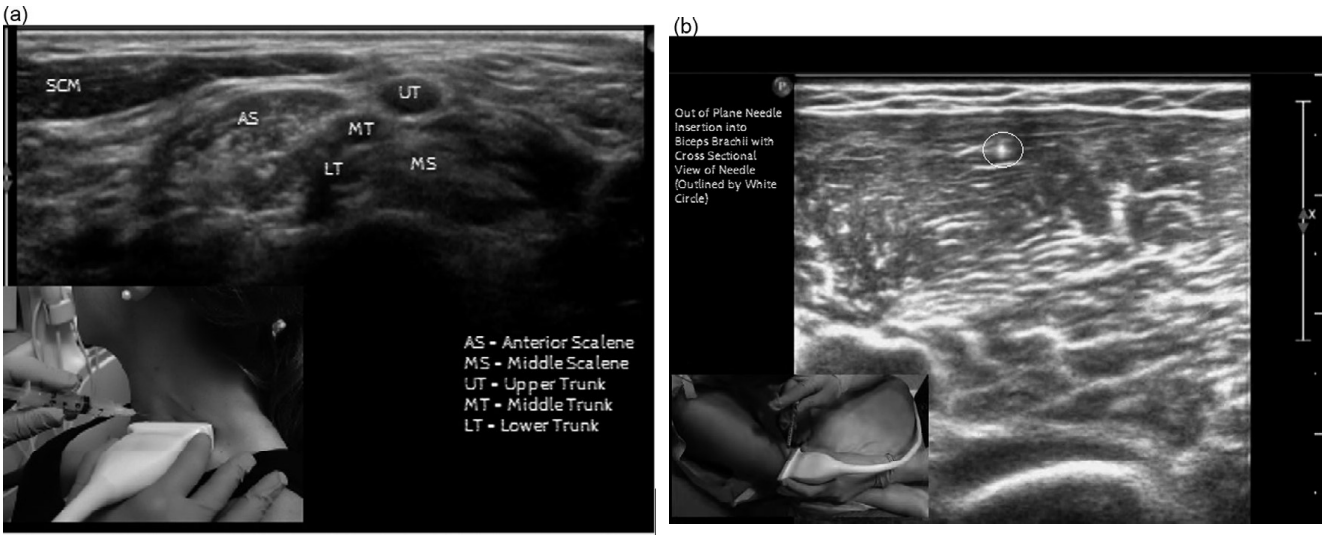

Fig. 36.2 (a) Interscalene triangle, short-axis B-mode Image (inset: transducer location/orientation, out-of-plane needle insertion technique). (b) short-axis B-mode US image, out-of-plane needle insertion technique, view of needle.

