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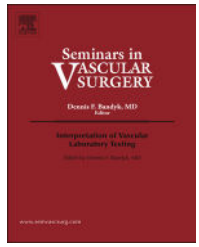

Thoracic outlet and pectoralis minor syndromes

Richard J. Sanders, MD, and Stephen J. Annest, MD*

Presbyterian/St. Luke's Medical Center, 1719 Gilpin, Denver, CO 80218

ARTICLE INFO

ABSTRACT

Compression of the neurovascular bundle to the upper extremity can occur above or below the clavicle; thoracic outlet syndrome (TOS) is above the clavicle and pectoralis minor syndrome is below. More than 90% of cases involve the brachial plexus, 5% involve venous obstruction, and 1% are associate with arterial obstruction. The clinical presentation, including symptoms, physical examination, pathology, etiology, and treatment differences among neurogenic, venous, and arterial TOS syndromes. This review details the diagnostic testing required to differentiate among the associated conditions and recommends appropriate medical or surgical treatment for each compression syndrome. The long-term outcomes of patients with TOS and pectoralis minor syndrome also vary and depend on duration of symptoms before initiation of physical therapy and surgical intervention. Overall, it can be expected that >80% of patients with these compression syndromes can experience functional improvement of their upper extremity; higher for arterial and venous TOS than for neurogenic compression.

1. Introduction

Compression of the neurovascular bundle of the upper extremity can occur above or below the clavicle. Above the clavicle, compression occurs in the scalene triangle, bordered by the clavicle, first rib, and scalene muscles, producing thoracic outlet syndrome (TOS). Below the clavicle, it occurs under the pectoralis minor muscle (PMM), producing pectoralis minor syndrome (PMS). For brachial plexus compression, it is common for both thoracic outlet and pectoralis minor areas to be involved simultaneously in the same patient, an example of the double crush syndrome [1].

Neurologic and vascular manifestations are essentially the same whether compression occurs above or below the clavicle. The nerves are components of the brachial plexus; the vascular structures are the subclavian artery and vein above the clavicle and the axillary artery and vein below the clavicle. The large majority of patients have brachial plexus compression giving rise to neurogenic TOS (NTOS) and/or neurogenic PMS (NPMS). Much less common is subclavian and axillary vein obstruction giving rise to venous TOS (VTOS) or venous PMS (VPMS). Least common is arterial compression causing arterial TOS (ATOS) occurring in <1% of all TOS patients. Arterial involvement of the axillary artery is much less common, seen primarily in athletes who engage in high-intensity, upper-extremity physical activity.

2. General features of TOS and PMS compression syndromes

2.1. *Classification*

There are three types of TOS and PMS: neurogenic, venous, and arterial. In the past, NTOS has been subdivided into three types: true, disputed, and traumatic. True NTOS was defined

* *Corresponding author.*
E-mail address: rsanders@ecentral.com (R.J. Sanders).

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as NTOS with objective findings, by imaging studies, electrodiagnostic studies, and physical findings of muscle atrophy. Disputed NTOS was defined as patients with the same symptoms but no objective findings. However, this classification of NTOS has been abandoned by most physicians treating TOS patients because the subdivision of NTOS is no longer useful and the term *disputed* has been discarded [2]. Another reason for eliminating the term is its implication suggesting that the patient's condition is not real.

Experiences over the past 50 years reveal that the vast majority of NTOS patients who fell into the disputed group do indeed have objective findings of abnormal electrodiagnostic studies, specifically abnormal measurements of the medial antebrachial cutaneous nerve and slowing of C8 nerve conduction velocity. In addition, follow-up of surgery for the true and disputed groups has shown that poorer results were seen in the true NTOS group because diagnoses were delayed and irreversible disabilities developed before atrophy occurred in the hand. In contrast, the patients in the disputed group are usually operated on before irreversible changes occur. Most of the patients in the true group had cervical or abnormal first ribs. It is now known that these patients should be operated on once symptoms develop. Once signs of atrophy occur, the changes can be permanent.

With regard to the classification of traumatic TOS, this was defined as patients who developed symptoms after fractures of the clavicle. However, many observations reveal that the majority of patients with NTOS develop symptoms after neck trauma, not clavicular fractures. There seems to be little use for the definition of traumatic TOS being used for clavicular fractures alone when other types of trauma are much more common but are not included. These other types include auto collisions, falling down stairs, slipping on floors or ice, and repetitive stress injuries from working long hours on assembly lines or typing on keyboards.

2.2. *Anatomy*

The contents of the neurovascular bundle remain essentially the same as the bundle passes from above the clavicle in the scalene triangle, to below the clavicle in the subpectoral area. The nerves and blood vessels make few changes, other than giving off a few small branches. In contrast, the structures surrounding the bundle change as the neurovascular bundle descends from the scalene triangle through the costoclavicular space and under the PMM (Fig. 1A).

The scalene triangle consists of two sides formed by the anterior and middle scalene muscles and the first rib forming its base (Fig. 1B). Inside the triangle are the roots and trunks of the brachial plexus and the subclavian artery. The subclavian vein lies just anterior to the anterior scalene muscle.

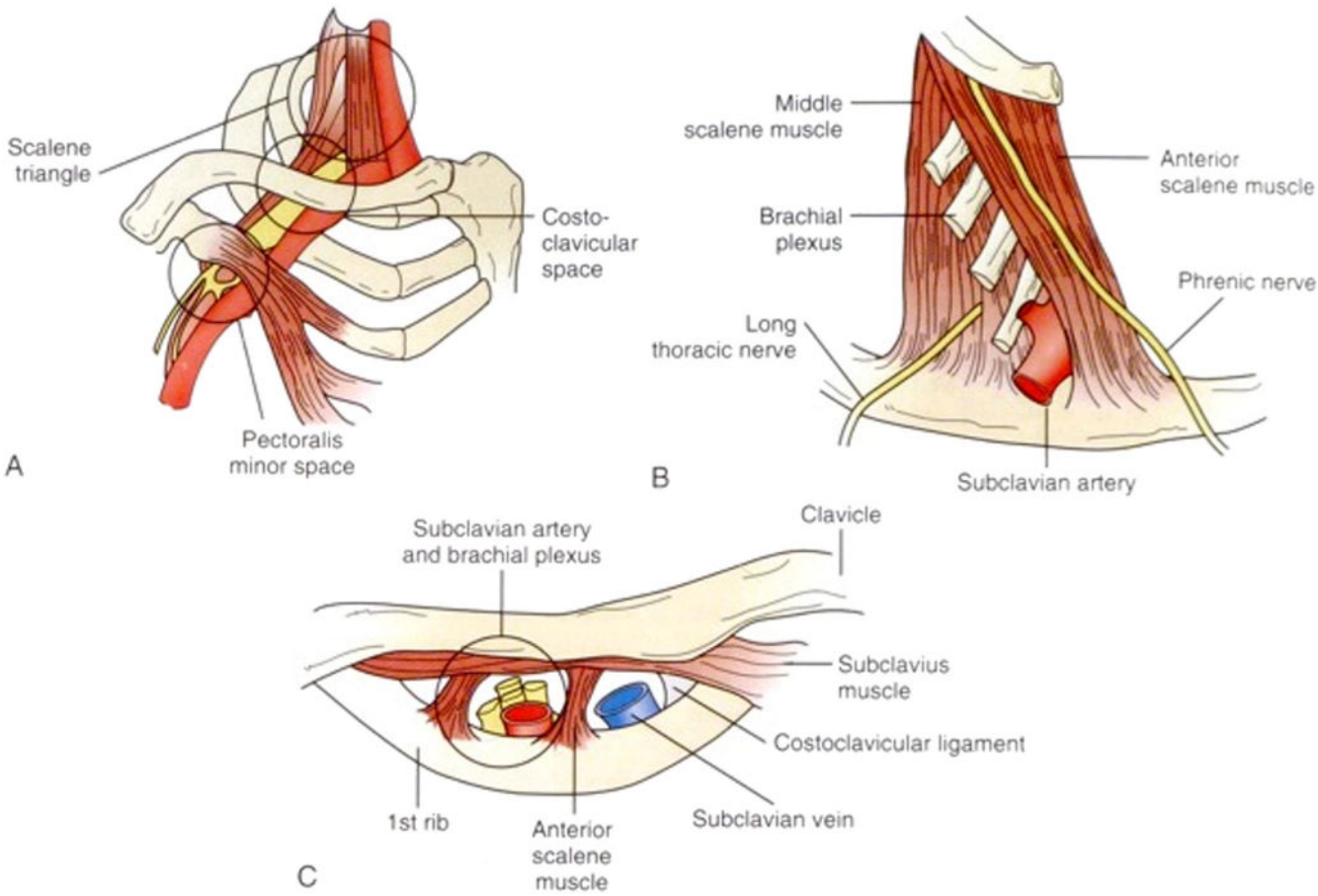

Fig. 1 – (A) The three spaces in thoracic outlet syndrome and pectoralis minor syndrome. (B) Scalene triangle. (C) Costoclavicular space. (Color version of figure is available online.)

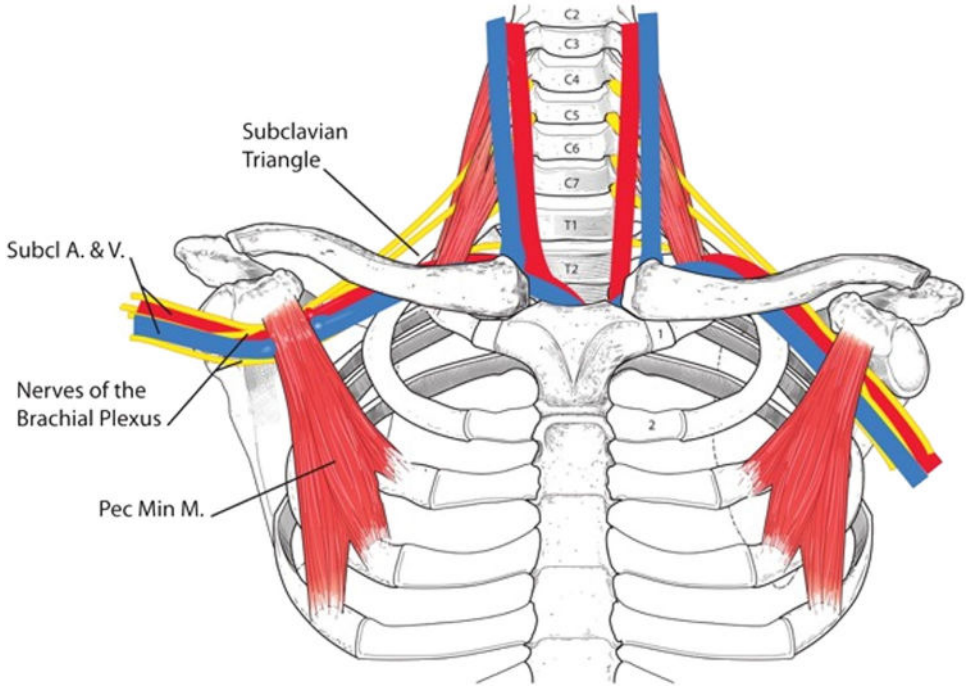

Fig. 2 – Pectoralis minor space. Pec Min M., pectoralis minor muscle; Subcl A. & V., subclavian artery and vein. (Color version of figure is available online.)

The bundle then passes below the clavicle and above the first rib into the costoclavicular space. This space may be important in VTOS, but its role in NTOS and ATOS is probably minimal (Fig. 1C). Passage under the PMM is the next space of importance, more important then the costoclavicular space, unless there is a clavicular deformity (Fig. 2).

2.3. *Etiology*

The several causes of TOS and PMS include congenital abnormal ribs, repetitive stress activities, acute trauma, and, rarely, clavicular abnormalities after clavicular fractures.

2.3.1. *Congenital*

Cervical and anomalous first ribs are often incorrectly regarded as the most common cause of TOS. Cervical ribs are uncommon, occurring in roughly 0.7% of the population, and are twice as common in women as in men [3]. The large majority of cervical ribs are asymptomatic and most patients with cervical ribs will live out their lives without being aware of their congenital extra rib. When cervical ribs do elicit symptoms, they are most likely to be neurogenic. ATOS is the next most common type of TOS caused by congenital rib abnormalities. VTOS is occasionally due to a cervical or anomalous first ribs, but this is rare.

2.3.2. *Repetitive activities*

A variety of repetitive activities can cause TOS, most often neurogenic but also venous and occasionally arterial. Working on assembly lines or keyboards can cause NTOS and/or NPMS.

VTOS or VPMS can also be caused by repetitive stress injury, particularly raising the arm repeatedly overhead. In children, particularly teenagers, competitive sports involving use of the upper extremities is the most common cause of NPMS and sometimes NTOS.

ATOS is usually associated with abnormal ribs, either complete cervical ribs or anomalous first ribs. Arterial pectoralis minor syndrome (APMS) is limited to athletes who throw overhead, particularly baseball pitchers and volleyball players, or workmen who work much of the time with their arms overhead.

2.3.3. *Acute trauma*

Hyperextension neck injuries, such as whiplash injuries, are the most common cause of NTOS. Other types of neck injuries that have in common stretching the neck backward include falls on ice, down stairs, or on slippery floors. These all can cause hyperextension of the neck or a sudden pull on the PMM. The shoulder strap of seat belts can injure the PMM in auto collisions. Most acute trauma injuries cause neurogenic, not vascular, symptoms.

3. Brachial plexus compression: NTOS and NPMS (Appendix I)

3.1. *Symptoms*

Symptoms of nerve compression or irritation in the upper extremity are the triad of pain, paresthesia, and weakness (Table 1). Pain can be in the hand, forearm, elbow, upper arm, shoulder, and/or over the trapezius muscle. Pain, regardless of how it is described (such as aching, general "discomfort," burning, or shooting), in all its forms is still pain. Much time is spent asking patients to describe the "nature" of their pain, but from a practical point of view, a description of the pain is seldom useful.

Table 1 – Symptoms of neurogenic thoracic outlet syndrome and pectoralis minor syndrome.

Pain (or soreness)
Neck
Trapezius
Supraclavicular area
Chest just below clavicle over pectoralis minor tendon
Axilla
Shoulder
Upper arm
Elbow
Forearm
Hand
Paresthesia
All five fingers
Fourth and fifth fingers
First to third fingers
Weakness
Hands
Arms

Paresthesia is a good term to describe numbness and tingling, as well as other "funny feelings" people experience but cannot articulate. Weakness usually does not appear until symptoms have been present for several months. Weakness is manifest by complaints of dropping things like dishes from the hands or by difficulty in gripping objects.

NTOS and NPMS have certain symptoms in common:

1. Pain in the trapezius, shoulder, arm, and forearm
2. Paresthesia in the hand, involving any combination of fingers. Involvement of all five fingers is most common, with more involvement of the fourth and fifth fingers. In some patients, the fourth and fifth fingers may be the only ones involved; less often the first three fingers are the only ones involved.
3. Weakness of the hand, dropping things, and poor grip are present in both conditions when symptoms have been present for more than several months.

3.2. *Distinguishing NTOS from NPMS*

There are differences in symptoms between NTOS and NPMS. In NTOS, occipital headaches and neck pain are often prominent complaints, while in NPMS, headaches and neck pain are absent or minimal. In contrast, NPMS patients have pain and/or tenderness in the anterior chest wall and in the axilla. If these symptoms are absent, NPMS probably is not present.

When major symptoms of occipital headache plus pain in the neck, chest, and axilla are all present, NTOS and NPMS coexist. This is the most common presentation seen in patients with brachial plexus compression.

3.3. *Physical examination*

Physical examination for brachial plexus compression is extensive. It includes eliciting tenderness over several muscles, Tinel's sign over specific areas, and four provocative maneuvers that are specific for brachial plexus compression. These are listed in Table 2. Although no single positive finding is diagnostic of brachial plexus compression, finding several positive responses on physical examination is highly suggestive. In addition, the four provocative maneuvers, and particularly the upper limb tension test [4] (Fig. 3), are extremely important. If the upper limb tension test is negative, the diagnosis of brachial plexus compression is very unlikely. False negatives are rare.

3.3.1. *Adson maneuver*

In 1927, Adson introduced his famous maneuver that is still taught more than 85 years later [5]. The Adson test is performed by feeling the radial pulse of the symptomatic side, while the head rotates to the same side and the patient takes a deep breath. A positive response is noting a decrease or absence of the radial pulse and the onset of the patient's neurologic symptoms of pain and paresthesia in that extremity. Over the years, many teachers have forgotten about the neurologic symptoms and simply emphasize the decreased pulse. This test is no longer recommended because it is too unreliable. Several investigators have demonstrated that many asymptomatic people lose their pulse with this maneuver while many symptomatic patients fail to do so [6–10]. The reason for this is that the test relies on a vascular sign to diagnose a neurologic condition.

The symptoms of pain, paresthesia, and weakness are common to neurogenic compression in several areas of the upper extremity. These areas include the wrist, forearm, elbow, pectoralis minor space, as well as the scalene triangle. For this reason, physical examination should routinely survey each of these areas to detect tender spots and positive Tinel's signs. Positive responses at the wrist, forearm, and elbow may be due to pathology in their respective areas, or they may be due to pathology in more proximal areas, such as the pectoralis minor space or thoracic outlet. Another possibility is that there may be pathology in more than one area of the upper extremity. This has been described as double crush syndrome [1]. Common double crush situations are the combination of NTOS with carpal tunnel syndrome or cuboid tunnel syndrome. Another double crush combination is NTOS and NPMS. When there is a double crush, it is

Table 2 – Physical examination for neurogenic thoracic outlet syndrome and pectoralis minor syndrome.

Areas of tenderness
Anterior scalene muscle
Biceps and rotator cuff tendons
Trapezius and rhomboid muscles
Pectoralis minor
Axilla
Tinel's sign
Anterior scalene
Carpal tunnel
Medial epicondyle
Pronator tunnel
Radial tunnel
Provocative maneuvers
Upper limb tension test (Fig. 3)
Elevated arm stress test
Neck rotation (Fig. 7B)
Head tilt (Fig. 7A)

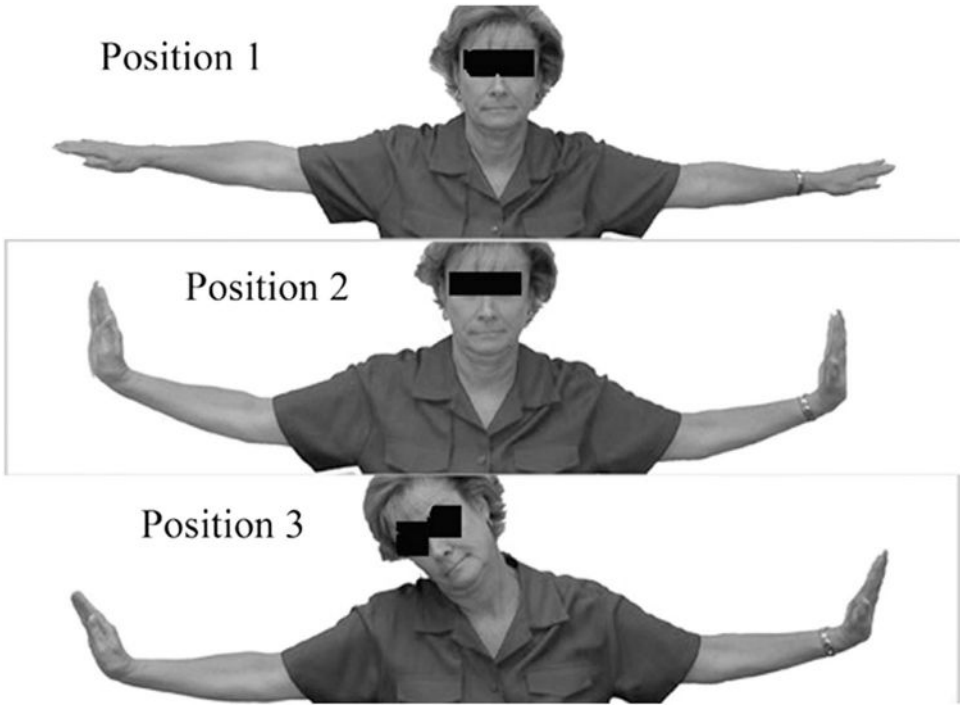

Fig. 3 – Upper limb tension test. Position 1: Arms at 90 degrees, elbows extended. Position 2: Wrists dorsi-flexed. Position 3: Head tilt.

sometimes possible to relieve symptoms by treating just one of the areas and the other site of compression becomes minimally symptomatic.