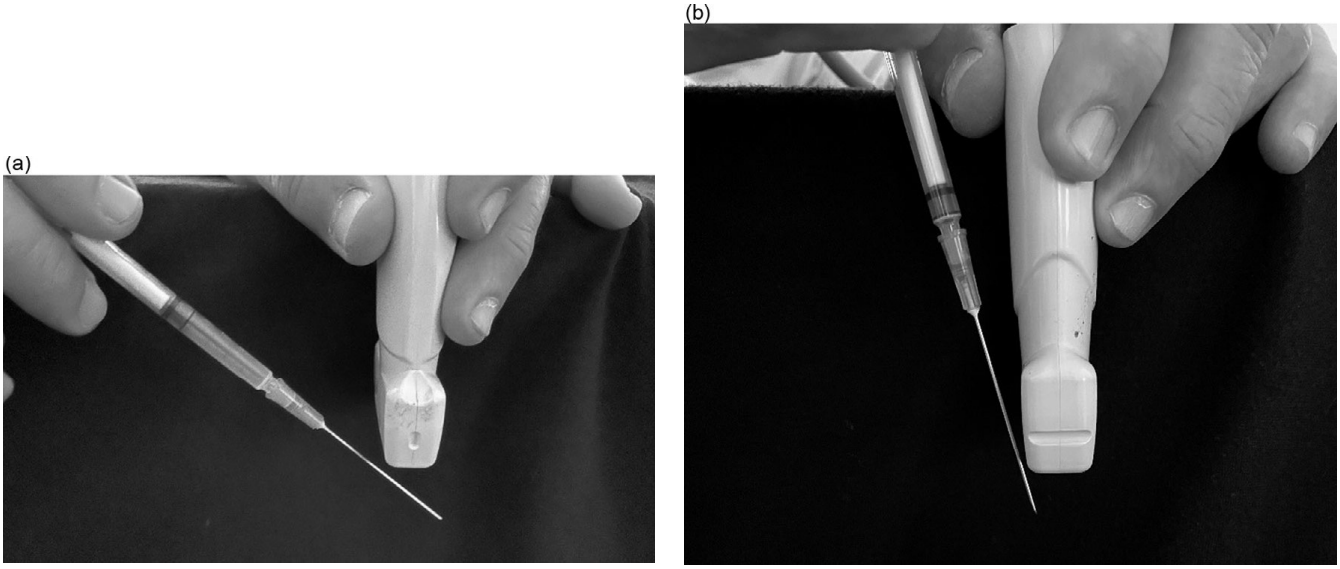

Fig. 36.3 (a) Out-of-plane technique with shallow angle of needle insertion. (b) Out-of-plane technique with steep angle of needle insertion.

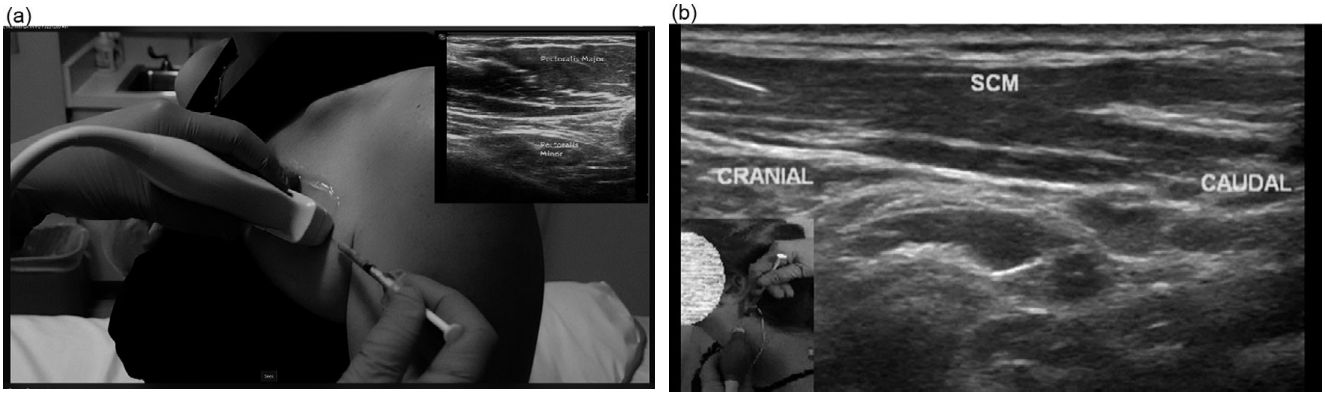

Fig. 36.4 (a) In-plane needle insertion technique, pectoralis major, minor (B-mode US scan inset). (b) B-mode In-plane needle view, long-axis scan sternocleidomastoid muscle (inset: transducer location/orientation).

increased needle anisotropy and decreased needle visualization. Use of beam steering or needle visualization features of the ultrasound instrument will often enhance needle visualization. Using a shallow angle of insertion may be difficult when targeting deeper structures, as it requires an oblique approach to the target and a longer needle (Farrell *et al.*, 2020).

Patient Positioning

When using either an in-plane or out-of-plane injection technique for injecting the scalenes or other cervical muscles, identifying a clear path for the needle to the target is paramount. To accomplish this, the authors recommend placing the patient in a seated or semi-recumbent position, with the head and neck first in neutral rotation, followed by scanning with the head and neck in maximal contralateral rotation. For injections performed within the anterior cervical region, maximum contralateral movement of the head may move neurovascular structures away from the target muscles, thereby providing a more direct path of the needle to the target (Park *et al.*, 2018; Farrell *et al.*, 2020). Due to anatomical differences, it is recommended that practitioners attempt to locate their target under US with the patient in both neutral cervical rotation and maximum contralateral rotation.

Anterior Scalene Injections

The anterior scalene is the most frequently targeted muscle for BoNT injections for TOS, although some clinicians target both the anterior and middle scalenes. Spasm or hypertrophy of the muscle may cause direct compression of neurovascular structures in the supraclavicular fossa, specifically within the interscalene triangle. Spasm or overactivity of the anterior scalene may also elevate the first rib, leading to compression of neurovascular structures (Jordan *et al.*, 2007; Torriani *et al.*, 2010).

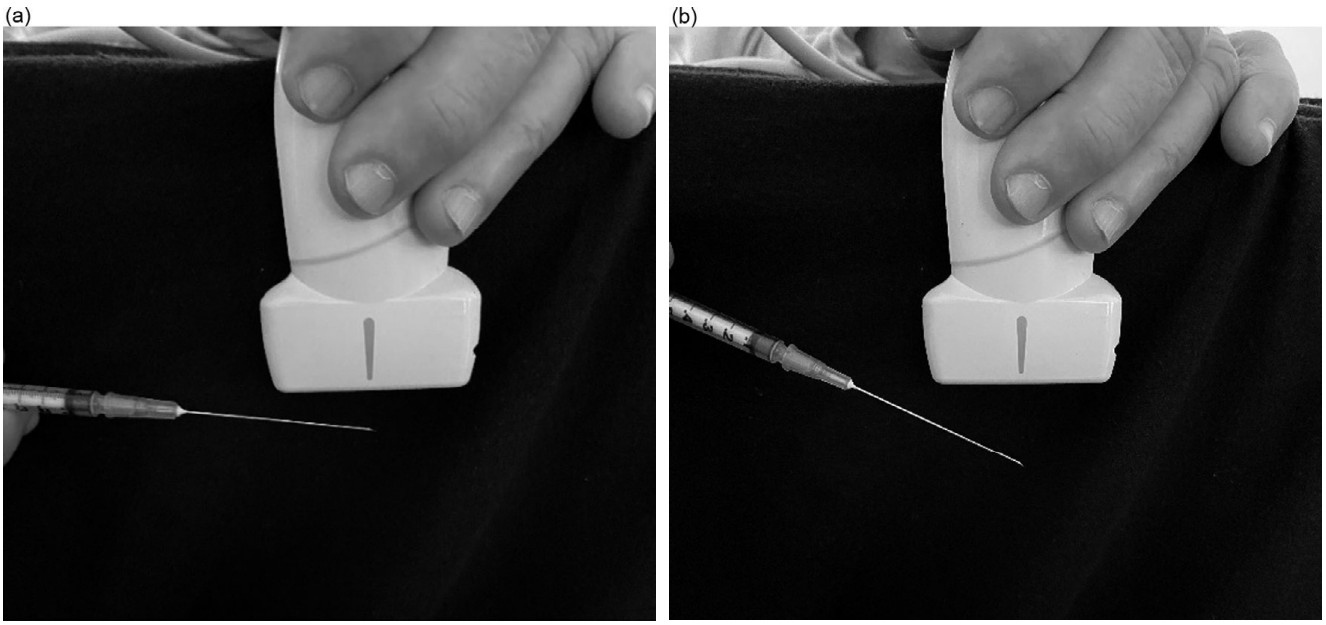

Fig. 36.5 (a) In-plane technique, flat angle of needle insertion. (b) In-plane technique, steep angle of needle insertion.

The scalene muscles run from their origin on the transverse processes of C3–C6 to their insertion on the first rib (anterior scalene). After exiting their respective neural foramina, the trunks of the brachial plexus descend into the interscalene triangle, running between the anterior and middle scalene muscles (Figs. 36.1a, 36.2a). This region is highly vascularized and includes the transverse cervical artery that transverses this space.

The scalene muscles are best viewed in short-axis, that is, transverse plane (Jordan *et al.*, 2007; Torriani *et al.*, 2010; Alter *et al.*, 2020). The transverse or short-axis (cross-sectional) imaging plane provides a more distinct view of the fascial planes between the sternocleidomastoid, anterior scalene, middle scalene and the adjacent neurovascular structures (brachial plexus, phrenic nerve, carotid, jugular, subclavian artery/vein, transverse cervical artery) (Figs. 36.2a, 36.6a,b).

In most nonobese patients, the scalene muscles are relatively superficial; therefore, a relatively high-frequency linear transducer (12 to 18 MHz to 4 or 5 MHz) is selected for imaging the cervical region including the TOS. Because US transducers create a narrow beam of US (thickness of a credit card), only a thin image slice is created. This mandates that the sonographer glides, slides or moves the transducer back and forth over a structure of interest to image it in its entirety.

For US-guided scalene injections, the transducer is placed over the interscalene triangle in a plane perpendicular to the underlying scalene muscles (i.e., transverse view of scalene muscles (Fig. 36.2a). The transducer is then moved, rocked or toggled to image the anterior scalene, middle scalene, brachial plexus and adjacent vessels and nerves (Fig. 36.6a) (Smith and Finnoff, 2009; Alter, 2010; Alter *et al.*, 2020).