3.4. *Etiology*

NTOS is caused by hyperextension neck injuries; NPMS is caused by activities that pull the scapula back, thereby stretching the PMM (Table 3). Both types of stretch injuries can occur from the same incident. Supporting evidence that the shoulder strap of a seat belt can traumatize the PMM below it is the observation that car drivers with seat belts over their left shoulder more often develop left NPMS, while passengers in the same accident develop right NPMS.

Falls down stairs, or on slippery floors or icy sidewalks, can frequently cause shoulder injuries along with hyperextension neck injuries. The immediate pain from the shoulder injury often predominates so that the injured person does not become aware of the symptoms from the neck injury until a few weeks, or even months, later. A major reason for delayed onset of compression symptoms is that the symptoms are due to the muscle scar tissue formation, a part of the healing process, which can take several months to develop. Thus, a detailed history of all injuries should be obtained from patients complaining of hand paresthesia.

Repetitive stress injuries occur from a variety of activities. Sitting and typing on keyboards and keypads is common. Assembly line workers doing the same motions for several hours a day, grocery store stockers and cashiers, and athletes in competitive sports involving throwing overhead, are other examples of occupations subject to repetitive stress injury. Competitive sports is another area where repetitive activities with the upper extremities can lead to neurogenic symptoms. This is the most common area for teenagers. In this category, the most frequent activities seen are swimming, volleyball playing, throwing balls, and weight lifting. People who exercise doing large numbers of pushups also put considerable strain on their scapulae, hence their PMMs. In all instances, the pathology is the same: muscle injury causing muscle fibrosis that results in nerve impingement.

Table 3 – Etiology of neurogenic thoracic outlet syndrome and pectoralis minor syndrome.

Neck trauma causing hyperextension injuries
Auto accidents—rear-end collisions causing whiplash injuries
Falls down stairs, on ice, on slippery floors
Repetitive stress injuries—working on keyboards, assembly lines, weight lifting
Cervical and anomalous first ribs—put pressure against the lower trunk of the brachial plexus causing symptoms in the fourth and fifth fingers
Competitive sports—swimming, volleyball, baseball, football
Predisposing factors
Cervical and anomalous first ribs
Elongated C7 transverse process

3.5. *Predisposing factors*

Cervical and anomalous first ribs are primarily predisposing factors that make a person more likely to develop symptoms should neck trauma occur. However, some patients with these abnormal ribs become symptomatic without recalling any previous trauma, in which case the abnormal rib is the etiology.

An elongated C7 transverse process is another congenital variation that is virtually always accompanied by a ligament or band. These congenital bands run from the tip of the C7 transverse process, through the belly of the middle scalene muscle, to insert on the first rib. Other congenital bands are also seen in the middle scalene muscle and these, too, act as predisposing factors. Patients with these predisposing factors usually remain asymptomatic until neck trauma occurs.

3.6. *Diagnostic tests*

Muscle blocks with local anesthetics are very helpful in confirming a diagnosis of brachial plexus compression. The block is performed with the patient either lying down or sitting up. The technique has been described using anatomical markers, ultrasound, electromyography (EMG), and computed tomography guidance. Some practitioners have employed brachial plexus blocks for the same purpose as the muscle blocks. While muscle blocks are both diagnostic and prognostic, a brachial plexus block renders the arm numb and weak. If effective, it will remove all sensation from normal people as well as those with brachial plexus compression; it does not confirm the diagnosis.

Muscle blocks are performed only over tender muscles. Most patients seen for symptoms of brachial plexus compression demonstrate tenderness over both the anterior scalene muscle and PMM. Therefore, blocks are usually performed over both muscles during the same examination. Physical examination is performed after each block so that the extent to which each muscle is contributing to the patient's symptoms can be assessed. In a few patients, tenderness is noted only over one muscle, in which case only that muscle is blocked.

3.6.1. *PMM block*

The technique without ultrasound begins with localizing the most tender spot over the PMM, about 4 to 5 cm below the clavicle. Our preference has been for short-acting lidocaine as the anesthetic of choice. This is preferred because, occasionally, lidocaine runs onto some of the nerve branches of the plexus, causing increased numbness and/or weakness in the upper extremity. By using a short-acting drug, the symptoms will last no more than 30 minutes. In the large majority of patients, the effect of lidocaine spilling onto nerves is gone in 5 or 10 minutes and the test can then be continued. However, if the effect on the brachial plexus lasts longer than 10 minutes, the test is of no value and must be repeated at a later date.

The block is performed by injecting 4 cc of 1% lidocaine into the PMM using a 5-cc syringe and a #22 needle, 1 ½-inch long. This is introduced at a 45-degree angle, pointing cephalad, to avoid entering the chest and causing a pneumothorax. Because the PMM is oriented in an anterior-posterior position, the lidocaine is spread out over an area approximately 2 cm wide and 1 to 2 cm deep by injecting 0.3 to 0.5 cc lidocaine into one spot at a time and repositioning the needle after each small injection. After each repositioning of the needle, the syringe is aspirated to assure that the needle tip is not in a blood vessel. If blood is aspirated, the needle is backed up a few millimeters, moved to a slightly different spot, and re-aspirated to make sure the injection avoids a blood vessel; the injection then continues.

An effective block is determined by loss of the tenderness over the PMM within 1 to 2 minutes. (If the patient has no PMM tenderness on initial physical examination, a PMM block is not performed). If there were symptoms of pain and paresthesia at rest before the block, the patient is asked to describe the degree of improvement that has occurred a few minutes after the block. The physical examination is then repeated within a few minutes of completing the block. The positive findings before the muscle block are the baseline from which improvement is measured. A good response to the block is improvement in the majority of positive finding that were present before the block. This response is recorded.

If residual symptoms remain after the repeat physical examination, a scalene muscle block is performed while the PMM block is still in effect. In this way it is possible to assess the roll of both NTOS and NPMS at the same time.

3.6.2. *Anterior scalene muscle block*

The most tender spot over the anterior scalene muscle is determined and the same protocol for the pectoralis minor block followed for the anterior scalene muscle block. The needle is inserted about 2 cm above the clavicle, through the sternocleidomastoid muscle and at a 45-degree angle, again to avoid entering the pleura. The muscle usually lies 1 to 3 cm below the skin, closer in thin-necked patients and deeper in thick-necked patients. As with the pectoralis minor block, the injection is spread out over an area about 1 to 2 cm wide and 1 to 3 cm deep. A good block is indicated by loss of tenderness in the area. Improvement in symptoms is recorded following the block, with the patient at rest. The physical examination is repeated after determining that the block was effective. As with the pectoralis minor block, a good response is improvement in the majority of positive findings that were present before the block.

3.6.3. *Electrodiagnostic tests*

In the past, EMG, with its many measurements, has been helpful in revealing changes typical of ulnar neuropathy. Unfortunately, this occurs in no more than in 5% of NTOS patients, specifically those with congenital cervical ribs, anomalous first ribs, or those with bands arising from a transverse process of C7. In the other 95% of NTOS patients who lack abnormal ribs or bands, the results of standard EMG studies are usually normal. However, it is now recognized that specific EMG measurements may be abnormal.

Measurement of the medial antebrachial cutaneous nerve (MAC) in NTOS patients was first introduced in 1993 by Nishida et al. [11]. Subsequently it was confirmed by Kothari et al. [12] in 1998. In both of these studies, patients had significant symptoms plus other objective findings. In 2004, Seror [13] reported that MAC sensory nerve action potential measurements were abnormal in 16 of 16 patients with clinical findings of mild NTOS. He described a significant reduction in the amplitude of the response to MAC stimulation. In 2008, Machanic and Sanders [14] reported MAC studies in 41 patients subsequently operated on for NTOS. Not only was reduction in amplitude noted, but also reduction in latency and slowing of C8 nerve stimulation. None of these patients had abnormal ribs, but all had clinical findings

supporting a diagnosis of NTOS. Forty of these 41 patients had abnormal findings in at least one of the four diagnostic criteria for MAC measurements. The four diagnostic criteria were: latency >2.4 ms, latency difference between sides of 0.3 ms or more, amplitudes <10 uv, and amplitude ratios of ≤0.5. C8 nerve root stimulation responses were below normal (56 M/s) in 54%. All of the patients who clinically improved and who were studied postoperatively showed improvement in their MAC measurements [14]. MAC and C8 nerve stimulation measurements have become the first consistent objective finding in the diagnosis of either NTOS or NPMS.

3.6.4. *Imaging studies*

In an earlier era, the only imaging study available was a plain anterior-posterior and lateral x-ray of the neck. This is still utilized, as it is the simplest and least expensive way of determining the presence or absence of a cervical or anomalous first rib (Figs. 4 and 5). Additionally, the lateral view can show straightening of the normal lordotic neck curve, indicating cervical spine muscle spasm or disease.

Today, a variety of imaging techniques are available that can be used to evaluate neck and shoulder pain. While these tests can be enlightening, they are unnecessary to diagnose NTOS or NPMS. With a good history, physical examination, positive response to muscle blocks, and abnormal responses to electrodiagnostic tests, additional, expensive, diagnostic studies are neither additive nor indicated. Magnetic resonance imaging (MRI) of the brachial plexus can at times show nerve compression, but many times will be normal [15]. However, the same MRI of the neck, when looking at the cervical spine, can demonstrate bulging discs or spinal or foraminal stenosis (Fig. 6). In the future, if MRI can detect intramuscular scarring, MRI could become more useful. Magnetic resonance angiography (MRA) is useful in diagnosing arterial or venous TOS, but is rarely indicated for neurogenic TOS. Ultrasound and computed tomography angiography (CTA) are seldom abnormal in patients with neurogenic TOS, although they may be helpful for arterial or venous TOS.

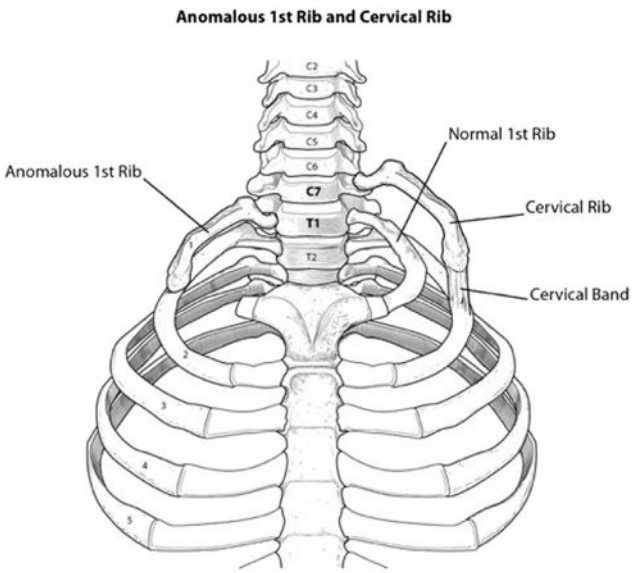

Fig. 4 – Rib cage showing anomalous first rib on right arising from T1 transverse process; cervical rib on left arising from C7 transverse process.

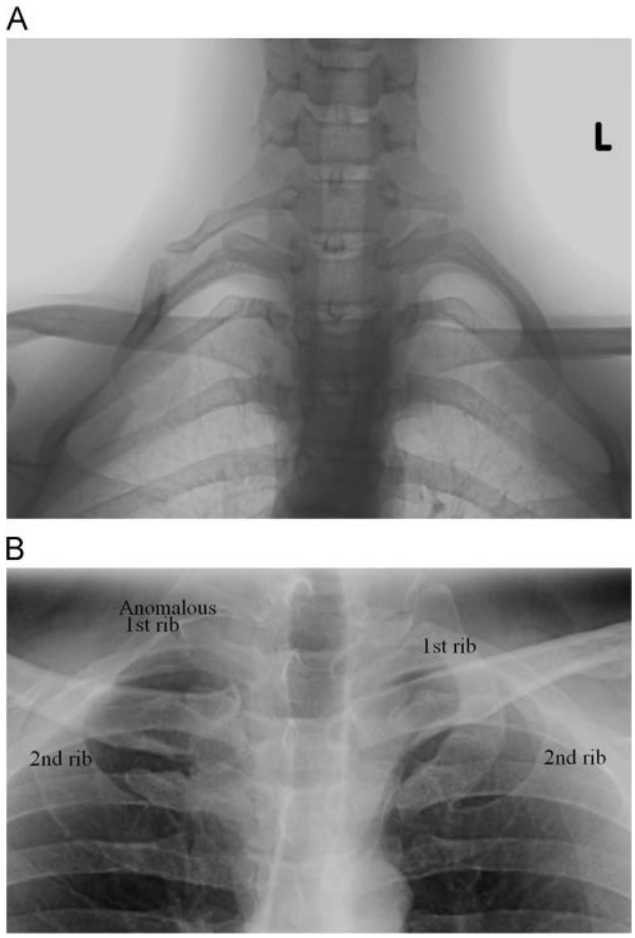

Fig. 5 – (A) X-ray of complete cervical rib with true joint to first rib on right; (B) x-ray of anomalous first rib on right.

3.6.5. *Neurography*

This is a technique that can demonstrate displacement or distortion of nerves of the brachial plexus [16]. This can help confirm a diagnosis when severe trauma has occurred. However, like other imaging techniques, when the diagnosis can be confirmed by less expensive methods, neurography is unnecessary.

3.7. *Differential and associated diagnoses*

The association of brachial plexus compression, both NTOS and NPMS, with other conditions is quite common and labeled double crush syndrome [1]. Among the diagnoses that can coexist with brachial plexus compression, or must be differentiated from it, are cervical spine disease, shoulder pathology, cuboid tunnel, carpal tunnel, pronator tunnel, and radial tunnel syndromes. Additional rare causes are Chiari malformation, Pancoast tumor, and multiple sclerosis. These are listed in Table 4. When associated diagnoses exist and conservative treatment has been unsuccessful, it is difficult to decide which diagnosis to operate upon first. In general, the condition that is most symptomatic will be treated first. If most symptoms are below the elbow, carpal, pronator, radial, and cuboid tunnel

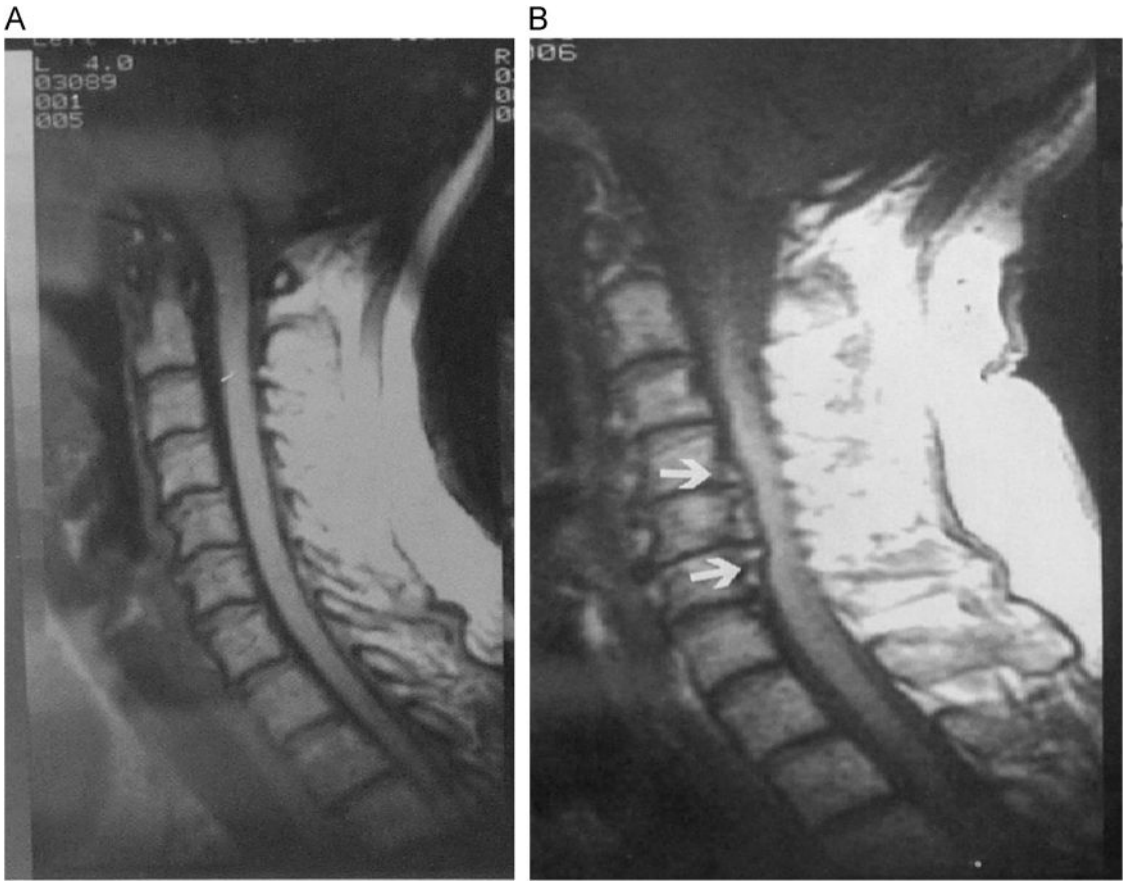

Fig. 6 – (A) Normal cervical magnetic resonance imaging (MRI). (B) MRI of cervical spinal stenosis (arrows).

compression will be treated first. When neck pain, occipital headaches, and shoulder girdle pain predominate, NTOS and NPMS will be treated first. Very often, symptoms from the second diagnosis may subside to the point that no further treatment is necessary. However, from time to time, a second or even third diagnosis will require treatment at a later time.

Coexistence of NTOS with NPMS is another form of double crush. Depending on responses to diagnostic tests and muscle blocks, just one of these sites of compression may be treated first and assessing the response before treating the second site.

3.8. *Treatment*

3.8.1. *Nonoperative treatment*

Treatment for NTOS and NPMS is either nonoperative or operative. Nonoperative therapy includes several modalities: Physical therapy (PT), chiropractic manipulation, osteopathic manipulation, acupuncture, anti-inflammatory drugs, muscle relaxers, and analgesics. In nearly all cases, with few exceptions, conservative therapy is tried first. If this fails, and if symptoms are severe enough to interfere with the patient's occupation, recreation, or activities of daily living, surgery becomes a consideration.

Table 4 – Differential and associated diagnoses.

Cervical spine disease
Shoulder pathology
Carpal tunnel syndrome
Cuboid tunnel syndrome (ulnar nerve compression at the elbow)
Pronator tunnel syndrome
Radial tunnel syndrome
Chiari malformation
Pancoast tumor
Multiple sclerosis

3.8.1.1. PT. NTOS and NPMS are treated together in PT. There are several protocols for PT, including the Edgelow protocol [17], which has been effective in some patients. Our preference for therapy includes the modalities listed in Table 5. The

Table 5 – Preferred and ineffective modalities of physical therapy for NTOS and NPMS.

Preferred modalities
Neck stretching
Pectoralis minor stretching
Posture correction
Nerve glides
Abdominal breathing
Dry needling
Ineffective modalities for NTOS and NPMS
Strengthening exercises
Resistance exercises
Therabands

Abbreviations: NPMS, neurogenic pectoralis minor syndrome; NTOS, neurogenic thoracic outlet syndrome.