To localize the scalene muscles, a useful technique, particularly for a less experienced sonographer, is to first scan the anterior neck in the transverse plane and locate the sternocleidomastoid muscle and carotid and jugular vessels. These structures serve as easily identifiable landmarks or reference structures or "home-base" (Fig. 36.6c), which facilitates identification or localization of the anterior and middle scalene muscles. The sternocleidomastoid muscle and carotid and jugular vessels are located anterior and superficial to the anterior scalene muscles.

As noted above, either an in-plane (Fig. 36.7a) or an out-of-plane technique (Figs. 36.2a, 36.7b) can be used for US-guided scalene injections. Many authors prefer an in-plane technique, where the needle is inserted from lateral to medial and advanced into the anterior or middle scalene (Fig. 36.7a) (Jordan *et al.*, 2007; Torriani *et al.*, 2010). However, for scalene injections, the authors prefer an out-of-plane technique, which often provides a more direct path to the muscle while avoiding the adjacent plexus and vessels. The in-plane technique may be challenging to perform in some patients because of the proximity of the carotid, jugular, transverse cervical artery and trunks of the brachial plexus obstruct a clear path to the scalene muscle targets. Physicians should utilize the technique that provides the best view of the muscle, structures to be avoided and path of the needle to the target.

In an out-of-plane injection technique for anterior scalene muscle (Fig. 36.7b), the transducer is positioned for a short-axis (cross-sectional) dosage. The injection of botulinum toxin for the treatment of TOS is "off-label" in the USA for all three serotype A products (abobotulinumtoxinA, incobotulinumtoxinA, onabotulinumtoxinA) and for the sole serotype B product (rimabotulinumtoxinB. For scalene injections, the dose range