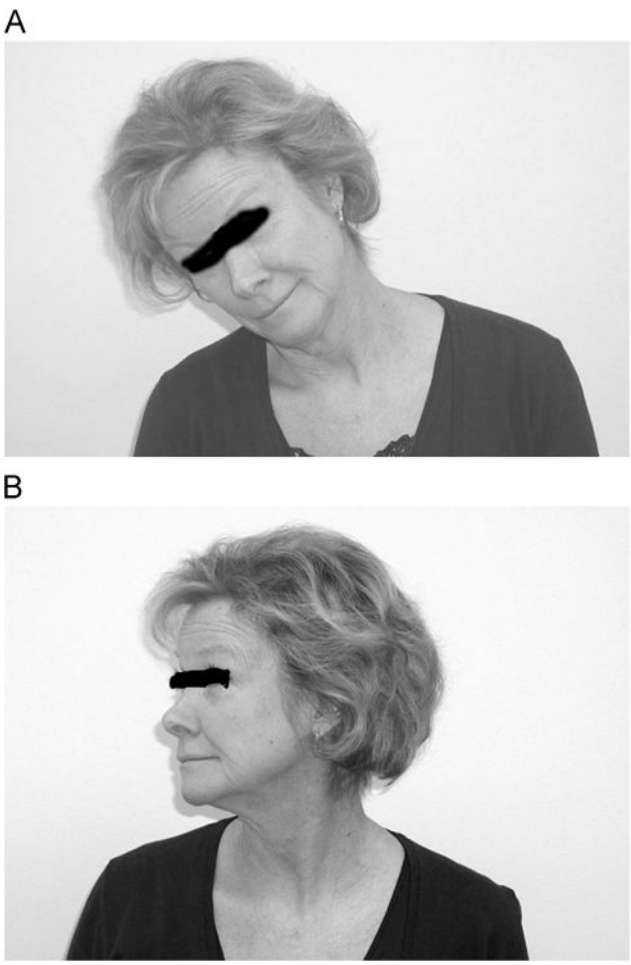

Fig. 7 – Neck stretching positions. (A) Neck rotation. (B) Head tilt.

stretching exercises should be performed three times daily. Each stretch is held for 15 to 20 seconds. Each stretch is followed by a rest for same length of time and is repeated two more times. Neck stretching includes two separate stretches: rotating the chin over the shoulder is one stretch (Fig. 7B); tilting the head, ear to shoulder (Fig. 7A), is the second neck stretch. Both neck stretches should be performed at each session. Pectoralis minor stretching can be performed in a few different ways. One of the preferred ways is standing in an open doorway with each hand on the door jam at shoulder height, the upper body falling forward without bending at the waist (Fig. 8). Another technique is placing the hand of the involved side against a wall with the elbow bent and rotating the body away from the hand.

Nerve glides are performed by extending the arms 90 degrees to the side with the elbows straight, and dorsiflexing the wrists back and forth 10 to 15 times.

Correct posture is holding the spine straight with the neck partially extended and chin flexed. The shoulders are in a neutral position, neither hunched forward nor hyperextended backward in military attention position (Fig. 9).

Abdominal breathing is performed with the patient lying supine, flat on the floor, preferably without a pillow. A hand is place on the abdomen over the umbilicus. Inspiration with the diaphragm pushes the hand upwards, expiration lets the hand fall. (With intercostal muscle breathing, the hand falls with inspiration and rises with expiration).

Therapy initially should continue for 3 months. Most modalities, such as stretching, nerve glides, and abdominal breathing, can be performed on a daily basis by the patient at home. An important role of the therapist is to instruct patients on how to perform each modality and follow-up with them to make sure they are performing them correctly. Some modalities, such as dry needling, manual therapy, and passive stretching, require hands-on treatment by a therapist. Other modalities include therapeutic ultrasound and electric nerve stimulation.

The Feldenkrais Method has been helpful to many patients [18]. The other forms of conservative therapy are often utilized on a trial and error basis. Each has had occasional success.

Nonoperative therapy is performed with the hope of avoiding surgery. In patients who partially improve with therapy, the prognosis for surgery is improved over those who show no improvement [19].

3.8.2. *Operative treatment*

Indications for surgery are symptoms severe enough to interfere with activities of daily living, work, and recreation, and inadequate relief of those symptoms by nonoperative modalities. The patient is clearly told that the results of

Fig. 8 – Pectoralis minor muscle stretch in open doorway.

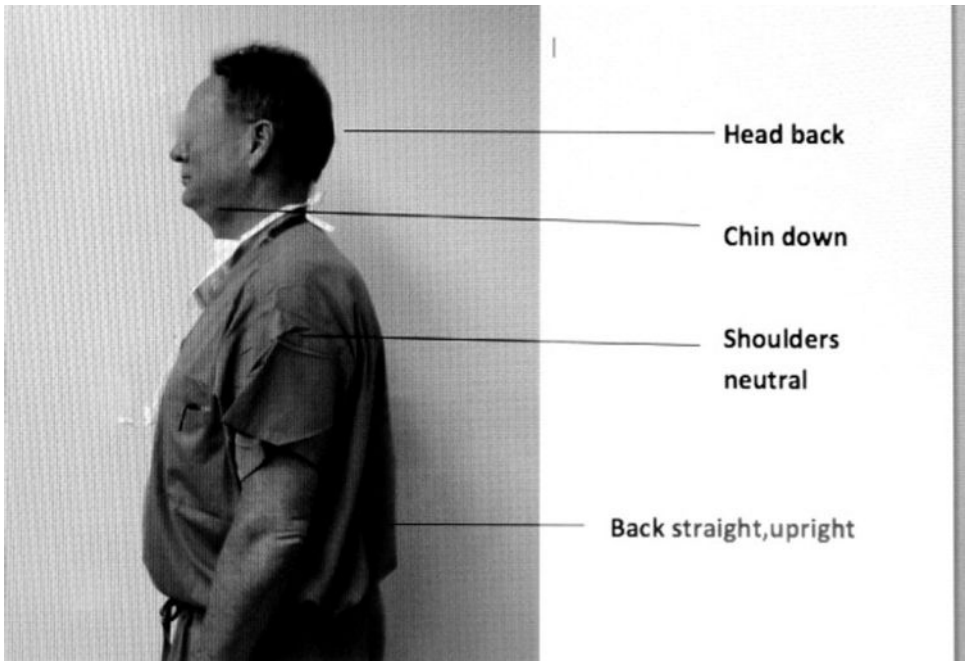

Fig. 9 – Correct posture. Head back; chin down; shoulders neutral, neither held forward nor backward, and spine straight, upright.

surgery cannot be guaranteed and, for most patients, good results means significant improvement, but not complete relief.

NTOS can be approached in five ways. The most direct way would be to remove the clavicle and put it back in place after completing the operation. However, this is rarely done because the clavicle does not always heal properly after it has been temporarily removed. Therefore, surgeons have found four other approaches to the area that leave the clavicle intact.

3.8.2.1. Approaches to the thoracic outlet. The four other approaches through which to reach the thoracic outlet area are: through the axilla—the transaxillary approach; going above the clavicle—the supraclavicular approach; going below the clavicle—the infraclavicular approach; or going behind the clavicle through the back, using a posterior incision along the scapula.

The last two approaches are seldom used. The posterior approach is used only by a few surgeons, usually those who were trained in thoracic surgery, and usually for recurrences. This approach requires cutting some of the back muscles, which on occasion, results in postoperative back pain, which can be debilitating and is hard to treat. Exposure of the plexus via the posterior approach is not as complete as through the supraclavicular approach.

The infraclavicular approach is used for reaching the anterior portion of the rib. It can be combined with a supraclavicular approach to achieve "complete" removal of the first rib, a procedure called "over and under" [20] or the paraclavicular approach [21]. However, it should be pointed out that in treating NTOS, even though the supraclavicular approach cannot reach the anterior portion of the first rib, experience has shown that it is unnecessary to remove the anterior rib portion when treating neural compression. On the other hand, when the anterior portion of the rib must be removed, which is true to VTOS, the infraclavicular approach can be used, particularly, if repair of the subclavian vein is anticipated. Otherwise, for VTOS, when only first-rib resection and subclavian vein venolysis is planned, the transaxillary approach is our preference.

The other two approaches, supraclavicular and transaxillary, are the most popular routes to decompress the thoracic outlet for NTOS. The two different approaches, through the neck or through the axilla, resolve the same symptoms. As of this writing, there is no significance difference in long-term results between the two operations [22].

3.8.2.2. The supraclavicular approach. This removes the pathologic scalene muscles that are compressing the brachial plexus. This approach provides the best exposure of the scalene triangle, its muscles, and the roots and trunks of the brachial plexus. Through this incision the entire anterior and middle scalene muscles can be excised, as well as cervical ribs and the posterior part of the first rib, the portion adjacent to the nerves. With this exposure one can determine whether the first rib can be spared or removed. This is the only approach that permits complete neurolysis of all brachial plexus roots and trunks. Additionally, it allows dissection of the dorsal scapular nerve in patients with prominent pain in the rhomboid area. In ATOS, this approach permits removal of abnormal ribs and also repair or replacement of the subclavian artery.

The primary disadvantage of the supraclavicular approach is that it is a slightly higher incidence of injury to the phrenic and long thoracic nerves and to the lymphatic ducts (Table 6). Its disadvantage in treating VTOS is that the anterior portion of the first rib cannot be removed and the subclavian vein cannot be decompressed.

3.8.2.3. The transaxillary approach. This takes the tension off the pathologic scalene muscles by shortening but not

Table 6 – Advantages and disadvantages of transaxillary and supraclavicular approaches.

Transaxillary approach
Advantages
Best approach for removal of anterior first rib
Probably an easier way to excise the middle of the first rib
Best approach for subclavian vein venolysis and venous TOS
Less chance of phrenic nerve injury
Do not have to dissect the entire brachial plexus
Rarely encounter lymphatics compared to supraclavicular approach
Disadvantages
Harder to excise posterior rib than SC approach
More likely to enter the pleura than SC approach
Exposure more difficult and a harder approach to teach to others
Cannot repair or replace subclavian artery through this route
Harder to repair injured subclavian artery or vein through this route
Cannot decompress the upper and middle trunk and C5, C6, and C7 roots
Supraclavicular approach
Advantages
Can remove most or all anterior and middle scalene muscles
Better exposure of all nerves and easier to identify congenital bands
Can elect to excise or leave the first rib as this rib is not the primary source of the pathology
Easier to evaluate and excise most cervical ribs
In arterial TOS, can repair or replace subclavian artery through neck
Can perform complete neurolysis of all five nerve roots
Easier to remove the posterior first rib close to the transverse process
Disadvantages
Cannot remove anterior part of first rib
More likely to injure phrenic and long thoracic nerves than transaxillary approach
Greater risk of lymphatic leakage than transaxillary approach

Abbreviations: SC, subclavicular; TOS, thoracic outlet syndrome.

removing the muscles. This is the best approach for freeing compressive structures from around the subclavian vein, including the anterior part of the first rib. This approach is convenient for performing pectoralis minor tenotomy (PMT) and then proceeding posteriorly to excise the first rib. The primary disadvantages of the transaxillary route are that exposure is more difficult and excision of the entire posterior portion of the rib may be harder than through the supraclavicular approach. In addition, because of the limited exposure, it is more complicated to teach trainees to perform this operation. The advantages and disadvantages of the two approaches are listed in Table 6.

3.8.2.4. Technique of supraclavicular approach. The patient is anesthetized and intubated, after which no further muscle-relaxing drugs are administered. The patient is placed in a beach-chair position, with the back elevated and legs down. A 5- to 7-cm transverse incision is made 1 to 2 cm above and parallel to the clavicle, beginning 1 cm off the midline (Fig. 10A). Subplatysmal flaps are elevated, sparing the external jugular vein (Fig. 10B). The sternocleidomastoid muscle is mobilized on its lateral edge down to the clavicle and 5 to 7 cm cephalad, seeking and avoiding sensory nerves, which are seen at this level. The sternocleidomastoid muscle is retracted medially.

A self-retaining retractor (Mini-Omintract®, Omni-tract Surgical, St. Paul, MN) is attached to the operating table and exposure achieved. One to two centimeters of the omohyoid are excised to prevent adherence of its ends to the brachial plexus. The scalene fat pad is opened vertically (Fig. 10C) and the C5 nerve root dissected free. It is unnecessary to initially identify the phrenic nerve. In most cases, the phrenic nerve is best sought after the brachial plexus has first been dissected. (However, an occasional lateral, main phrenic nerve is encountered next to C5 and this must be identified and protected.)

After finding C5, a small branch arising on its medial side should be sought, as it is the C5 contribution to the phrenic nerve (Fig. 10D). This is sometimes called the accessory phrenic. This branch, when present, is protected as C6 and C7 are dissected free. C8 and T1 are freed next, or if the lateral edge of the anterior scalene muscle is seen to be covering the lower brachial plexus (Fig. 10E), the anterior scalene muscle is dissected and the main phrenic nerve identified, usually on the medial edge of the anterior scalene muscle (Fig. 10F). In 13% of patients, the phrenic nerve lies on the lateral side of the anterior scalene muscle [23]. This makes dissection more difficult because this nerve, lying in the middle of the operative field, must be preserved. When necessary, a soft vessel loop is carefully used to move the phrenic aside.

The anterior scalene muscle is divided at its first-rib insertion with a Harmonic scalpel (Fig. 11, first from left) or scissors and bipolar cautery. The anterior scalene muscle is grasped and elevated with a clamp, freed from its attachments, and divided as high as safety permits, above the brachial plexus nerve roots (Fig. 10G). At this point, it is helpful to mobilize and control the subclavian artery for safety and to improve exposure of Sibson's fascia and its constricting bands.

With the anterior scalene muscle gone, exposure of the C8 and T1 nerve roots and lower trunk is now easier. Mesoneurial neurolysis of the brachial plexus is then completed by removing scar tissue and bands but leaving the loose areolar tissue between the nerve roots. A vessel loop around the brachial plexus is also helpful.

The middle scalene muscle is now dissected lateral to C5. Because the C5 and C6 branches of the long thoracic nerve lie in the belly of the middle scalene muscle, dissection is performed slowly, taking a few fibers at a time, to identify and preserve the long thoracic nerve and its two branches (Fig. 10H). The middle scalene muscle is dissected down to the first rib, removing enough fibers so that none are left contacting the brachial plexus. The C7 branch to the long thoracic nerve usually arises from C7 at a lower level than the C5 and C6 branches. Occasionally it is seen, but usually it is not in the field.

If the patient complained of pain along the medial edge of the scapula, over the rhomboid muscle area, dissection of the middle scalene muscle extends cephalad until the dorsal scapular nerve is identified and freed.

A decision is now made as to whether or not to excise the first rib. If the lower trunk of the brachial plexus is within 1 to

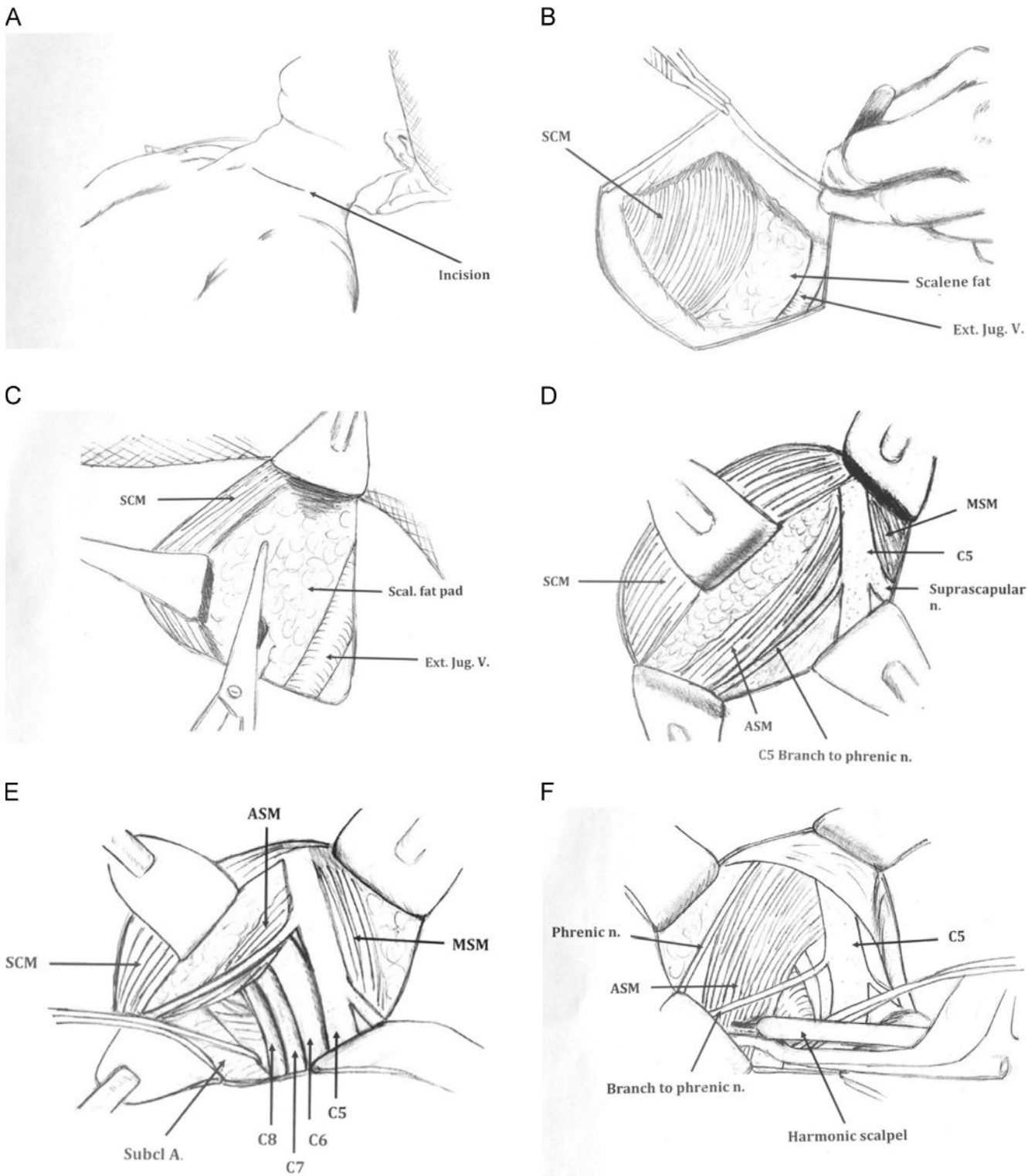

Fig. 10 – Technique of supraclavicular scalenectomy and first-rib resection. Drawings by artist Amalia Christman. (A) Incision, 1 to 2 cm above clavicle and 5 to 7 cm long. (B) Dissecting upper skin flap just superficial to sternocleidomastoid muscle (SCM) and below fat underneath platysma. Flap is elevated as far as possible. Ext. Jug. V., external jugular vein. (C) Vertical division of scalene fat pad. (D) Exposure of C5 and C5 branch of phrenic nerve (also called accessory phrenic nerve). Lateral edge of anterior scalene muscle (ASM) may be seen. Middle scalene muscle (MSM) is lateral to C5. (E) C5, C6, C7, C8, and T1 are dissected free. Subclavian artery (Subcl A.) exposed and surrounded with vessel loop. (F) ASM is divided near first-rib insertion with Harmonic scalpel. Phrenic n., phrenic nerve. (G) Freed lower end of ASM is grasped with clamp, elevated, and proximal end of ASM divided above C5 with Harmonic scalpel. If space is too tight, bipolar cautery and scissors are used. (H) MSM is divided with Harmonic scalpel after identifying C5 and C6 branches of long thoracic nerve (Long thoracic n.). Note vessel loop surrounds brachial plexus. (I) Neck of first rib is divided with Raney rongeur. If there is enough room, the Shumaker rib cutter may be used, seen in Fig. 11

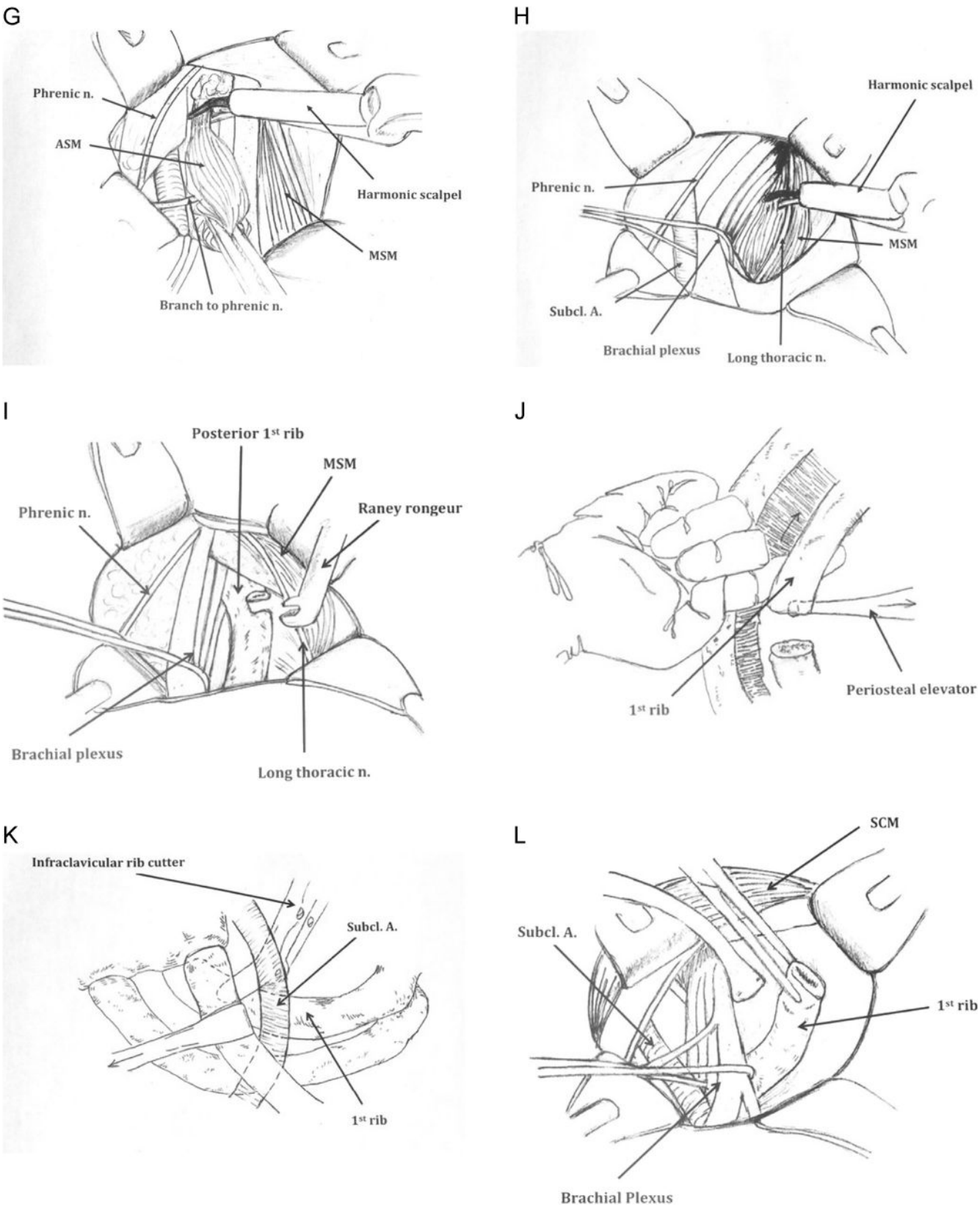

Fig. 10 – Continued. (J) One cm of first rib was excised and finger frees pleura from underside of rib. Rib is elevated with right angle end of a periosteal elevator, which allows finger to get behind rib. (K) Anterior end of rib is divided by an infraclavicular rib cutter (by Pilling). Subclavian artery is retracted upward and two retractor blades are removed to permit rib cutter to reach rib. Anterior rib stump is smoothed with Raney rongeur to prevent tip of rib from injuring subclavian artery lying above it. (L) Divided rib section is extracted from behind plexus and subclavian artery with Kocher clamp. Reprinted from Sanders RJ, Annest SJ. Technique of Supraclavicular Decompression for Neurogenic Thoracic Outlet syndrome. J Vasc Surg 2015;61:821-5, with permission.