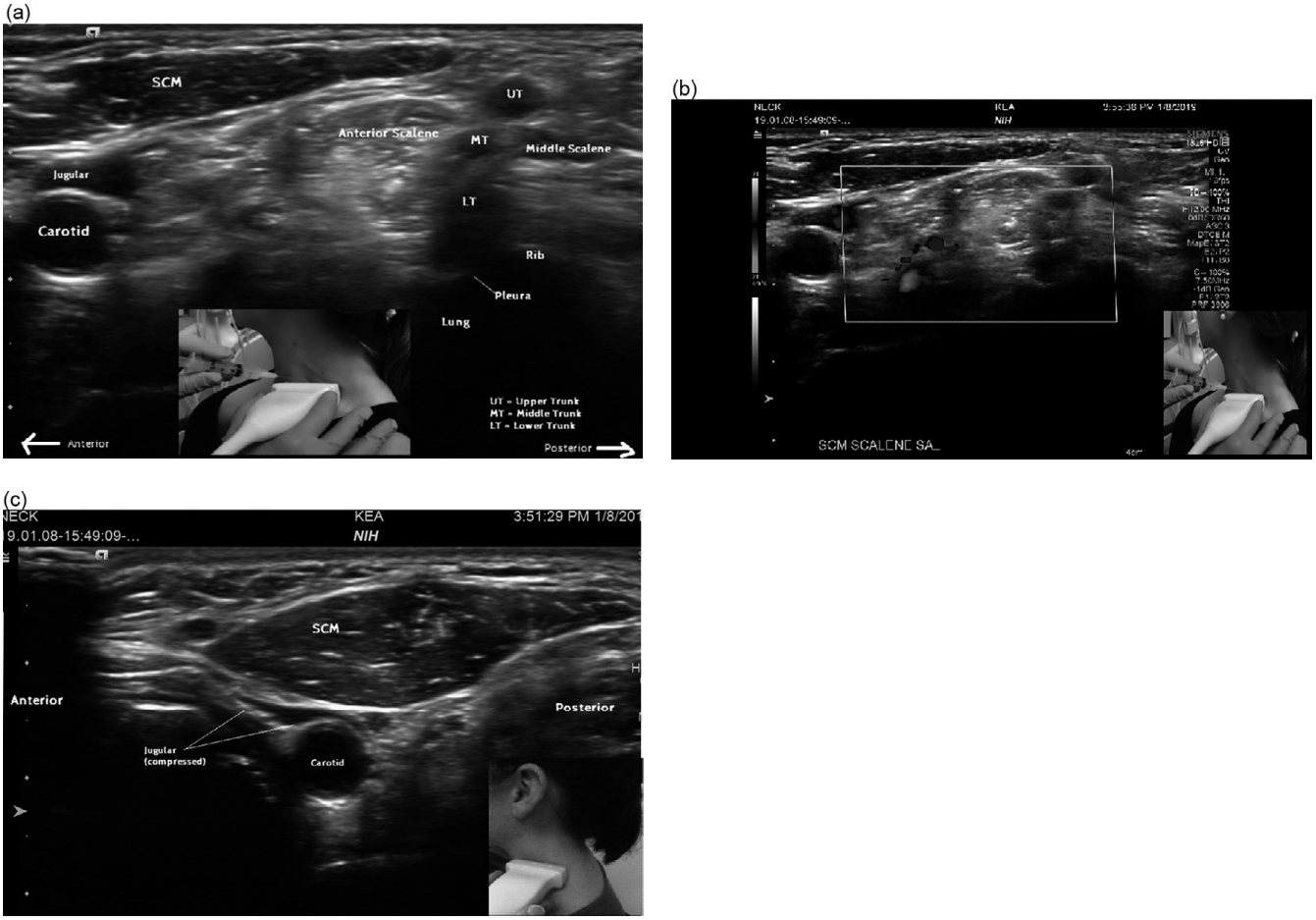

Fig. 36.6 (a) Short-axis (transverse) B-mode scan anterolateral neck (transducer location/orientation interscalene triangle). (b) short-axis color Doppler image, interscalene triangle. (c) Short-axis (transverse/cross-sectional) view sternocleidomastoid muscle (inset:transducer location/orientation).

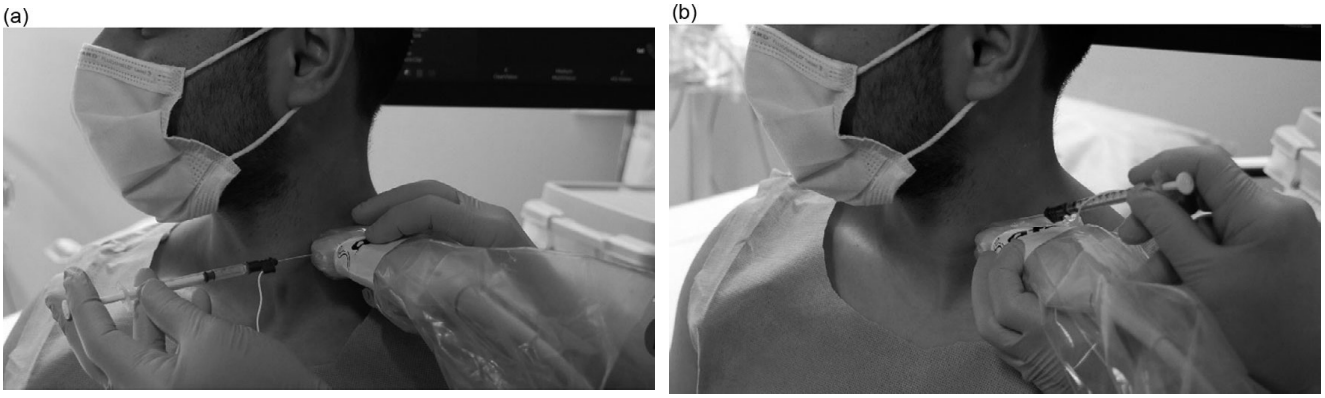

Fig. 36.7 (a) Scalenes in-plane approach. (b) Scalenes short-axis, out-of-plane approach.

reported in the literature for onabotulinumtoxinA is from 12 to 37.5 U in the anterior scalene and from 20 to 37.5 U in the middle scalene (Jordan *et al.*, 2007; Danielson and Odderson, 2008; Le *et al.*, 2010; Torriani *et al.*, 2010; Finlayson, 2011; Donahue, *et al.*, 2020). Up to 30 units of "BTX-A (product not specified)" is reported in the middle scalene muscle (Rahman, *et al.*, 2019). Reconstitution in all studies was with preservative-free saline. The reconstitution volume was not reported by all authors and varied from 1–2 ml per 100 units of onabotulinumtoxinA (Jordan *et al.*, 2000; Torriani et al., 2010) up to 10 ml per 30 units