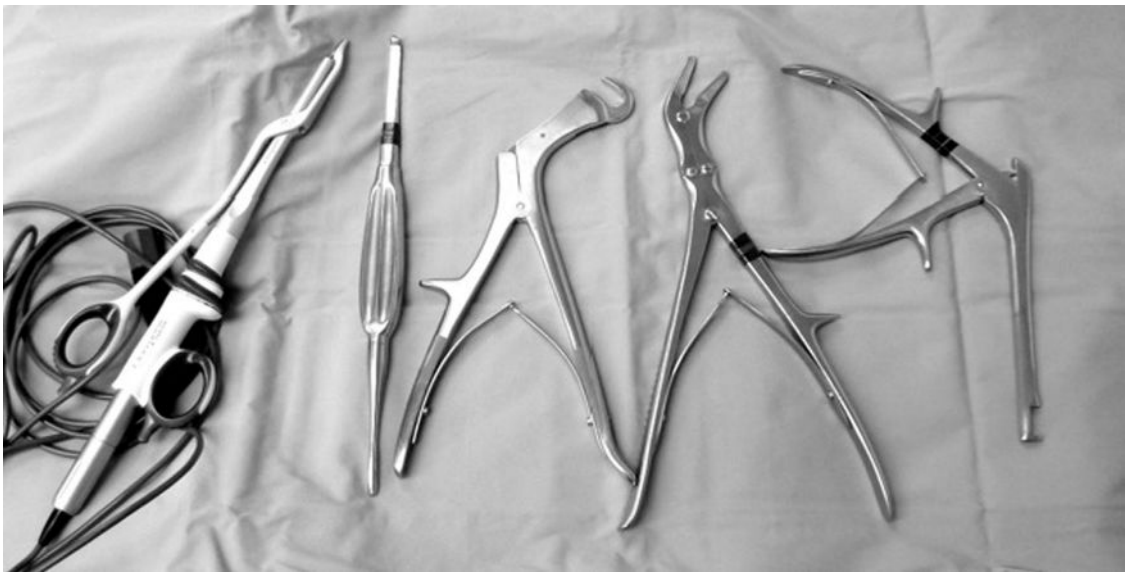

Fig. 11 – Instruments for supraclavicular rib resection: from left to right, Harmonic scalpel (Ethicon Endovascular, division of Johnson and Johnson, Piscataway, NJ), Overholt #1 periosteal elevator (V. Mueller, Oklahoma City, OK), Shoemaker Rib Cutter (Stille-Giertz,Bedford, VA), supraclavicular rib cutter (GP Pilling,Philadelphia,PA). and Raney rongeur 7" (Midwest Surgical, St. Louis, MO).

2 mm of touching the rib, the rib is removed. If there is 2 mm or more space between nerve and rib, the rib can be left intact. If there is a sharp, inner edge of the first rib, we are more likely to remove it. With experience, it has been observed that first ribs that are fairly straight and lie high in the neck usually require excision; first ribs with a wide "C" curve, the ones harder to excise, can be left (Fig. 12).

First-rib resection begins by excising enough middle scalene muscle to expose the neck of the rib. The intercostal muscles are detached with a Harmonic scalpel or periosteal elevator (Fig. 11, first and second from left). The medial rib edge is freed with the elevator. Rib edges are freed beneath the brachial plexus and subclavian artery with an elevator or by finger dissection. The posterior rib is then divided with a rongeur (Raney; Fig. 11, fifth from left) or rib cutter (Shoemaker; Fig. 11, third from left) (see Fig. 10I). The rib is freed from the pleura by finger dissection (Fig. 10J) and the anterior end is cut with an infraclavicular rib cutter (by Pilling) (Fig. 10K) (Fig. 11, fourth from left). The divided piece of rib is carefully extracted from behind the brachial plexus, avoiding traumatizing the nerves with the sharp edges of the rib (Fig. 10L). The anterior rib end, lying just below the subclavian artery, and the posterior stump are smoothed with a Raney rongeur (Fig. 11, fifth from left).

We assume the pleura has been opened even if not visualized. The anesthesiologist expands the lung and maintains the patient in positive end-expiratory pressure (PEEP) until the wound is completely closed. This will avoid a postoperative pneumothorax. A suction drain (#10 French channel) is placed deep in the wound and the scalene fat pad, platysma, and skin closed. A small catheter can be placed in the subplatysmal space for administration of local anesthesia for 24 to 48 hours postoperatively for pain control.

3.8.2.5. Technique of transaxillary first-rib resection. The patient is anesthetized and intubated, after which no further muscle relaxing drugs are administered. The patient is placed in a straight, lateral decubidus position with one Stahlberg padded hip positioner in front and one behind to fix the patient in a true lateral position (Fig. 13A). The arm and hand of the operated side

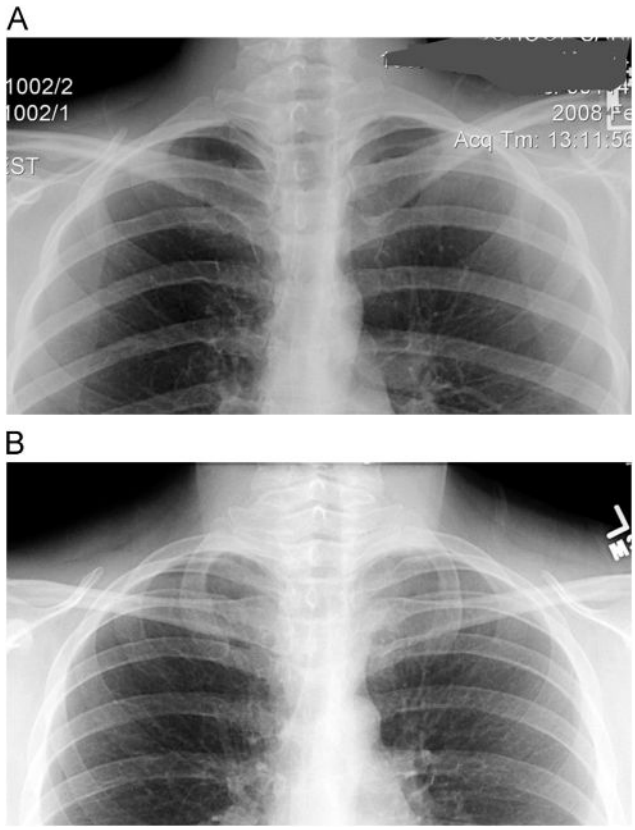

Fig. 12 – X-ray of first rib helps decide whether or not to remove rib. (A) Curved first rib can usually be left in place. (B) Straight first rib usually requires excision.

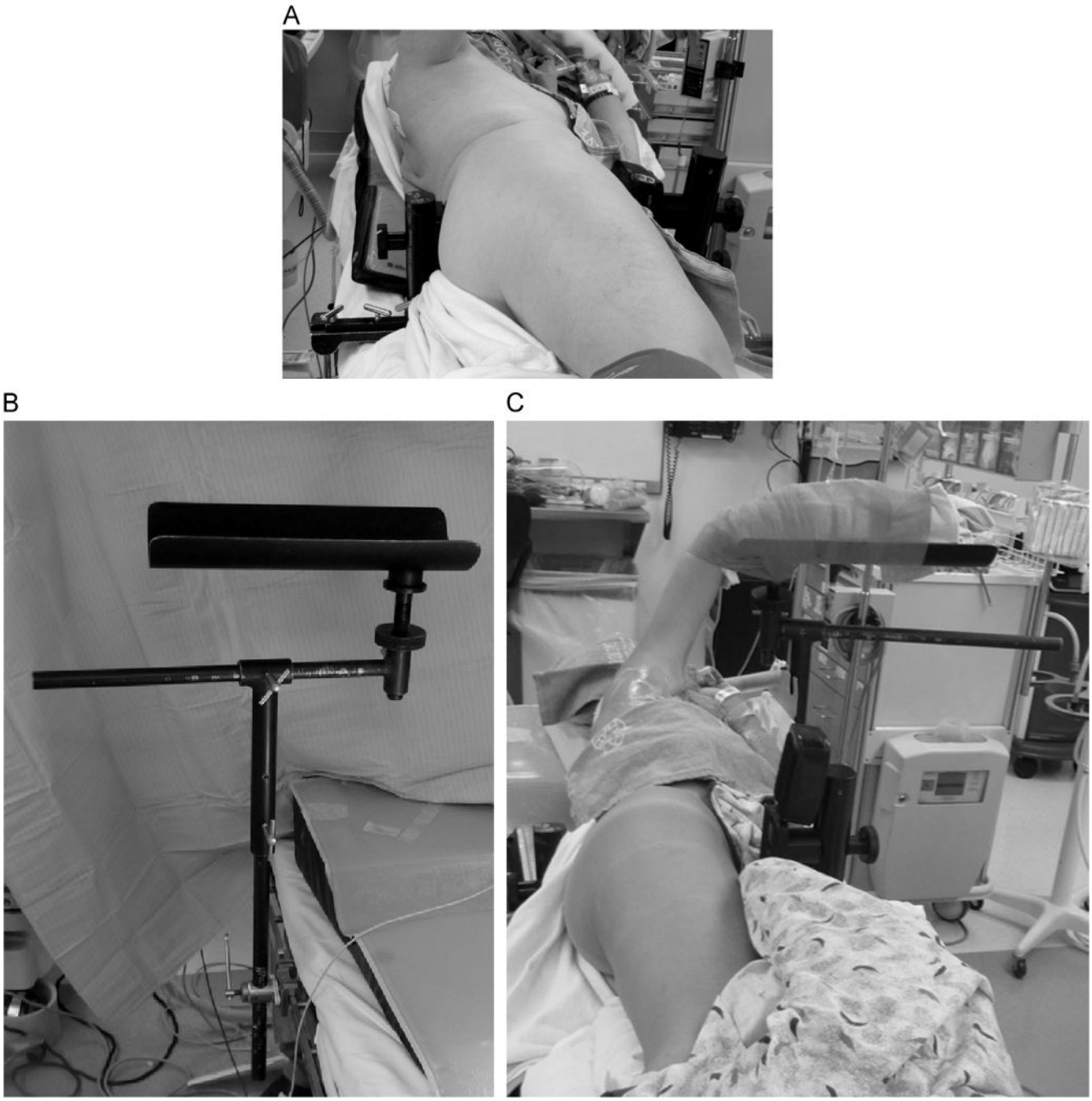

Fig. 13 – (A) For transaxillary first-rib resection, the patient is held in a straight lateral position by Stahlberg padded hip positioners (Innomed Instruments, Savannah, GA). The anterior positioner has two pads that lie against the abdomen. The posterior positioner has one pad and stabilizes the back. (B) Sterile adjustable arm holder, designed by Dr. James Sessions and Dr. David Roos, attaches to the table. (C) Patient in arm holder, viewed at end of operation.

is elevated, aided by Chinese finger traps. After prepping the skin and draping the table, a self-retaining arm holder (originally manufactured by Dr. James Sessions, Atlanta, GA) is installed to hold the arm (Fig. 13B and C). This setup is extremely helpful to provide the best exposure for this operation.

A 5- to 7-cm oblique incision is made 1–2 cm above the bottom of the hairline, beginning at the anterior axillary fold and descending posteriorly. Subcutaneous tissue is divided, trying to identify and preserve the second intercostal brachial cutaneous nerve. If the nerve is in the center of the field, the nerve is divided because an overstretched nerve causes much more discomfort than a numb patch of skin under the arm.

If a PMT is planned to be performed simultaneously, the PMM is identified, divided with a Harmonic scalpel, and 2 cm of PMM excised. The neurovascular bundle is unroofed by removing all clavipectoral fascia from the clavicle, medially, to the axilla, laterally. (see description of PMT-Section 4.5.1.1. Technique.)

The first rib is now sought, going posterior to the lymphatics exposing the serratus anterior muscle on the chest wall and identifying the first rib at the subclavian vein. The

intercostal muscles are divided on the inferior border of the rib, staying inside the posterior scalene muscle to the neck of the rib. A long renal vein retractor is placed inside the posterior scalene muscle to protect the long thoracic nerve from injury. The medial and lateral rib edges are completely freed with a Harmonic scalpel or periosteal elevator (Overholt #1). The middle scalene muscle is excised with the Harmonic scalpel from the medial and superior surfaces of the rib. During middle scalene muscle dissection, the arm is lowered and moved forward and the lower nerve trunk is protected with a Roos paddle as the middle scalene is divided. The anterior scalene muscle is exposed and freed from attachments to the subclavian vessels and divided. At the anterior end of the rib, the costoclavicular ligament is divided and the connection between rib and vein separated.

The middle section of the rib is divided with a large rib cutter (Bethune) with clear visualization of the subclavian vein anteriorly and lower trunk and subclavian artery posteriorly. The divided section of rib is removed. The anterior rib stump is shortened to the costal cartilage with rongeurs and a box cutter obtaining a smooth surface. The posterior stump is harder to visualize. Rongeurs and a box cutter shorten the posterior stump, while again protecting the lower trunk and T1 with a special Roos nerve paddle. The posterior stump is shortened to behind the lower nerve trunk, assuring that there is no sharp edge. The goal is to leave a stump no longer than 1 cm. This is the most difficult portion of the operation. Neurolysis of the lower trunk and T1 complete the decompression.

We assume the pleura may have been opened. The anesthesiologist expands the lung and maintains the patient in positive end-expiratory pressure (PEEP) until the skin is completely closed. This will avoid a postoperative pneumothorax. A suction drain (#19 channel drain) is placed deep in the wound and a small catheter is placed in the subcutaneous space for administration of local anesthetics for 24 to 48 hours postoperatively for pain control.

3.9. *Results of treatment for NTOS*

Nonoperative treatment results depend on how soon treatment begins after the onset of symptoms. Patients who begin treatment within the first month of the onset of symptoms have very good results, regardless of the type of the treatment. This is because the majority of NTOS patients develop symptoms after some type of trauma. Particularly if the trauma was acute, most patients will see improvement within the first few months even without treatment. Therefore, if treatment is started in the first few weeks after an accident, it is impossible to know if that patient is improving because of the treatment or because of the passage of time and nature doing its healing.

On the other hand, if treatment does not begin until months or years after symptoms began, the success rate decreases significantly. If symptoms continue after several months of therapy, the treatment options are to continue to live with the symptoms and use home exercises and medication; seek a different diagnosis; or consider surgery. When symptoms are mild and tolerable, most patients will elect continued nonoperative management.

If symptoms are severe, but show some improvement after 3 months of therapy, therapy is continued until symptoms either improve significantly or plateau. At that point, many patients will consider surgery. If symptoms are severe and show no improvement after 3 months of appropriate therapy, continuing therapy rarely leads to improvement.

Results of surgery for NTOS vary among the many reports in the surgical literature. One reason for this is the criteria for evaluating success vary greatly across the world. There are now three or four questionnaires that different medical centers use to describe symptoms before and after surgery. These can be helpful, but it must be realized that data from patients describing their symptoms are subjective, not objective. In general, we have found that asking patients to simply state how much improvement they notice compared to before surgery is just as reliable as burdensome questionnaires. We essentially want to know first, if the operation failed to provide any improvement, and second, if there is improvement, is it fair, good, or excellent?

The other factor in evaluating results is time. It has been well established that many patients are significantly improved in the first 3 months after surgery. However, over the next 24 months, recurrent symptoms will appear in about 15% of those patients. At least half of the recurrences appear within 6 months of surgery and 80% are present by 2 years [22]. This must be considered when looking at success rates.

The failure rate for thoracic outlet decompression is about the same after supraclavicular or transaxillary approach [22]. Although this study was reported in 1989, no other similar study has been reported showing anything different where both approaches were performed by the same surgeons. In that study, the overall success rate was about 75% at 2 years. During the next 13 years, the success rate fell $<5\%$. Since that study was published, surgical techniques have changed only little. Statistics are a little better, but are hard to compare because long-term results over 10 to 15 years are seldom published.

3.10. *Recurrent NTOS*

Recurrence or persistence of symptoms occurs in at least 10% to 20% of the patients operated on for NTOS. The symptoms and physical findings are similar to the original symptoms and findings. Diagnostic tests should first be directed to looking for other diagnoses. If treatable conditions are found in the cervical spine or shoulder, these should be evaluated and treatment considered. Evaluation for NPMS should also be performed and, if present, it should be treated first. If PMM stretching is unsuccessful, PMT carries a much lower risk than reoperation for NTOS [24,25].

If pectoralis minor release does not provide adequate relief, reoperation for NTOS can be performed. The choice of procedure depends upon what was done at previous operations.

1. If the first procedure was transaxillary first-rib resection, reoperation is supraclavicular scalenectomy and brachial plexus neurolysis. If there is a long posterior rib stump, it should be shortened.
2. If the first procedure was supraclavicular scalenectomy without first-rib resection, reoperation is transaxillary

first-rib resection plus consideration of supraclavicular neurolysis.
3. If the first operation was supraclavicular scalenectomy and first-rib resection, reoperation is supraclavicular neurolysis, as this allows exposure of all the nerve roots and trunk.
4. If all of the above procedures have been done and have failed, a last resort procedure is neurolysis and wrapping of the brachial plexus with a flap of latissimus dorsi muscle. This is an extensive procedure requiring several months for total recovery [26].

4. NPMS

The majority of patients with NPMS also have NTOS. Most of the time the two are treated simultaneously. However, it is important to recognize each condition separately, particularly if NPMS is the predominate diagnosis. This is an example of a double crush phenomenon in which treating just one of the diagnoses may reduce symptoms enough so that treating the second diagnosis is unnecessary.

4.1. *Symptoms*

In some patients NPMS is the only diagnosis. Paying close attention to the symptoms and physical findings can make the diagnosis. Symptoms of pain or tenderness in the anterior chest wall and axilla are usually the chief complaint. Pain over the trapezius muscle and paresthesia in the hand are additional frequent symptoms. Neck pain and occipital headaches are either absent or minimal.

When symptoms are on the left side, pain in the left chest wall and axilla plus tingling in the left hand mimic angina pectoris. It is not unusual to see young, physically active patients with left chest pain who have been seen in an emergency center and received a completely normal workup for cardiac ischemia. Some patients have even had cardiac catheterization and coronary angiograms with normal findings.

4.2. *Physical examination*

The significant physical findings are tenderness below the clavicle (over the PMM) and in the axilla. The upper limb tension test is usually mildly positive. Abducting the arm to 90 degrees in external rotation and the elevated arm stress test may be mildly positive as well [27]. Shoulder abduction with shoulder retraction usually elicits pain. There is minimal tenderness in the supraclavicular fossa over the scalene muscles.

4.3. *Etiology*

Isolated NPMS, with little or no NTOS, is usually seen in teenagers or young adults who participate in competitive sports. The sports are those that require repetitive movements of the upper extremities above shoulder level, such as swimming, throwing baseballs or footballs, volleyball, and weight lifting. These sports have in common scapular retraction with each arm motion. This stretches the PMM, which inserts into the coracoid process of the scapula. Initial symptoms may be chest pain or hand paresthesia.

Many of these patients also have mild symptoms of NTOS, brought about by hyperextension of their neck while they are performing their sport. Therefore, it is not unusual to find these patients being first diagnosed as having NTOS. It is only by detailed history and physical examination that the diagnosis of NPMS is made, along with the diagnosis of NTOS. However, the response to muscle blocks will demonstrate the relative contribution of NPMS to symptoms.

The importance of recognizing NPMS in children was revealed in a study of the results of surgery for NTOS and NPMS in children. We became aware of NPMS in 2005. A study of the results of surgery from 2000 to 2011, was separated into two time periods, before and after learning about NPMS. During the first 5 years, before learning about NPMS, all of the operations were thoracic outlet decompression procedures. In the next 5 years, after becoming aware of NPMS, 80% of the operations were PMT alone, and only 20% were thoracic outlet decompressions. Based on outcomes, the conclusion from this analysis was that in the first 5 years, a significant number of patients receiving thoracic outlet procedures would likely have received equal benefit from PMT [28].

4.4. *Diagnostic tests.*

4.4.1. *Diagnostic muscle block*

The pectoralis minor block was described earlier in the see Section 3.6.1. PMM block. A good response to the block is strong evidence to support the diagnosis of NPMS. A good response to the block is defined as significant relief of most symptoms at rest and greatly reduced positive findings when the physical examination is repeated after the block. When this occurs, no further block is needed. If some symptoms and positive physical findings remain, a scalene muscle block is performed. If this second block relieves the rest of the symptoms and findings, a diagnosis of both NPMS and NTOS is made. If the patient's relief of symptoms was much greater with the pectoralis minor block, pectoralis minor decompression alone is strongly considered. In such patients, the residual symptoms of NTOS can often be tolerated. If symptoms are significant, thoracic outlet decompression can be performed at a later date.

4.4.2. *Electrodiagnostic study*

As stated earlier, measurement of the medial antebrachial cutaneous sensory nerve action potential is an objective test that is frequently positive [14]. A positive response is indicative of brachial plexus compression; it cannot separate compression at the scalene triangle from compression at the subpectoral area. But putting the results together with the clinical picture and response to muscle blocks will help guide therapy.

4.5. *Treatment*

Physical therapy is always tried first. For NPMS, the therapy is PMM stretching (Fig. 8). If performed when symptoms are only a few months old, the success rate is quite good. However, if there is no improvement with 3 months of therapy, surgery is considered.

4.5.1. *Surgery*

Pectoralis minor tenotomy, with partial myomectomy, is the treatment for NPMS. The operation can be performed through an incision on the chest wall preferably just below the clavicle or through a transaxillary approach. Our preference is a transaxillary incision in the anterior portion of the axilla just above the hairline [29]. Through this approach, it is easier to explore the axilla to divide thickened bands of clavipectoral fascia and the occasional Langer's arch, which arises from the latissimus dorsi muscle [30]. These bands of tissue can compress the axillary neurovascular bundle in the subpectoral space and must be released for an effective outcome. They are much harder to identify and divide through an infraclavicular approach.

Pectoralis minor tenotomy is a minimal risk, outpatient surgical procedure. Because this procedure has minimal risks, it can be performed under general anesthesia or heavy sedation and local anesthesia, and because recovery time is only a few days, PMT can be offered to patients with less concern than for thoracic outlet decompression.

4.5.1.1. Technique. A 4- to 7-cm transverse incision is made 1 cm above the bottom of the axillary hairline beginning at the anterior axillary fold. Beginning anteriorly helps avoid the second intercostal brachial cutaneous nerve, which usually lies more posteriorly. Subcutaneous tissue is divided, the PMM identified by its attachment to the coracoid process, and the muscle divided at the coracoid with the Harmonic scalpel or bipolar scalpel and scissors. The end of the muscle is grasped with a long clamp and 2 cm excised with the Harmonic scalpel. In a few patients, the pectoralis minor is adherent to the pectoralis major. Pectoralis minor is then identified as the muscle inserting at the coracoid. It is important to avoid the pectoral nerve branches, which usually lie about 3 cm from the coracoid and penetrate pectoralis minor en route to pectoralis major. Any bands of clavipectoral fascia and the occasional Langer's arch muscle fibers [30] are also excised, leaving nothing tight that can compress the neurovascular bundle that lies under the PMM. Subcutaneous tissue and skin are closed with two layers of buried sutures without a drain.

4.6. *Results of treatment*

Success rate for PMT alone in 52 patients was good in 84%, fair in 8%, and failed in 8% with 1- to 3-year follow up. For patients diagnosed with both NPMS and NTOS, the results of PMT alone were good in 35%, fair in 19%, failed in 46% [29]. Most of the 65% who were fair or failed subsequently underwent thoracic outlet decompression at a later date. In 20 children, the success rate of PMT alone was good to excellent in 75%, fair in 10%, and failed in 15% [28].

5. Venous compression: VTOS and VPMS (Appendix II)

5.1. *Classification*

The definition of VTOS is axillosubclavian venous obstruction with or without thrombosis. In either case, the symptoms are the same: arm swelling and tightness. The thrombotic form has greater swelling and discomfort than the nonthrombotic form. Often the symptoms of axillosubclavian vein thrombosis are preceded by milder symptoms of intermittent nonthrombotic obstruction, so the two conditions should be regarded as two stages of the same condition. VTOS or VPMS can be primary or secondary.

Secondary venous obstruction indicates that there is a known cause for the condition. The more common cause is catheters or wires inserted into the venous system. The catheters are placed for patients requiring short or long-term intravenous chemotherapy for infections, cancer, for feeding purposes, or for patients with renal failure who are on dialysis. The wires connect subcutaneous pacemakers and defibrillators with the heart, in patients with cardiac arrhythmias.

Primary venous obstruction is defined as venous obstruction or thrombosis with no obvious known cause. It is also called Paget-Schrotter syndrome [31,32], effort thrombosis, idiopathic, spontaneous, or traumatic thrombosis. This type is seen more often in teenagers, young people, muscular athletes, people who lift weights and in hikers with backpacks.

5.2. *Symptoms*

Symptoms of VTOS or VPMS include swelling and cyanosis of the involved upper extremity. The onset may be sudden, occurring within 24 hours, or may be subtle, occurring over several days or weeks. A feeling of tightness and fullness of the extremity is quite common, while pain may or may not be present. Paresthesia is not usually a prominent feature, but may occur when swelling is significant.

5.2.1. *Thrombotic versus nonthrombotic obstruction*

The symptoms of nonthrombotic venous obstruction are the same as those of thrombotic obstruction. However, in nonthrombotic obstruction, the symptoms are intermittent, usually coming with elevation or activity of the arm and receding or even disappearing when the arm is at rest. When thrombosis occurs, the symptoms are usually constant, more intense, and persist for days or weeks. They do not disappear quickly unless treated. Failure to treat may result in an arm with chronic pain, swelling, and dysfunction.

5.3. *Physical examination*

Swelling of the hand and entire arm is the main finding in almost all patients. In some patients, the swelling may be minimal and can only be detected by measuring the circumference of both arms to find an increase in size of as little as 1 cm above and below the elbow. The swelling is usually not pitting.

Color changes in the involved arm are not always present, or may be subtle. The arm is usually darker in color, often with a blue or purplish tinge. The color change is associated with distended superficial veins on the involved arm. Veins are often visible over the shoulder and chest wall of the involved side. Comparing the veins on the two sides of the chest wall should reveal the distended veins on the involved side.

5.4. *Etiology*

The body's clotting system involves a delicate balance between clot formation and clot lysis. Secondary VTOS or VPMS obstruction has several causes that can upset that balance. The most common is the presence of foreign bodies, such as catheters or wires, inside the vein. It can also be caused by coagulopathies, such as Factor 5 genetic abnormality, Protein S or Protein C deficiency, and other types of coagulation defects.

Primary VTOS and VPMS were initially regarded as venous obstruction of unknown cause. However, since Paget in 1875 [31] and von Schrotter in 1884 [32] initially described this condition, much has been learned about the causes of venous obstruction. The anatomy of the subclavian vein as it joins the innominate vein is instructive. The subclavian vein at this point is surrounded by four structures: The first rib inferiorly, the subclavius tendon superiorly, the costoclavicular ligament medially, and the anterior scalene muscle laterally (Fig. 14). In early development, if the subclavian vein lies a little too medially, the costoclavicular ligament can indent the vein; if the subclavian vein lies a little too high, the subclavius tendon can indent it. These anatomical variations can act as predisposing causes to develop obstruction if the arm performs repetitive activity above shoulder level. Overhead activities include such maneuvers as athletes throwing or swimming, people stocking shelves or painting above their heads, backpacking, and weight lifting.

The pathophysiology in these cases begins with the vein being irritated by pressure from the ligament, tendon, or bone lying against the vein. The intima of the vein develops inflammatory changes that lead to thickening and fibrosis inside the lumen of the vein. As the process continues, fibrosis builds up inside the vein, eventually producing internal scarring, synechia, and finally, significant stenosis.

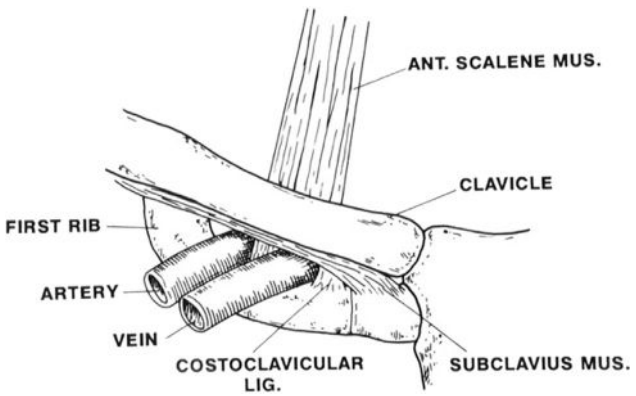

Fig. 14 – The costoclavicular space. The subclavian vein is bounded by the costoclavicular ligament (COSTOCLAVICULAR LIG.) medially, the anterior scalene muscle (ANT. SCALENE MUS.) laterally, the subclavius muscle tendon superiorly, and the first rib inferiorly. Reprinted from Sanders RJ, Haug CE. Thoracic outlet Syndrome: A Common Sequela of Neck Injuries. Philadelphia: Lippincott: 1991:236, with permission.

In turn, this results in turbulent blood flow, and eventually reduced blood flow to a point where swelling develops in the arm. With inflammation and reduced flow, platelets and clotting factors combine to form thrombus.

The same process can occur in the axillary vein from pressure by the PMM, resulting in the less common axillary vein obstruction and VPMS.

5.5. *Diagnostic tests*

Ultrasound duplex scan is a noninvasive technique that is readily available. When it demonstrates total occlusion of the axillosubclavian vein, the diagnosis is definitive. However, demonstrating flow in the area around the clavicle can be difficult and misleading. When the diagnosis is uncertain, dynamic venography is diagnostic.

Venography, injecting dye into the brachial or basilic vein of the symptomatic arm is the more reliable diagnostic test for axillosubclavian vein obstruction. The basilic or brachial vein access is the preferred route, especially if treatment is to be performed during venography. If injection is through the cephalic vein, a major segment of the basilic and axillary vein is bypassed and only the subclavian-innominate vein section seen. In the majority of patients, the main pathology will be in this area and will be detected with cephalic vein injection. However, cephalic vein injection can appear normal while missing an abnormality in the brachial and axillary veins. Ultrasound guidance is used to cannulate the basilic or brachial system.

Venography begins with the arm near the patient's side. If it shows total occlusion, additional injections with the arm elevated are unnecessary. If the resting position reveals normal venous flow, dynamic positioning should follow, repeating the injection with the arm at 90 degrees and again near 180 degrees. Particularly in patients with nonthrombotic obstruction, the venogram may be completely normal at rest, but demonstrate stenosis or occlusion with significant collateral venous filling when the arm is elevated.

Patients who are allergic to injectable iodine can be treated with a steroid preparation before the injection. This has helped many patients avoid reactions who have had them in the past. (Currently, we use prednisone 50 mg 13 hours, 7 hours, and 1 hour plus Benadryl, 50 mg, one hour before procedure by mouth.)

MRA is an alternate choice. Although MRA can reveal a patent or occluded venous system, it does not give the details sometimes needed to plan treatment, nor does it allow treatment at the time of study.

5.6. *Treatment*

Basically, there are two choices of therapy: anticoagulation or a three-step program of thrombolysis, surgery, and possibly balloon angioplasty or surgical reconstruction. The choice may be guided by the facilities available to the patient. Rural areas, or areas without medical facilities, may be limited to anticoagulation. Where interventional medical care is available, the three-step program will lead to faster recovery and less chance of persistent venous obstruction with disability.

6. VTOS

There are three steps in treating VTOS.

Step 1. Dissolve the clot by fibrinolysis. If this is non-thrombotic, go to step two.

Step 2. Treat the extrinsic problem by removing the structures outside the vein wall that are compressing the vein. For VTOS, these are the first rib, costoclavicular ligament, subclavius tendon, and anterior scalene muscle. For VPMS, this is the PMM.

Step 3. Treat the intrinsic problem, fibrosis or residual thrombus inside the vein, in order to lessen the chance of persistent symptoms after the extrinsic problem has been resolved.

6.1. *Step 1. Dissolve the clot*

Acute thrombosis of the axillosubclavian vein requires early, not emergent, treatment. When a patient presents with a history of recent onset of arm swelling, cyanosis, and tightness or pain in the arm, the diagnosis of axillosubclavian vein thrombosis should be immediately considered. The diagnosis can be confirmed with ultrasound duplex scanning. If catheterization and thrombolytic therapy can be started within the next few days, arrangements should be made to do so. Heparin can be started when the patient is initially seen, either intravenously or in the form of low-molecular-weight heparin (1 mg per kg body weight every 12 hours).

If facilities for catheter thrombolysis are not readily available, the initial therapy is anticoagulation. Subcutaneous heparin and warfarin are started immediately. This therapy is not as successful as thrombolytic therapy, but it will suffice for a few days until thrombolysis can be started. In rare patients, clots have disappeared with just anticoagulation, but the success rate is far inferior to that of early thrombolysis [33].

Fibrinolysis requires hospitalization with intensive care monitoring for the duration of therapy. A catheter is inserted into the basilic or brachial vein using ultrasound to cannulate the vein. The catheter is passed up to the axillary vein and a venogram performed. If a clot is demonstrated, a wire is used to traverse the thrombus, the catheter is then positioned in the clot and thrombolysis begun. Alternatives at this point include infusion of a thrombolytic agent using either a multiple, side-holed catheter or an EndoWave catheter system (EKOS Corp, Bothell, WA). Intravenous heparin is concurrently infused threw the side port of the sheath to prevent propagation of thrombus around the catheter in the involved arm. A venogram is performed at regular intervals to follow the progress of thrombus dissolution. Successful clot lysis may require several hours.

Reported success rates vary from 62% to 84% [34,35]. Even if the thrombus persists, thrombolytic therapy is stopped after 48 hours. The use of pharmacomechanical thrombectomy may improve patency and reduce treatment time and costs [36,37]. Treatment of thrombus older than 6 weeks has less success. One report noted only 50% could be partially opened, none were completely recanalized [34].

If clot dissolution is unsuccessful, further treatment depends on residual symptoms. Options include anticoagulation therapy for 3 to 6 months, first-rib resection with venolysis followed by anticoagulation for 3 to 6 months and then repeat venogram, or first-rib resection with venous reconstruction. A prerequisite for venous reconstruction is adequate inflow through the axillary vein. It should be noted that many patients placed on long-term anticoagulation may obtain good relief of symptoms without surgery (personal communication, Dr. Kai Johanson). This is the result of either clot lysis over several months on long-term anticoagulation and/or the development of sufficient venous collaterals to allow venous drainage during use of the arm (Fig. 15).

If thrombolysis is successful, the post-thrombolytic venogram is studied. Before leaving the catheterization suite, while the catheter is still in place, some physicians will attempt to balloon dilate residual stenosis (angioplasty), as the acute clot has now been resolved. Angioplasty at this time is occasionally successful. However, most of the time, balloon angioplasty fails when performed before the extrinsic pressure on the vein has been relieved because the balloon cannot stretch out the compressive structures surrounding the vein. In fact, balloon dilatation at this time can traumatize the endothelium, resulting in rethrombosis. However, our practice is to dilate the vein with a 6-mm balloon in an effort to remold the residual thrombus and maintain venous patency until surgical decompression is performed in the next few days.

Stenting the subclavian vein is fraught with a high incidence of fracture and rethrombosis. Even if stents are employed after first-rib resection, stents often fail because they are still compressed by the clavicle when the arm is elevated.

6.2. *Step 2. Treating the extrinsic factor*

While there is no urgency to perform surgery immediately after thrombolysis, the presence of residual thrombus and stenosis causes rethrombosis within 4 weeks in as many as 30% of patients who initially were reopened. Until the external pressure on the vein is relieved, reclotting can occur,

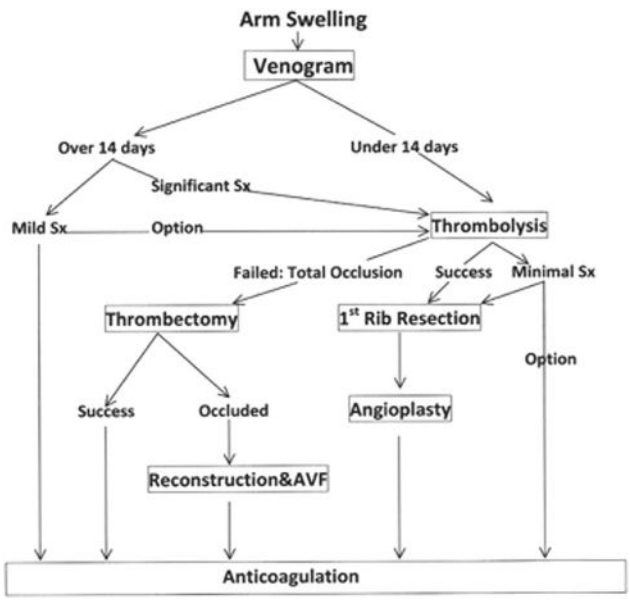

Fig. 15 – Algorithm of management of venous thoracic outlet syndrome. Sx, symptoms.

as nothing yet has been done to remove the underlying cause of thrombosis [38]. The highest success rate is achieved by performing first-rib resection and venolysis in the next few days. Until surgery is performed the patient should remain anticoagulated. At the time of surgery, we adjust the level of anticoagulation seeking an international normalized ratio of 1.8 to 2.4. Postoperatively, anticoagulation is continued for at least 3 months.

Surgical approaches must provide a means to remove not only the anterior part of the first rib, but to divide all ligaments, tendons, and muscles surrounding the subclavian vein (venolysis) (Fig. 14). The most popular approach, and the one we prefer, is transaxillary first-rib resection. However, this approach does not allow for venous reconstruction. First-rib resection can also be done through an infraclavicular incision, although exposure is more difficult going under the clavicle.