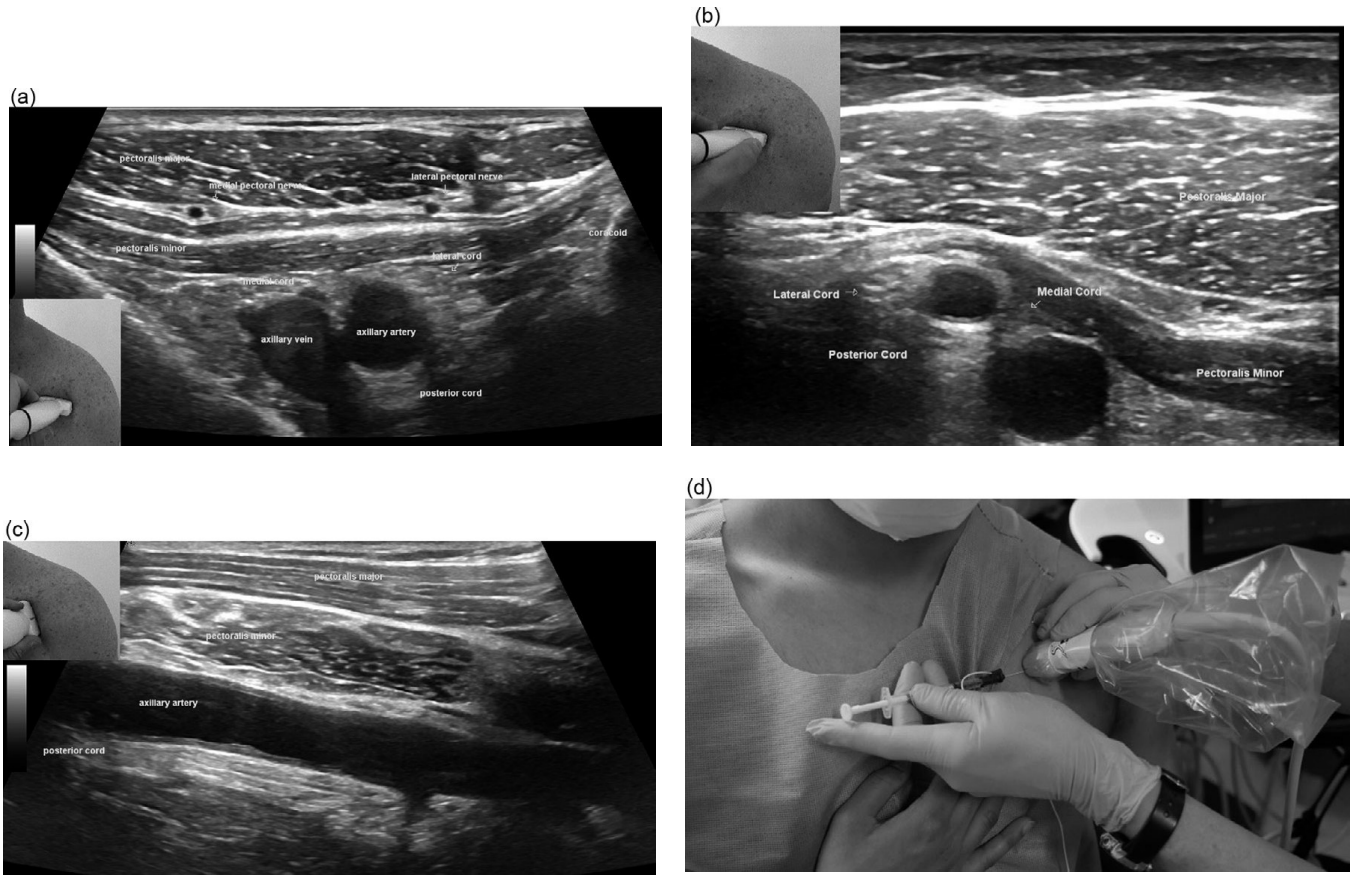

Fig. 36.8 (a) Short-axis B mode image, pectoralis minor, subclavicular brachial plexus, normal/asymptomatic (B-mode image provided by Jeff Strakowski). (b) Short-axis B mode image, pectoralis minor bowing compressing subclavicular brachial plexus, symptomatic patient (B-mode image provided by Jeff Strakowski). (c) B-mode US image, long-axis image, infraclavicular brachial plexus, axillary artery (B-mode image provided by Jeff Strakowski). (d) Pectoralis minor oblique long-axis, in-plane injection approach.

of "BTX-A" (Rahman *et al.*, 2019). Medline and PubMed searches did not elicit dosages of other BoNT products for the scalene muscles for treatment of TOS.

Pectoralis Minor Injections

The pectoralis minor lies deep to the overlying pectoralis major. The origin is on ribs 3–5, and insertion is on the coracoid process of the scapula. The pectoralis major forms a bridge from the thorax to the coracoid process/upper limb beneath which the brachial plexus and vascular structures travel into the arm (Fig. 36.8a). The actions of the pectoralis minor are to depress the shoulder and draw the scapula inferior and forward toward the chest wall and the posterior angle away from the chest wall. The cords of the brachial plexus may be compressed under the muscle fibers or the tendon of the pectoralis minor (Fig. 36.8b).