6.2.1. *Technique of transaxillary first-rib resection*

The operation for VTOS through the axilla is the same as described for NTOS (see Section 3.8.2.3. The transaxillary approach). However, after the center of the rib has been removed and the rib removed anteriorly to the costal cartilage, the subclavian vein is totally decompressed by excising connective tissue, the costoclavicular ligament and subclavius muscle tendon, and any remaining anterior scalene muscle fibers.

6.3. *Step 3. Treating the intrinsic factor*

Once the first rib has been excised and the subclavian vein freed from extrinsic pressure, stenosis may still exist inside the subclavian vein lumen. This is determined by routine postoperative subclavian venography. After decompressive surgery, some patients will have persistent symptoms, while others are symptoms free. In the symptom-free patients, a follow-up venogram is obtained to make sure there is no significant stenosis. If there is stenosis of $<50\%$, nothing further need be done, except placing the patient on therapeutic doses of warfarin for 3 months. After this, anticoagulation may be halted.

Patients with residual symptoms postoperatively, and those who are asymptomatic but have subclavian vein stenosis $>50\%$ postoperatively, are treated by balloon angioplasty of the subclavian vein. Now, the vein is much more responsive to dilatation as the extrinsic compressing structures have been removed. Stenting should be avoided, as reclotting has been reported as high as 40% [39].

6.4. *Anticoagulation*

Anticoagulation is the alternative to thrombolysis and first-rib resection. It is also a supplement to the 3-step approach. After each step, there is intimal injury of the subclavian vein. While this will eventually endothelialize, until it does, the vein is at risk to reclot. Therefore, the patient is anticoagulated between steps and after any reconstruction or bypass procedure.

If the patient is not already anticoagulated, heparin, or one of its derivatives is begun and warfarin is then started. Once therapeutic international normalized ratios are reached, bridge therapy with heparin is stopped. Currently, oral therapy with Factor 10A inhibitors is an alternative.

The length of time anticoagulation continues is variable. The standard recommendation is for 3 to 6 months. Continuing anticoagulation beyond his time is determined on a risk-to-benefit basis [40].

6.5. *Results of early treatment*

The success rate for treatment of VTOS depends on the time interval between the onset of symptoms and beginning of treatment. Treatment that begins within a few days of the onset of arm swelling, has at least a 90% chance of clot dissolution and resolution of symptoms [33]. Even treatment begun within the first 7 to 14 days of the onset of arm swelling, has a high success rate.

If the time from the onset of symptoms to initial treatment is >14 days, the success rate is reduced but may still occur. Fibrinolysis may still succeed if the clot is <30 days old, but the greater the delay, the poorer are the results. Patients seen after 30 days, or those who failed to recannulate with thrombolysis, are anticoagulated for at least 6 months at therapeutic levels. Studies have shown that the longer the patient is anticoagulated, the higher the success rate. But, there is not much further improvement after 3 to 6 months Rarely has success been achieved in passing a guide wire through a thrombus in a vein that was totally occluded.

6.6. *Venous reconstruction*

6.6.1. *Chronic venous obstruction: postphlebitic and nonthrombotic*

Persistent symptoms of arm swelling, tightness, and even paresthesia occur in some patients who have had acute thrombosis treated with anticoagulants alone. Included in this patient group are those who have undergone first-rib resection and venolysis, but have rethrombosed their axillo-subclavian vein. In some patients, these symptoms exist with venous stenosis with no history of previous thrombosis. These patients all fit into the category of chronic venous obstruction.

Treatment depends on the intensity of symptoms and the degree to which symptoms interfere with patients' lives. A variety of therapies are available to restore venous flow from the arm, but these options all involve surgery with variable success. In general, people whose lives are sedentary may feel they can tolerate these symptoms if they know the condition will not progress. On the other hand, people who are physically active and wish to participate in strenuous work or recreational activities, may desire interventions to improve arm function. Available approaches are endovenectomy, bypass grafting, or interposition grafting.

A preoperative digital subtraction venogram is performed to define the anatomy. As with any vascular reconstruction, adequate inflow and outflow is a prerequisite. If first-rib resection and venolysis have not yet been performed, they should be done either before or along with the endovenectomy.

6.6.2. Subclavian vein thrombectomy/endovenectomy with vein patch closure

The indications to perform an operation for chronic venous obstruction are a patent axillary and internal jugular vein, and significant symptoms interfering with activities of work, sleep, or recreation.

One form of venous repair is to open the vein, remove fresh clot, excise the scarred, thickened intima, and close the vein with a venous patch angioplasty. This may be done through an infraclavicular incision, but will occasionally require a median sternotomy extension [41]. Although there are reports of gaining exposure via claviculectomy, this is rarely done because it can be partially disabling for active people.

6.6.2.1. Technique. The procedure is performed by exposing the subclavian vein through an 8- to 10-cm skin incision about 2 cm below, and parallel to, the clavicle and beginning 3 to 4 cm from the midline. The venous pathology usually begins near the costoclavicular ligament close to the subclavian-innominate junction. The pectoralis major muscle is split in the direction of its fibers to find the subclavian vein, which may be tucked under the clavicle. The vein is mobilized from the costal cartilage to as far laterally as needed until the soft, uninvolved segment of axillosubclavian vein is identified. The patient is heparinized and vessel loops or vascular clamps used for proximal and distal control. If fresh clot exists, it is evacuated. If fresh clot exists in the distal vein, it can be remove with a balloon catheter and/or thrombectomy forceps. Venous intima cannot be treated by the same techniques employed for arterial endarterectomy. No plane of dissection can be developed between venous intima and adventitia. The scarred intima must be excised sharply, with a scissors or scalpel, avoiding transmural injury. Some intimal scar is necessarily left on the vein wall. A patch angioplasty is performed, preferably with saphenous vein, or other suitable material, such as homologous cryopreserved vein (Fig. 16).

In patients whose disease process extends so far proximally that control cannot be obtained through the infraclavicular incision, exposure of the more proximal vein can be obtained by splitting the sternum from manubrium to the first interspace, then opening the interspace laterally, far enough to lift the clavicle and costosternal junction. This provides excellent exposure of the innominate-subclavian vein junction and the internal jugular vein. The approach described here for chronic venous occlusion is applicable to acute venous thrombosis, particularly when fibrinolysis has failed [41]. Reiterating, an essential criteria to performing subclavian endovenectomy includes a widely patent inflow tract from the axillary vein and distally.

6.6.3. Claviculectomy

Although removing the clavicle provides exposure of the subclavian vein, it is rarely performed because of its cosmetic defect and because it partially destabilizes the shoulder girdle. Twice we have performed claviculectomy, both times in patients weighing >300 lb. Both patients had successful outcomes, but some instability of the shoulder girdle. A long-term follow-up of patients undergoing claviculectomy found objective deficits in mobility and strength, although most of these patients had normal self-perceptions of overall health and upper-extremity function [42].

6.6.3.1. Technique. A 12- to 15-cm incision is made directly over the clavicle beginning at the sternoclavicular joint. The clavicle is circumferentially dissected free and divided laterally with a Gigli saw or an electric saw. The point of division should be far enough laterally to remove at least two thirds of

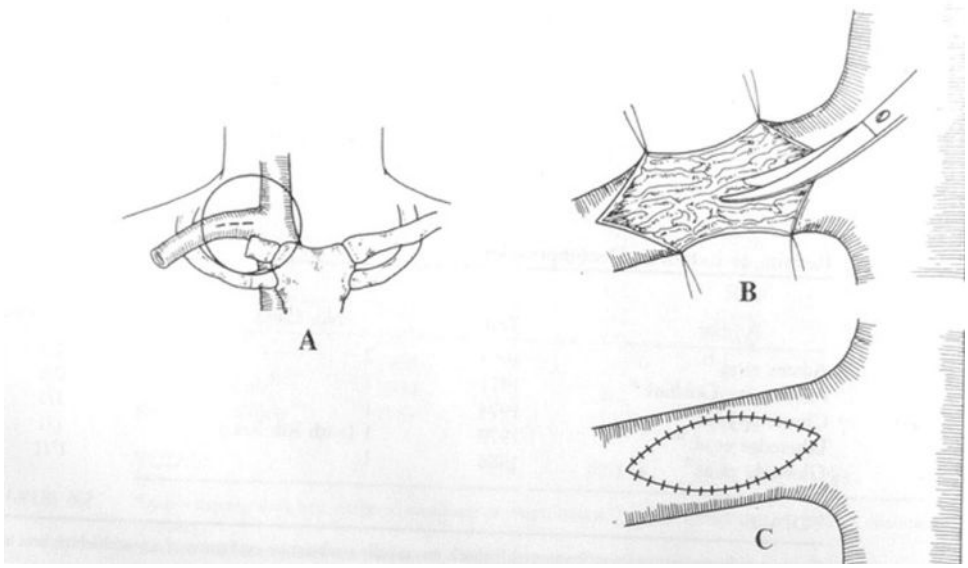

Fig. 16 – Subclavian vein endovenectomy. (A) Incision is 2 cm below the clavicle. Pectoralis major muscle is split between sternal and clavicular heads. The subclavian vein is mobilized, occluded proximally and distally, and an incision made on its anterior surface. (B) Thickened intimal is excised sharply leaving about 1 to 2 mm of scar to avoid puncturing the vein wall. (C) Patch graft closure with autogenous vein or prosthetic material. Reprinted from Sanders RJ, Haug CE. Thoracic outlet Syndrome: A Common Sequela of Neck Injuries. Philadelphia: Lippincott: 1991:252, with permission.

the clavicle, as removing just the medial half leaves too long a lateral half, which can obstruct the axillosubclavian vein. The medial end is disarticulated at the sternoclavicular joint. While attempts to replace the excised piece of clavicle at the end of the procedure can be successful, many times they have failed. We have not attempted it (Fig. 17).

6.6.4. *Axillojugular vein transposition*

The alternative to axillosubclavian vein endovenectomy is to bypass the occluded vein. The best conduit for this is the internal jugular vein because of its large diameter and convenient location. The indications to perform a bypass are chronic obstruction of the subclavian vein, a patent axillary vein into which to sew the internal jugular vein, and significant symptoms interfering with activities of work or recreation. If the axillary vein is also occluded, the bypass is not an option because the internal jugular vein will not stretch much farther laterally than the proximal end of the axillary vein. In such circumstances, the only alternative is an interposition graft.

6.6.4.1. Technique. An 8- to 12-cm incision begins 2 cm below and parallel to the clavicle and 4- to 6-cm from the midline. The pectoralis major muscle fibers are split between sternal and clavicular origins. The axillary vein is exposed by dividing the PMM. The axillosubclavian vein is mobilized and isolated between vessel loops. The internal jugular vein is mobilized circumferentially through two transverse neck incisions. The internal jugular vein is freed to the base of the skull, suture ligated, and divided. The anterior surface is marked with a stitch to prevent twisting when the internal jugular vein is brought down to the axillary vein.

Through the lower transverse neck incision the internal jugular vein is mobilized to the subclavian-jugular junction. The subclavius muscle is divided and a portion of it excised to create a retroclavicular tunnel through which to pass the divided end of the internal jugular vein to meet the axillary vein.

The internal jugular vein is now brought down in the neck and passed through the tunnel below and behind the clavicle to comfortably reach the axillary vein (Fig. 18). A final check on orientation of the internal jugular vein can be made by passing a small plastic catheter (intratracheal works fine) through the open end of the internal jugular vein to make sure it passes smoothly into the innominate vein before the internal jugular vein is sewn in place.

Once this is complete, the patient is heparinized and the subclavian vein ligated proximal to the proposed anastomotic site so blood does not collect and thrombose in the proximal blind stump (Fig. 19). The axillary vein is incised on its superior surface to receive the internal jugular vein in end-to-side fashion using fine polypropylene suture. Once completed, the anastomotic site is marked with a hemoclip for future x-ray purposes. Alternately, the axillary vein can be totally divided and end-to-end anastomosis performed.

6.6.5. *Arteriovenous fistula*

The benefit of an arteriovenous fistula (AVF) to enhance patency after venous repairs was nicely shown by an experiment in dogs and then one patient. Venous repairs were demonstrated to have a much higher patency than controls when an AVF was placed distal to the venous repair [43].

A temporary AVF is created distal to the anastomosis to increase the pressure and flow through the juguloaxillary anastomosis. Because venous surgery is in a low-pressure system, venous repairs or bypass grafts have a greater tendency to thrombose than do such repairs in arterial systems. By inserting a temporary AVF distal to the site of an endovenectomy or venous bypass, patency is improved by increasing venous flow across the new anastomosis. The fistula is closed after 2 to 3 months.

Two types of AVFs have been used for venous repairs in the axillosubclavian vein area. One is axillary artery to axillary vein, just distal to the anastomosis. A piece of ringed polytetrafluoroethylene is used. The graft is looped up to the subcutaneous tissue so when it is closed, it can easily be found and divided (Fig. 20, AVF). It is unnecessary to remove the pieces of the graft down to their origin. Leaving the divided ends of polytetrafluoroethylene have caused no problems after being followed for many years postoperatively.

The other type of AVF is a distal forearm construction, such as a Cimino fistula. These are easily closed when no longer needed.

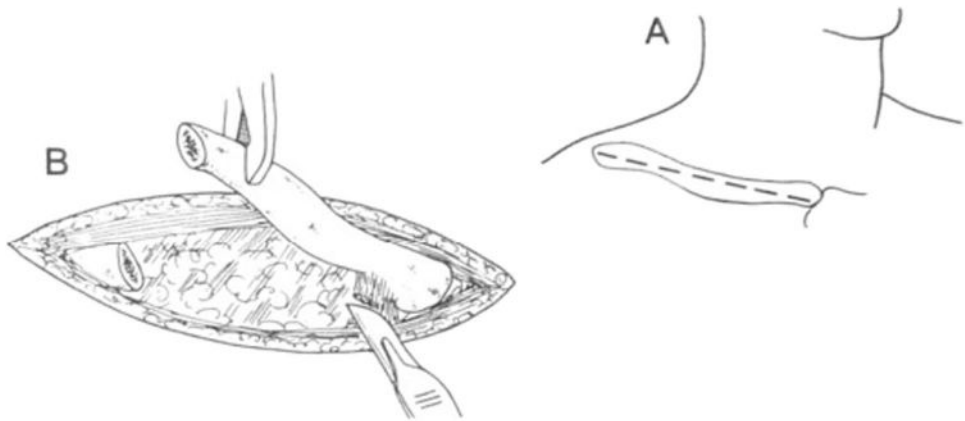

Fig. 17 – Claviculectomy. (A) The incision is made directly over the clavicle. (B) The clavicle is dissected circumferentially and divided at least two-thirds of the way towards the acromion process. The medial two-thirds is elevated and the medial end is disarticulated from the sternoclavicular joint.

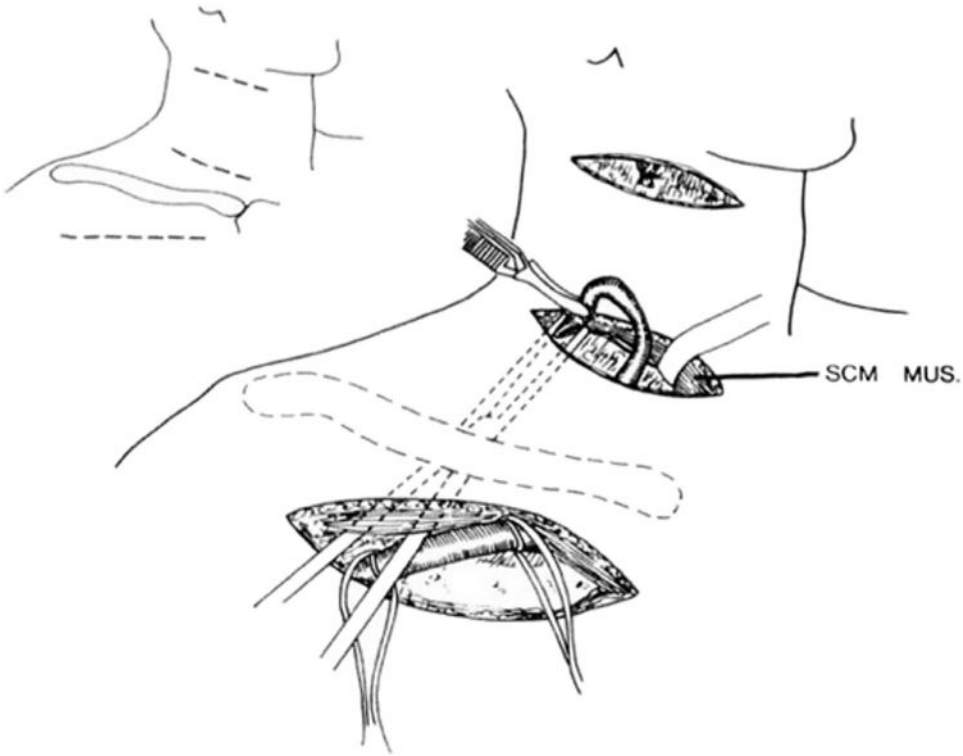

Fig. 18 – Axillojugular vein bypass. Left: Placement of the three incisions. Right: the internal jugular vein has been dissected free, suture-ligated at the base of the skull, and passed down to the lower neck incision. The vein is being passed through the tunnel below the clavicle into the infraclavicular incision. The axillosubclavian vein has been freed in preparation for the axillojugular anastomosis. Reprinted from Sanders RJ, Haug CE. Subclavian vein obstruction and thoracic outlet syndrome. Ann Vasc Surg 1990;4:397–410, with permission.

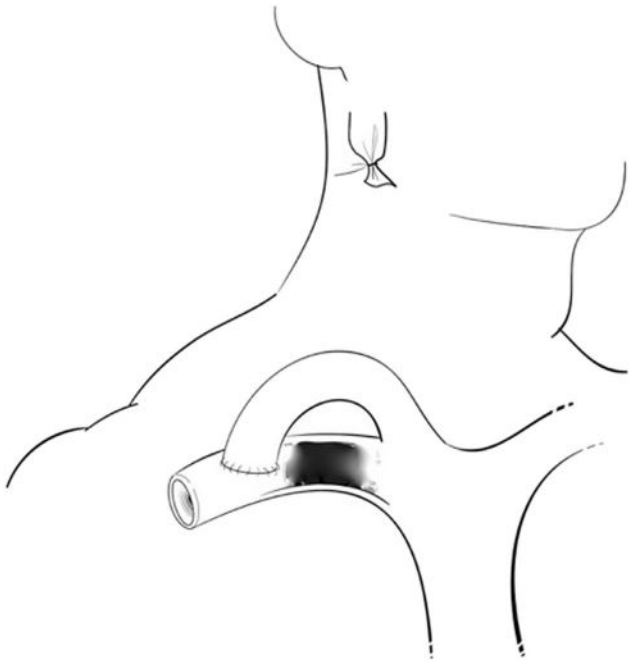

Fig. 19 – Completed axillojugular anastomosis bypassing subclavian vein occlusion.

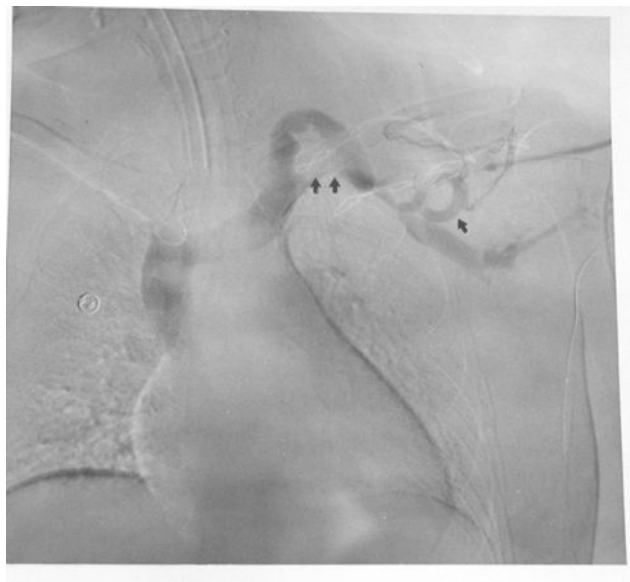

Fig. 20 – Arteriovenous fistula between axillary artery and vein (single arrow). Functioning turndown axillojugular bypass around occluded subclavian vein (double arrow). Reprinted from Sanders RJ, Haug CE. Thoracic outlet Syndrome: A Common Sequela of Neck Injuries. Philadelphia: Lippincott: 1991:256, with permission

6.7. Treating the opposite side

Patients with unilateral symptoms of axillosubclavian vein compression have a high incidence of bilateral involvement . In a study of 15 patients with unilateral symptoms of subclavian vein compression, venograms demonstrated bilateral subclavian vein compression in 80% of the patients [44]. As a result, any patient evaluated for subclavian vein obstruction is offered bilateral dynamic venography. The incidence of symptoms developing in the asymptomatic contralateral arm is estimated to be 15%.

Because this is a prophylactic procedure, patients are presented with the findings on the other side. Some will elect a prophylactic first-rib resection and venolysis. To date, we have not seen another study of the incidence of thrombosis in the contralateral arm. We have not seen any symptoms develop in the patients who underwent prophylactic first-rib resection.

7. VPMS

VPMS is axillary venous thrombosis. The subclavian vein becomes the axillary vein at the lateral border of the first rib. The axillary vein has three segments: one is proximal to the PMM, the second is under the pectoralis minor and the third is lateral to the muscle and extends to the teres major muscle.

7.1. Incidence

VPMS is rare. The first time a series appeared was in 2007 when seven instances of VPMS in six patients were reported [45]. Before that date, there had been only four individual case reports. The only recognized cases have been axillary vein obstruction without thrombosis. Axillary vein thrombosis alone, without subclavian vein thrombosis, has not been reported. Thrombosis of the entire axillosubclavian vein is treated essentially as subclavian vein thrombosis.

7.2. Symptoms

Arm swelling, pain, and cyanosis are the only symptoms of axillary vein obstruction. However, most of the patients seen with VPMS also had additional symptoms of nerve compression, the most common being weakness and paresthesia. This is expected, as some of the pressure exerted by the PMM is often against the whole axillary neurovascular bundle. Because some of the cords of the brachial plexus are adjacent to the axillary vein, this explains the presence of concomitant neurogenic symptoms. Arterial symptoms of arm claudication and ischemia are rarely seen with VPMS.

7.3. Physical examination

The only finding on physical examination is mild arm swelling. This is not as extensive as the swelling seen with subclavian vein obstruction. All of the findings are subtle and mild. Color changes are occasionally seen.

7.4. Etiology

There is no clear-cut single cause that has been recognized as the etiology of VPMS. It may be linked to neurogenic PMS in which the etiology is often repetitive activities with the upper extremities that require pulling back the scapula, such as weight lifting, throwing, swimming, and similar sports. This causes neurogenic symptoms more often than venous ones, but the two conditions may be seen together.

7.5. Diagnostic tests

Venography is the only diagnostic test that can differentiate subclavian vein from axillary vein compression. Duplex scanning is usually not helpful, as it might not distinguish axillary vein occlusion alone from axillosubclavian vein occlusion.

Venography is helpful in that it can detect various degrees of partial obstruction of the axillary vein, but even this is difficult. We have found the most effective technique is to perform dynamic fluoroscopy venography while the arm is moved through from resting at the side to 180-degree abduction.

7.6. Treatment

Surgery is the only effective treatment for axillary vein obstruction. Pectoralis minor tenotomy can be performed through incisions below the clavicle or through the axilla. Our preference is the transaxillary route. Through this approach it is possible to divide any bands of tight clavipectoral fascia as well as any anomalous bands of muscle, or Langer's arch [30], which can occasionally compress the axillary vein and branches of the brachial plexus.

8. Arterial compression: ATOS and APMS (Appendix III)

8.1. Incidence

Although ATOS comprises <1% of all TOS patients, it is the most serious type because ATOS often threatens viability of the hand. The consequences of failing to recognize and treat ATOS properly can be gangrene of digits or the hand.

8.2. Classification

ATOS includes compression, stenosis, occlusion, and aneurysms of the subclavian and axillary arteries. Management of the subclavian artery pathology is different from management of axillary artery pathology. While the presentations of the two conditions are often similar in that they both present with arterial ischemia in the hand, the type of patient, treatment, and prognosis are quite different for the two. It is important to determine the specific site of pathology and to distinguish which of the two sites is the cause. ATOS involving the subclavian artery is usually due to an osseous abnormality, such as a cervical rib, anomalous first rib, or

callus formation from a fractured clavicle. These conditions are identified on plain x-rays of the chest or neck.

In contrast, ATOS involving the axillary artery or its branches occurs less often and is seen primarily in competitive athletes, specifically those who are overhead throwing athletes, such as baseball pitchers and volleyball players, and in people whose occupations require repetitive movements of their arms overhead. Because these two conditions, subclavian artery and axillary artery compression, can be differentiated, we suggest that axillary artery pathology be called arterial pectoralis minor syndrome because the PMM lies over the axillary artery and all of the injuries to the axillary artery and its branches lie either under this muscle or very close to it. Using separate terms for the two conditions helps separate the very different therapeutic approaches for them. This term is in line with the already accepted terms of NPMS and VPMS for neurogenic and venous PMS, respectively.

8.3. *History of terminology*

The suggestion of the term *arterial pectoralis minor syndrome*, or APMS, is in keeping with the introduction of the term *thoracic outlet syndrome*. Beginning in the early 1900s, what is called TOS today was then labeled by the specific anatomical structure, such as scalene anticus syndrome, Naffziger syndrome, scalene medius syndrome, costoclavicular syndrome, cervical rib syndrome, cervical rib syndrome without a cervical rib, first-rib syndrome, anomalous first-rib syndrome, and hyperabduction syndrome. It was suggested in 1956 by Peet et al. [46] and in 1958 by Rob and Standeven [47], that a single term be used to include all of these syndromes under one name, because the symptoms of each syndrome were the same. Because all of these anatomical structures lay behind the clavicle, the area where blood vessels exit the chest, or the thoracic outlet area, the term *thoracic outlet syndrome* was suggested independently by each of the groups [46,47]. The term was readily accepted by physicians because the symptoms for each of these separate syndromes were essentially the same, namely, pain and paresthesia in the upper extremity. Because the specific anatomical structure involved was unknown until further studies or even surgery had been performed, it made sense to have an all-inclusive, but nonspecific term to indicate the area involved.

TOS is defined as upper-extremity symptoms due to compression of the neurovascular bundle in the thoracic outlet area. Recognition that the bundle was composed of arteries, veins, and nerves soon led to qualifying the types of TOS as arterial, venous, or neurogenic, so that the terms *ATOS*, *VTOS*, and *NTOS* appeared shortly after 1956. Realization that subpectoral compression of the same neurovascular bundle that ran through the thoracic outlet area produced the same symptoms as TOS, led next to the introduction of the terms *venous pectoralis minor syndrome* and *neurogenic pectoralis minor syndrome*. Thus, it is only appropriate that recognition of the different types of arterial compression will come from calling compression above the clavicle *arterial thoracic outlet syndrome* and calling compression below the clavicle, in the region of the PMM, *arterial pectoral minor syndrome*.

9. ATOS

9.1. *Anatomy*

The subclavian artery exits the thoracic outlet anterior and inferior to the nerves of the brachial plexus between the scalene muscles in the scalene triangle. It travels over the first rib, beneath the clavicle, and is renamed the axillary artery at the lateral border of the first rib. It is surrounded by the cords of the brachial plexus. It travels under the PMM and next becomes the brachial artery at the lower border of the teres major muscle. Cervical ribs or anomalous first ribs occur in $<1\%$ of the population and can compress the subclavian artery and adjacent nerves in the scalene triangle. Cervical ribs lie in the midst of the middle scalene muscle.

9.2. *Etiology and pathophysiology*

Subclavian artery involvement in ATOS usually results from a cervical rib or anomalous first rib. These bony structures exert pressure on the inferior wall of the subclavian artery, elevating and angulating the artery in the neck. With each heart beat, the artery encounters this unyielding bone. Arm movement increases arterial wall compression. Over time, injury to the arterial intima occurs and fibrosis in the arterial wall develops, causing narrowing within the arterial lumen. Like rapids in a river, the velocity of flow increases through the stenosis, resulting in turbulence and vibrations in the arterial wall. When the turbulence is enough to cause vibrations and a thrill in the arterial wall, elastin fibers in the media are weakened and the wall becomes more distensible, resulting in dilatation and aneurysm formation [48]. Along the wall of the aneurysm, mural thrombus forms, which is the source of distal emboli.

Thrombus can also develop distal to a stenosis without aneurysm formation. This too can embolize distally. Intimal injury causes platelet deposition, which can become microemboli producing Raynaud's phenomenon and digital ischemia in the presence of palpable pulses. Stenotic vessels can also totally thrombose. This stenosis and thrombosis leads to collateral blood flow and milder symptoms to the arm; it can also lead to retrograde thrombosis with the remote possibility of cerebral emboli causing a stroke (Fig. 21).

In the space between the clavicle and first rib, the costoclavicular space, callus resulting from a fracture of the clavicle or first rib, can compress the axillosubclavian artery. Normally, shoulder abduction narrows the costoclavicular space as the clavicle moves posteriorly. Callus from these fractures further narrows this space. Nonosseous causes of arterial occlusion include a fibrous band arising from an elongated transverse process of C7 and compressing the subclavian artery. In athletes, muscle hypertrophy can compress the axillary or subclavian artery. The suprascapular artery can be compressed by the anterior scalene muscle or by the transverse scapular ligament.

9.3. *Symptoms*

Symptoms of arterial ATOS are those of reduced blood flow to the arm. They may start with stenosis in the subclavian

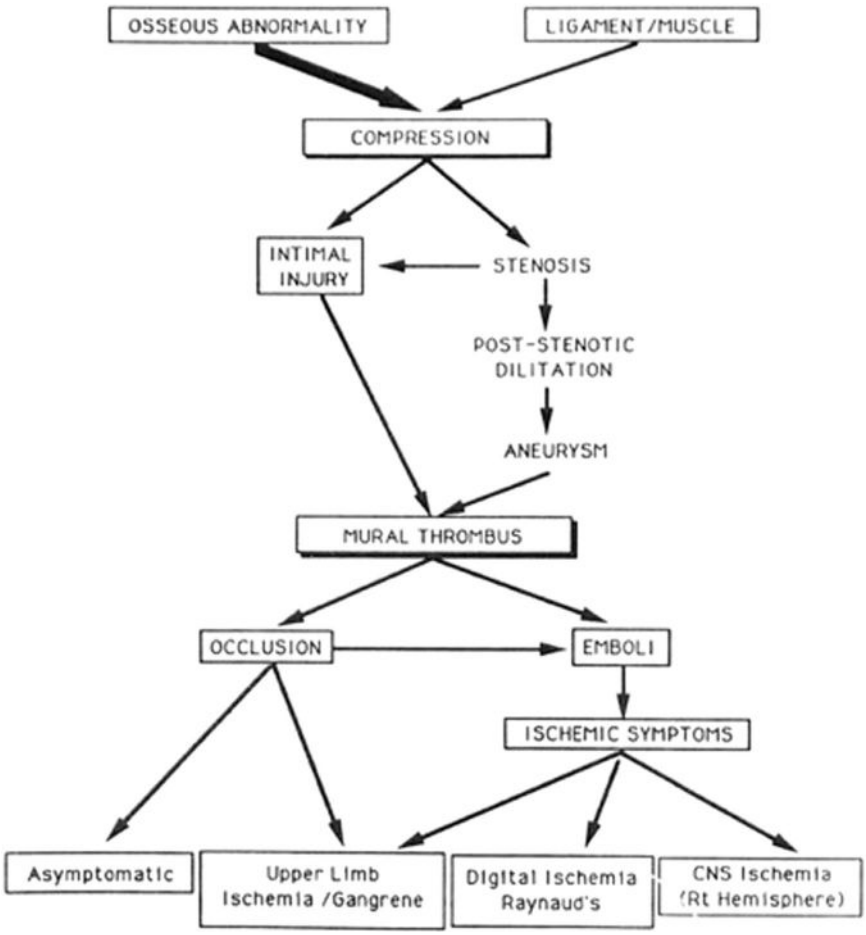

Fig. 21 – Algorithm of pathophysiology of arterial thoracic outlet syndrome. Most cases are associated with a cervical or anomalous first rib. Intimal injury is the primary pathology.

artery or they can begin with micro- or macroemboli below the elbow. Symptoms are similar whether from the subclavian or axillary artery, although larger emboli arise from the subclavian. Symptoms may be vague, insidious, and undiagnosed until acute thrombosis and/or embolus occurs and causes severe arterial insufficiency. The typical symptoms are fatigue and unilateral Raynaud's phenomenon with pallor, cyanosis, cold hypersensitivity, discoloration of fingertips, and digital ischemia. Symptoms may also be severe, developing acutely from larger or multiple emboli and include pain, coldness, digital ulcers or even gangrene.

9.4. *Physical examination*

Cervical ribs may present as a palpable bony mass, sometimes pulsatile and tender, in the supraclavicular fossa. A bruit may be heard in the same space either with the arm at rest or elevated. The brachial, radial, and ulnar pulses may be absent or diminished at rest or normal at rest but absent or diminished with arm abduction.

Blood pressure may be reduced compared to the uninvolved arm. A difference in blood pressure of 20 mm Hg is considered to be significant. The blood pressure should be retaken by the examiner if the first blood pressures taken by others are normal. Digital changes in the nail beds or fingertips may be seen; the hand may be cooler than the uninvolved hand.

Allen's test can reveal occlusion of the radial or ulnar artery. Allen's test is performed by occluding one of these arteries while the pulse in the other artery is either felt or detected by Doppler examination.

9.5. *Diagnostic tests*

Pulse-volume recordings reveal diminished waveforms in the arm and forearm distal to the point of stenosis or obstruction. Digital photoplethysmography reveals digital obstruction rather than vasoconstriction in some or all of the digits. The diagnosis can be aided by warming the hand and noting flat digital waveforms, which indicate obstruction not vasospasm.

Duplex-ultrasound has the advantage of being low-cost, noninvasive, and can be performed in a standing position. It can show stenosis, with increased velocity of flow through the narrowing. Other findings are mural thrombus, arterial dilatation, aneurysms, or thrombotic obstruction with collateral formation. Repeating the study with dynamic positioning may show abnormalities not present at rest.

Imaging studies include cervical spine x-rays with anterior-posterior, lateral and oblique views, and posterior-anterior

and lateral views of the chest. These can reveal osseous abnormalities of the neck, ribs, and clavicle, such as fractures and congenital anomalies. The presence of a cervical or anomalous first rib suggests that the subclavian artery is the source of the pathology. When the chest x-ray is normal, and the patient is an overhead-throwing athlete, such as a baseball pitcher or volleyball player, axillary artery, rather than subclavian artery involvement, should be considered.

MRA can demonstrate impinging soft-tissue structures and arterial irregularity or obstruction. Its accuracy is improved by dynamic positioning in resting and elevated positions. Its advantages are that it does not require ionizing radiation or iodinated dye and is the best technique to demonstrate soft-tissue structures. Disadvantages of MRA include cost, the need for the patient to lie still for prolonged period of time, inability to visualize bony structures, and the need to perform it in a supine position. It cannot be performed in patients with metal implants, such as hip or knee prostheses. Rarely will a thrombus be missed.

CTA has the advantage of excellent arterial visualization, rapid acquisition of images, and demonstration of bony structures in relation to the artery. Here too, dynamic imaging with arm elevation improves accuracy of the diagnosis. The disadvantages of CTA include exposure to ionizing radiation, requirement of an iodinated contrast agent, failure to demonstrate soft tissues as well with MRI, and the need for acquisition in a supine position, whereas the symptoms occur when the patient is upright.

Digital subtraction arteriography (DSA) is the intra-arterial injection of contrast material with a technique that removes obstructing bone from the films. DSA has the advantage of demonstrating the entire arterial anatomy from shoulder to fingertip, it can be used along with intravascular ultrasound to demonstrate subtle arterial wall irregularities, it can be performed with dynamic positioning, and it can allow therapeutic procedures to be performed at the same sitting if indicated. Disadvantages of DSA include invasiveness of the procedure, which requires an arterial puncture, the need for iodinated contrast, and radiation exposure.

9.6. *Treatment*

ATOS is a surgical problem. Acute arterial symptoms are an indication for immediate surgical intervention. Delay in treatment can lead to further complications and worsen prognosis. In contrast, subtle, chronic symptoms allow for study and planning of a treatment approach. Physical therapy has nothing to offer before surgery.

Treatment includes 3 steps: remove, repair, restore.

1. Remove the cause: Removing the cause of the arterial emboli is achieved by excising the abnormal cervical or anomalous first rib, whenever present. If the cause is a hypertrophied muscle, this is excised.
2. Repair or replace the artery: The subclavian artery can best be approached with a supraclavicular incision through which resection of cervical and first ribs can be achieved. This incision allows complete examination of the artery and permits its repair or replacement through the same incision. In some cases after rib excision, the abnormal segment of artery can be resected and an end-to-end anastomosis performed. Once the osseous abnormality has been removed, the artery can drop caudally permitting resection of 1 to 2 cm of artery and still allowing a tension-free end-to-end anastomosis. If the length of artery to be resected is too long for an end-to-end anastomosis, an interposition or bypass graft can be performed by exposing the axillary artery through a second, infraclavicular incision, in the same surgical field.
   There are several types of graft material from which to choose. When available, autologous saphenous vein is the material of choice. It will maintain patency at low-flow rates at which prosthetic grafts will thrombose.
3. Restore distal circulation: Emboli to the forearm and hand are often the cause of symptoms. Attempts to restore the distal circulation should be performed during the same procedure that corrects the proximal artery. To accomplish this, the entire upper extremity is prepped and draped into the operative field at the same time that the proximal areas are prepped. The distal brachial artery and origins of the ulnar and radial arteries are exposed at the antecubital fossa. With systemic heparinization, Fogarty balloon embolectomy catheters are passed proximally and distally with the goal of restoring normal blood flow. Post-embolectomy arteriograms are performed on the operating table to confirm distal patency. If small-vessel patency is not achieved, thrombolytic infusion is undertaken by slow intra-arterial injection into the peripheral area, over 30 to 60 minutes through the open vessel. In rare cases, a distal bypass graft to the wrist may be required for limb salvage. Postoperatively, anticoagulation and antiplatelet therapy may be required.

9.6.1. *Dorsal sympathectomy*

When thrombi in the digital arteries persist in spite of thrombolytic therapy, and if digital ischemia persists, dorsal sympathectomy has been performed in an effort to increase small vessel flow.

9.7. *Results of treatment*

The causes and results of treatment for 137 patients operated upon for ATOS between 1970 and 1990 are summarized in Table 7 [49]. An osseous etiology was noted in 88% of the patients, and nonosseous causes were seen in 12%. Rib resection without arterial reconstruction was performed in 30%, while the other 70% required some type of arterial repair. In 26%, resection of the diseased arterial segment with end-to-end anastomoses was possible, while 22% required an interposition or bypass graft. The success rate was good to excellent in 84%, failure in 10%, and 3% required amputation. Strokes from retrograde cerebral emboli occurred in 3%.

10. Arterial pectoralis minor syndrome

10.1. *Anatomy*

The axillary artery has six branches, one proximal to the PMM, two deep to the muscle belly, and three distal to the

Table 7 – Treatment and results for arterial thoracic outlet syndrome 137 patients: summary of 17 series from 1970 to 1990 [49].

	%
Etiology	
Cervical ribs	66
Anomalous first ribs	19
No osseous abnormality	12
Fracture clavicle or first rib	3
Treatment	
Resection cervical and/or first rib	30
Arterial resection, end-to-end anastomosis	26
Replacement graft	22
Repair—endarterectomy and/or patch graft	17
Thrombectomy/embolectomy	4
Results of treatment	
Good to excellent	84
Failed	10
Amputation	3
CVA (stroke)	3

Abbreviation: CVA, cerebrovascular accident.

lateral border of the muscle. The three distal branches are the posterior circumflex humeral, subscapular, and anterior circumflex humeral arteries. The posterior circumflex humeral artery (PCHA), traverses the quadrilateral space (between teres minor, teres major, long head of the triceps, and humerus) and can be compressed along with the axillary nerve (Fig. 22 quadrilateral space, axillary artery). The PCHA is the branch most often involved in APMS.