For US-guided BoNT injections, the pectoralis minor is visualized in short axis at its insertion on the coracoid process (Figs. 36.4a, 36.8a). The muscle can also be imaged in an oblique imaging plane visualizing the underlying neurovascular structures in long-axis (Fig. 36.8c) or in longitudinal plane (Fig. 36.8d) (Torriani *et al.*, 2010). The coracoid process is used as a landmark, and the transducer is moved inferiorly and medially to visualize the pectoralis muscle. The position of the axillary vessel and nerve are observed. An in-plane injection approach from lateral to medial is used, inserting the needle parallel to the chest wall superficial to the ribs to avoid entry into the intercostal space and underlying lung tissue (Fig. 36.8d). As noted above, the injection of botulinum toxin for TOS, regardless of botulinum toxin serotype, product or muscle is "off-label" in the USA. Reported doses of onabotulinumtoxinA for the pectoralis minor muscle range from 15 U (Torriani *et al.*, 2010) to 35 U (Jordan *et al.*, 2007). Reported dilution of 2 ml preservative-free saline per 100 U onabotulinumtoxinA was reported by Torriani *et al.*, (2010) and up to 30 units in 10 ml for "BTX-A" (Rahman *et al.*, 2019).

Subclavius Muscle Injections

The origin of the subclavius muscle is at the junction of the costal cartilage and first rib. The muscle runs from its origin to its insertion on the underside of the middle one-third of the

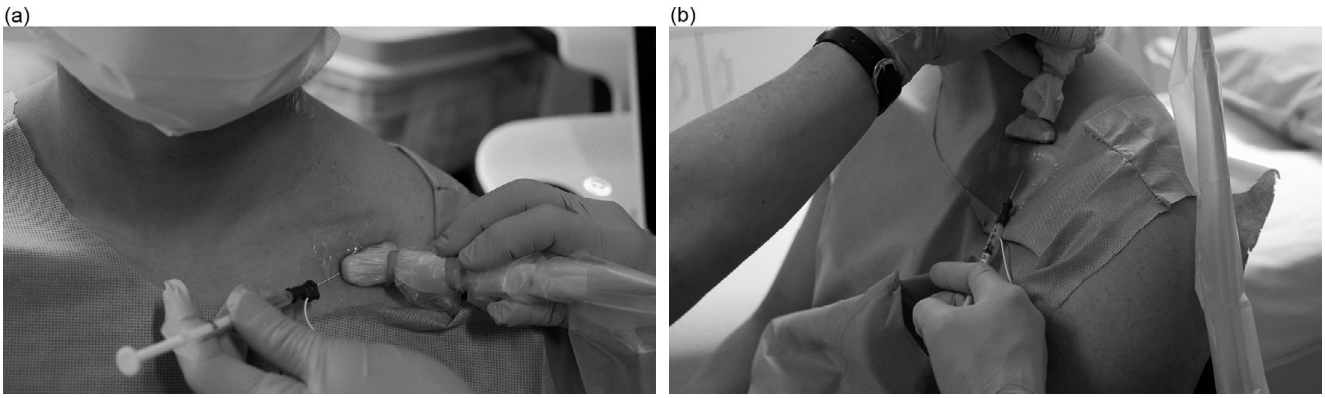

Fig. 36.9 (a) Subclavius long-axis, in-plane approach. (b) Subclavius, out-of-plane approach.