10.2. *Etiology and pathophysiology*

The axillary artery, and particularly its PCHA, can be compressed in a different way than the subclavian artery. The PMM itself can compress the portion of the axillary artery beneath it. The subscapular artery can be compressed adjacent to the PCHA by compressing it in the quadrilateral space. The head of the humerus can compress the lateral portion of the axillary artery during vigorous throwing. The axillary artery or its circumflex humeral or subscapular branches can also be injured by blunt trauma. Because the PCHA winds around the humeral head, it can be stretched with forceful overhead motions of the arm. This produces traction at the junction of the PCHA with the axillary artery, thereby weakening the wall at this junction. This can lead to intimal lesions, aneurysmal degeneration, and subsequent thrombosis. As overhead action continues, the thrombus is now compressed and portions can enter the axillary artery and be washed downstream as emboli.

10.3. *Symptoms*

Symptoms of APMS are similar to those of ATOS, but they tend to be more mild and subtle. Some pitchers note a decrease in speed or loss of control while pitching. Coolness in the fingers of the pitching hand and loss of endurance during competition or practice are early signs of axillary artery stenosis resulting in diminished blood flow to the extremity. Arm claudication, fatigue, and eventually pain, numbness, tingling, and digital ulcerations are evidence of progression. Neurological symptoms of pain and paresthesia may accompany arterial symptoms because the axillary nerve can be compressed in the quadrilateral space along with the PCHA.

10.4. *Physical examination*

The specific findings on physical examination with axillary artery involvement are tenderness just below the clavicle over the PMM and/or tenderness over the quadrilateral space, located just behind the posterior axillary fold. Radial and/or ulnar pulses may be diminished or absent; blood pressure may be diminished and coolness and color changes may be detected when compared to the good arm and hand.

10.5. *Diagnostic tests*

The imaging tests for APMS are the same as for ATOS and are described in see Section 3.6.1. PMM block. It should be emphasized that detailed views of the axillary artery and each of its branches should be visualized to insure an accurate diagnosis. Although DSA is the technique most often employed, in some instances, MRA or CTA may provide additional information.

10.6. *Treatment*

Treatment of APMS is less complicated than that for ATOS. In APMS, an osseous cause is not involved; the only pathology is

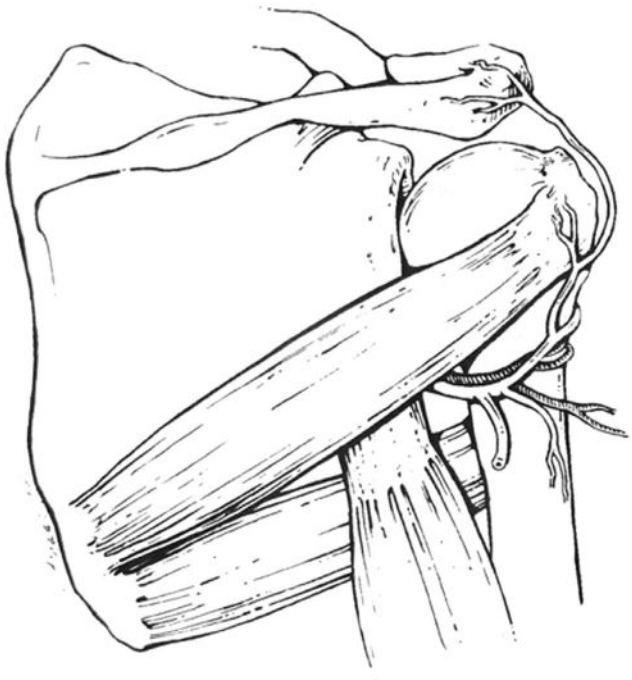

Fig. 22 – Quadrilateral space bounded by teres major inferiorly; teres minor superiorly; long head of triceps medially; humerus laterally. The posterior circumflex humeral artery and axillary nerve wrapping around head of humerus.

Table 8 – Results of axillary artery treatment for arterial pectoralis minor syndrome.

First author, year	Pathology	No.	Treatment	Result	Follow-up
Atema, 2012 [50]	PCHA Occl	3	Ligation and oversew ostium	All 3 returned to their sport	6–12 mo
Duwayri, 2011 [51]	AxArtOccl	4	Interposition graft	All 9 patients Returned to their sport	Mean 15 mo (3–120 mo)
	AxArtStn	3	Lateral repair		
	PCHA	2	Ligation PCHA		
Arko, 2001 [52]	PCHA Occl	4	Lateral repair	6 of 7 returned to their sport	Mean 42 (2–72 mo)
		2	Interposition graft		
		1	Antiplatelet drug		
Schneider, 1999 [53]	AxArtAneur	1	Interposition graft	Returned to pitching	3 mo
Durham, 1995 [54]	AxArtHuHdC	4	Lateral repair	4 of 5 returned to pitching or work	
	PCHA Aneur	1	PMT		
Finkelstein, 1993 [55]	AxArtComp By PMM	1	PMT	Total recovery	9 mo
McCarthy, 1989 [56]	AxArtComp By PMM	3	PMT	All 3 returned to pitching	Not stated
Totals		29		27 of 29 returned to competition	

Abbreviations: Aneur, aneurysm; AxAHuHdC, axillary artery-humeral head compression; AxArt, axillary artery; Comp, compression; Occl, occlusion; PCHA, posterior circumflex humeral artery; PMM, pectoralis minor muscle; PMT, pectoralis minor tenotomy.

in or around the axillary artery. Angiography that details the specific area to be treated is essential.

Thrombolysis, either before or during operative repair, can dissolve thrombi in the digital vessels. In the operating room, the entire upper extremity should be prepped into the field for possible angiography and thrombolysis via the antecubital space.

Axillary artery repair is performed through an upper arm incision, extended over the lateral border of the pectoralis major muscle. This allows exposure and release of the PMM and ligation of involved branches and/or repair of the axillary artery.

When the pathology is thrombosis of the posterior circumflex humeral artery, that artery should be divided at or near its axillary artery origin. If the origin encroaches on the axillary artery, as aneurysms can do, repair of the artery with a vein patch will prevent a stricture at this spot. If the axillary artery is occluded or narrowed over a significant distance, an interposition graft may be required.

Interposition grafts in the axillary artery require special attention to ensure that the repaired artery will not be reinjured by overstretching when overhead activity is resumed. In the operating room, the length of the graft should be carefully measured with the arm fully extended. The graft material should be autogenous vein if at all possible, and the graft should have widely beveled end-to-end anastomoses [50,51]. Completion arteriography should be performed immediately after completing the anastomoses.

10.7. *Results of treatment*

In a review of 29 published cases of APMS, 7 required interposition grafts, 11 were treated with lateral repairs and patch grafts, 5 were treated with simple ligations of the posterior circumflex humeral artery, 5 were treated with PMT, and 1 was treated with antiplatelet drugs [50–56] (Table 8). Two patients were operated on a second time for additional decompression. Twenty-seven of the 29 returned to their previous work or competitive sports. The other two were symptom free but retired from their previous activity. There were no amputations and no disability.

Appendix I. Brachial plexus compression: NTOS and NPMS

Presentation: Pain in upper extremity, neck, and anterior chest; occipital headaches; and numbness, tingling, and weakness of the upper extremity.

Physical examination: Tenderness over the scalene muscles above the clavicle and PMMs and axilla below the clavicle. Positive responses to four provocative maneuvers: Upper limb tension test, elevated arm stress test, head rotation, and head tilt.

Pathology: NTOS is due to compression of the brachial plexus by scarred scalene muscles or a cervical or anomalous first rib. NPMS is due to a scarred PMM.

Etiology: Neck trauma: auto collisions or falls can cause symptoms of NTOS and/or NPMS; repetitive stress injury from upper extremity sports can cause NPMS. Cervical ribs are usually a predisposing factor.

Diagnostic tests: Pectoralis minor and scalene muscle blocks; measurement of sensory medial antebrachial cutaneous nerves, C8 nerve conduction velocity.

Differential and associated diagnoses: Several conditions can coexist with brachial plexus compression or must be differentiated from it. These include cervical spine disease, shoulder pathology, cuboid tunnel, carpal tunnel, pronator tunnel, and radial tunnel syndromes, multiple sclerosis, and cerebellar tonsillar herniation (Chiari malformation).

Treatment: Treatment options include PT or surgery. Surgery for NTOS is either transaxillary first-rib resection and neurolysis or supraclavicular scalenectomy and neurolysis with or without first-rib resection. Surgery for NPMS is pectoralis minor tenotomy alone, or in combination with thoracic outlet decompression for NTOS.

Results: Results from physical therapy depend on duration of symptoms before initiation of physical therapy. This is in part because many patients improve without any therapy. Physical therapy that begins many months after the onset of symptoms has a smaller percentage of success. The results of surgery for NTOS by either transaxillary or supraclavicular approach are about the same: good to excellent improvement in 75% to 80% of patients. Results of surgery for NPMS alone are about the same.

Appendix II. Venous compression: VTOS and VPMS.

Presentation: Arm swelling, discoloration, and possibly pain, and paresthesia.

Pathology: Obstruction, with or without thrombosis, of the subclavian or axillary vein.

Physical examination: Swollen, discolored arm and venous distension

Etiology: Repetitive overhead upper-extremity activity plus predisposing anatomical factor of subclavian vein lying in contact with the costoclavicular ligament or subclavius tendon and may also have a hypercoagulable state. VPMS is much less common and is due to a tight PMM compressing the axillary vein.

Diagnostic tests: Ultrasound duplex scan and dynamic venography

Treatment: 3 steps for venous obstruction with thrombosis:

1. Pharmacomechanical thrombolysis (clot dissolution);
2. First rib resection and venolysis for VTOS; pectoralis minor tenotomy for VPMS; and
3. Venogram and if necessary, balloon angioplasty or rarely open angioplasty and venous reconstruction

For venous obstruction without thrombosis: first-rib resection for VTOS; pectoralis minor tenotomy for VPMS

Results: For VTOS with thrombosis, if treatment is initiated within 7 to 14 days, >95% success. If longer than 14 days until initiation of therapy, 50% to 75% success. For venous obstruction without thrombosis, either VTOS or VPMS, success is >90%.

Appendix III. Arterial compression: ATOS and APMS

Presentation: Cold, discolored, painful hand; possible numbness or tingling; or possible ischemia or gangrene of fingers

Physical examination: Sometimes no wrist pulse, reduced blood pressure, and ischemic fingers

Etiology: ATOS is due to a cervical or anomalous first rib causing subclavian artery thrombus, with or without aneurysm formation, and emboli to the hand or fingers. APMS is usually seen in competitive athletes who throw overhead and is due to obstruction or aneurysm formation with thrombosis of the axillary artery or one of its branches causing emboli to the hand or fingers.

Diagnostic tests: Pulse-volume recording (vascular lab), arteriogram, neck and chest x-ray

Treatment: For ATOS, resection of cervical rib and/or first rib and repair or replacement of the artery as indicated. For APMS, ligate axillary branch for axillary branch aneurysm; replace the diseased segment for axillary artery pathology.

Results: For ATOS, success is >80% good, 10% fair, 3% amputation, and 3% stroke. For APMS, results are much better, >95% successful.

REFERENCES

[1] Upton ARM, McComas AJ. The double crush in nerve-entrapment syndromes. Lancet 1973;2:359–62.
[2] Illig KA, Thompson RS, Freischlag JA, et al. eds. Thoracic Outlet Syndrome. New York: Springer; 2013: XIV.
[3] Haven H. Neurocirculatory scalenus anticus syndrome in the presence of developmental defects of the first rib. Yale J Biol 1939;11:443–8.
[4] Elvey RL. The investigation of arm pain. In: Grieve GP, ed. Modern Manual Therapy of the Vertebral Column. Edinburgh: Churchill Livingstone; 1986:530–5.
[5] Adson AW, Coffey JR. Cervical rib: a method of anterior approach for relief of symptoms by division of the scalenus anticus. Ann Surg 1927;85:839–57.
[6] Gergoudis R, Barnes RW. Thoracic outlet arterial compression: prevalence in normal persons. Angiology 1980;31:538–541.
[7] Warrens A, Heaton JM. Thoracic outlet compression syndrome: the lack of reliability of its clinical assessment. Ann R Coll Surg Engl 1987;69:203–4.
[8] Colon E, Westdrop R. Vascular compression in the thoracic outlet: age dependent normative values in noninvasive testing. J Cardiovasc Surg 1988;29:166–71.
[9] Plewa MC, Delinger M. The false-positive rate of thoracic outlet syndrome shoulder maneuvers in healthy subjects. Acad Emerg Med 1998;5:337–42.
[10] Nord KM, Kapoor P, Fisher J, et al. False positive rate of thoracic outlet syndrome diagnostic maneuvers. Electromyogr Clin Neurophysiol 2008;48:67–74.
[11] Nishida T, Price SJ, Minieka MM. Medial antebrachial cutaneous nerve conduction in true neurogenic thoracic outlet syndrome. Electromyogr Clin Neurophysiol 1993;33:255–8.
[12] Kothari MJ, Macintosh K, Heistand M, Logigian EL. Medial antebrachial cutaneous sensory studies in the evaluation of neurogenic thoracic outlet syndrome. Muscle Nerve 1998;21:647–9.
[13] Seror P. Medial antebrachial cutaneous nerve conduction study, a new tool to demonstrate mild lower brachial plexus lesions. A report of 16 cases. Clin Neurophysiol 2004;115:2316–22.
[14] Machanic BI, Sanders RJ. Medial antebrachial cutaneous nerve measurements to diagnose neurogenic thoracic outlet syndrome. Ann Vasc Surg 2008;22:248–54.
[15] Boulanger X, Ledoux JB, Brun AL, et al. Imaging of the non-traumatic brachial plexus. Diagn Interv Imaging 2013;94:945–56.
[16] Filler A. Magnetic resonance neurography and diffusion tensor imaging: origins, history, and clinical impact of the first 50,000 cases with an assessment of efficacy and utility in a prospective 5000-patient study group. Neurosurgery 2009;65(suppl):A29–43.
[17] Edgelow PI. Physical therapy for NTOS. In: Illig KA, Thompson RW, Freischlag JA, et al. eds. Thoracic Outlet Syndrome. London: Springer-Verlag; 2013:167–73.

[18] Buchanan PA1, Ulrich BD. The Feldenkrais Method: a dynamic approach to changing motor behavior. Res Q Exerc Sport 2001;72:315–23.
[19] Chandra V1, Olcott C 4th, Lee JT. Early results of a highly selective algorithm for surgery on patients with neurogenic thoracic outlet syndrome. J Vasc Surg 2011;54:1698–705.
[20] Robicsek F1, Eastman D. "Above-under" exposure of the first rib: a modified approach for the treatment of thoracic outlet syndrome. Ann Vasc Surg 1997;11:304–6.
[21] Thompson RW, Schneider PA, Nelken NA, et al. Circumferential venolysis and paraclavicular thoracic outlet decompression for "effort thrombosis" of the subclavian vein. J Vasc Surg 1992;16:723–32.
[22] Sanders RJ, Pearce WH. The treatment of thoracic outlet syndrome: a comparison of different operations. J Vasc Surg 1989;10:626–34.
[23] Sanders RJ, Roos DB. The surgical anatomy of the scalene triangle. Contemp Surg 1989;35:11–6.
[24] Ambrad-Chalela E, Thomas GI, Johansen KH. Recurrent neurogenic thoracic outlet syndrome. Am J Surg 2004;187:505–10.
[25] Sanders RJ. Recurrent neurogenic thoracic outlet syndrome stressing the importance of pectoralis minor syndrome. Vasc Endovasc Surg 2011;45:33–8.
[26] Annest SJ, Sanders RJ. Assessment and treatment of recurrent NTOS. In: Illig KA, Thompson RW, Freischlag JA, et al. eds. Thoracic Outlet Syndrome. London: Springer-Verlag; 2013:281–9.
[27] Sanders RJ, Hammond SL. Diagnosis of thoracic outlet syndrome. J Vasc Surg 2007;46:601–4.
[28] Sanders RJ, Annest SJ, Goldson E. Neurogenic thoracic outlet and pectoralis minor syndromes in children. Vasc Endovasc Surg 2013;47:335–41.
[29] Sanders RJ, Rao NM. The forgotten pectoralis minor syndrome: 100 operations for pectoralis minor syndrome alone or accompanied by neurogenic thoracic outlet syndrome. Ann Vasc Surg 2010;24:701–8.
[30] Magee C1, Jones C, McIntosh S, Harkin DW. Upper limb deep vein thrombosis due to Langer's axillary arch. J Vasc Surg 2012;55:234–6.
[31] Paget J. Clinical lectures and essays. London; 1975.
[32] von Schrotter L. Handbuch der allgemeinen pathologie und therapie (Nothnagel). Berlin: A. Hirschwald; 1884:533 [Cited by Sampson JJ. Am Heart J 1943;25:313].
[33] Heron E, Lozinguez O, Laurian C, et al. Long term sequelae of spontaneous axillary subclavian vein thrombosis. Ann Int Med 1999;131:510–3.
[34] Beygui RE, Olcott C 4th, Dalman RI. Subclavian vein thrombosis: outcome based on etiology and modality of treatment. Ann Vasc Surg 1997;11:247–55.
[35] Doyle A, Wolford HY, Davies MT, et al. Management of effort thrombosis of the subclavian vein: today's treatment. Ann Vasc Surg 2007;21:723–9.
[36] Lin PH, Zho W, Dardik A, et al. Catheter directed thrombolysis versus pharmacomechanical thrombectomy for treatment of symptomatic lower extremity deep venous thrombosis. Am J Surg 2006;192:782–8.
[37] Malgor RD, Gasparis. Pharmaco-mechanical thrombectomy for early thrombus removal. Phlebology 2012;27:155–62.
[38] Illig KA, Doyle AJ. A comprehensive review of Paget-Schroetter syndrome. J Vasc Surg 2010;51:1538–47.
[39] Perler BA, Mitchell SE. Percutaneous transluminal angioplasty and transaxillary first rib resection. A multidisciplinary approach to the thoracic outlet syndrome. Am Surg 1986;52:485–8.
[40] Antithrombotic Therapy and Prevention of Thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines. Chest 2012;141(suppl).
[41] Molina JE. A new surgical approach to the innominate and subclavian vein. J Vasc Surg 1998;27:576–81.
[42] Rubright J, Kelleher P, Beardsley C, et al. Long-term clinical outcomes, motion, strength, and function after total clavicuelectomy. J Shoulder Elbow Surg 2014;23:236–44.
[43] Johnson V, Eiseman B. Evaluation of arteriovenous shunt to maintain patency of venous autograft. Am J Surg 1969;118:915–20.
[44] Kunkel JM, Machleder HI. Treatment of Paget-Schroetter syndrome: a staged, multidisciplinary approach. Arch Surg 1989;124:1153–8.
[45] Sanders RJ, Rao NM. Pectoralis minor obstruction of the axillary vein: report of six patients. J Vasc Surg 2007;45:1206–11.
[46] Peet RM, Hendriksen JD, Anderson TP, et al. Thoracic outlet syndrome: evaluation of a therapeutic exercise program. Proc Mayo Clin 1956;31:281–7.
[47] Rob CG, Standeven A. Arterial occlusion complicating thoracic outlet compression syndrome. Br Med J 1958;2:709–12.
[48] Roach MR. Changes in arterial distensibility as a cause of poststenotic dilatation. Am J Cardiol 1963;12:802–15.
[49] Sanders RJ, Haug CE. Thoracic outlet syndrome: a common sequela of neck injuries. Philadelphia: Lippincott; 1991;226.
[50] Atema JJ, Unlu C, Reekers JA, et al. Posterior circumflex humeral artery (PCHA) injury with distal embolizaion in professional volleyball players: discussion of three cases. Eur J Vasc endovascular Surg 2012;44:195–8.
[51] Duwayri YM, Emery VB, Driskill MR, et al. Position compression of the axillary artery causing upper extremity thrombosis and embolism in the elite overhead throwing athlete. J Vasc Surg 2011;53:1329–40.
[52] Arko FR, Harris EJ, Zarins CK, et al. Vascular complication in high-performance athletes. J Vasc Surg 2001;33:935–42.
[53] Schneider K, Kasparyan NG, Altchek DW, et al. An aneurysm involving the axillary artery and its branch vessels in a major league baseball pitcher. Am J Sports Med 1999;27:370–5.
[54] Durham JR, Yao JST, Pearce WH, et al. Arterial injuries in the thoracic outlet syndrome. J Vasc Surg 1995;21:57–70.
[55] Finkelstein JA, Johnston KW. Thrombosis of the axillary artery secondary to compression by the pectoralis minor muscle. Ann Vasc Surg 1993;7:287–90.
[56] McCarthy WJ, Yao JST, Schafer MF, et al. Upper extremity arterial injury in athletes. J Vasc Surg 1989;9:317–27.