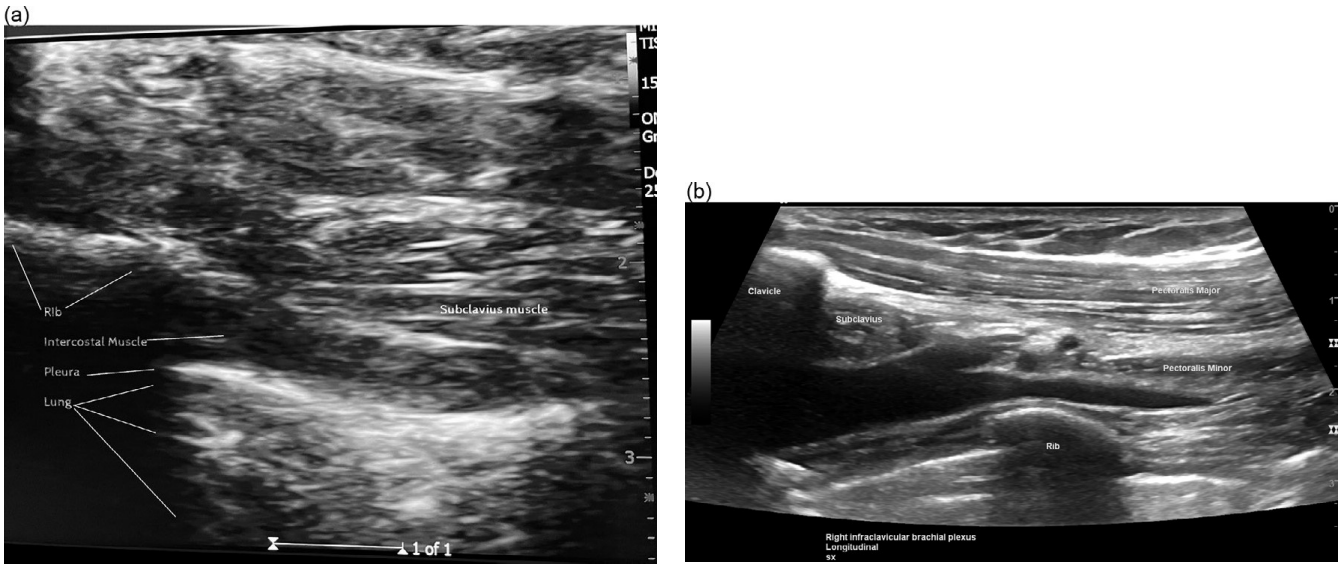

Fig. 36.10 (a) Long-axis B-mode image of subclavius muscle. (b) B-mode US image, subclavius, infraclavicular brachial plexus. (Provided by Jeff Strakowski.)

clavicle. It has several actions, including drawing the shoulder medial, raising the first rib or depressing the clavicle. Because the subclavius narrows the space between the first rib and clavicle, structures traversing this region may be compressed by a hypertrophied or overactive muscle or poor shoulder posture.

A hypertrophied or aberrant subclavius is reported to be one potential source of neurovascular compression in TOS. Ultrasound-guided injections are reported by several authors (Ozçakar *et al.*, 2010; Jordan *et al.*, 2007; Torriani *et al.*, 2010, Cavallieri et al., 2021); however, the precise techniques for US-guided injection of this muscle is not described in any of the published articles.

Ultrasound guidance for subclavius injections is typically performed with the transducer placed inferior and parallel to the clavicle (Fig. 36.9a). Less frequently, the transducer is placed superior to the clavicle (Fig. 36.9b). The transducer is then rocked or toggled to steer the US beam under the clavicle to visualize the muscle. The smaller footprint of a hockey stick transducer is useful when scanning the subclavius. The needle is then inserted using an in-plane approach from medial to lateral, directing the needle between the clavicle and first rib (Fig. 36.9a). Care must be taken to keep the needle parallel and superficial to the underlying chest wall to avoid penetration of the intercostal muscles and lung or neurovascular structures (Fig. 36.10a,b). Botulinum toxin for the treatment of TOS, regardless of muscle, botulinum toxin serotype or manufacturer, is "off-label" in the USA. Dosage for onabotulinumtoxinA injections in the subclavius ranged from 12 U (Torriani *et al.*, 2010) to 35 U (Jordan *et al.*, 2007). Torriani *et al.* (2010) reported a dilution of 2 ml preservative-free saline per 100 U onabotulinumtoxinA. Cavallieri *et al.* (2021) reported a dosage of 100 units of abobotulinumtoxinA diluted in 100 ml preservative-free normal saline into the subclavius muscle for TOS symptoms.

Summary

While some studies report that BoNT provides long-lasting symptomatic relief for some patients with TOS, others have reported only a short duration of, or limited degree of, benefit. Possible factors in this variation include patient selection, proper diagnosis, dose of BoNT, injection-targeting technique and post-injection therapy or intervention. Injections of BoNT are clearly less invasive than surgical intervention and may provide some patients with long-lasting symptomatic improvement. In other patients in whom BoNT injections provide inadequate or short-lived improvement, this intervention may help to guide surgical procedures.

Because of the risks associated with needle insertion and injection of target muscles affected in TOS, clinicians who perform BoNT injections must be highly skilled and should consider US or other imaging-based guidance (fluoroscopy or CT) for these injections. The addition of EMG to US guidance provides information about muscle activity, which may be useful when selecting muscles for injection. The muscles targeted for TOS injections are adjacent to many important neurovascular structures and other organs; consequently, US guidance reduces the risks associated with these high-stakes procedures. Additional research is necessary to clarify the role of BoNT therapy for TOS, including; whether specific subtypes of TOS or patient populations may benefit from BoNT intervention, the optimal dosage of each of the available botulinum toxin products and the role of guidance or injection technique.

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