Thoracic outlet syndrome— A myofascial variant: Part 1. Pathology and diagnosis

BENJAMIN MARC SUCHER, DO

Four cases of thoracic outlet syndrome are described, with observations to support a primary myofascial etiology involving the scalene and smaller pectoral muscles. It is believed that thermography can be extremely helpful as an aid in diagnosis of thoracic outlet syndrome and, when combined with Doppler blood flow studies and photoplethysmography, can help localize the site of the pathosis. Thermography is a sensitive, non-invasive test that most clearly demonstrates pathology in the hand views. Global changes throughout the entire hand suggest vascular or reflex autonomic involvement, while a more dermatomal distribution suggests specific neurologic involvement. Vigorous stretching and a unique form of myofascial release manipulation has been successful in rapidly treating patients with thoracic outlet syndrome. Follow-up thermography (perhaps hand and forearm views only) can be used to monitor response to treatment and to objectively document improvement.

The syndrome of pain, paresthesia, and weakness, or "heaviness," in the arms is fairly well known to clinicians, especially physiatrists and neurologists. This syndrome is particularly common following trauma,¹⁻⁴ and it often

is aggravated by elevation of the arms or by exaggerated movements of the head and neck. The diagnosis of thoracic outlet syndrome (TOS) must be considered, especially when the electromyographic examination reveals no evidence of radiculopathy, carpal tunnel syndrome, or other forms of nerve injury. However, controversy persists regarding the diagnosis and existence of TOS,⁴⁻⁶ especially as to whether the pathology is primarily vascular or neurogenic.⁷ Interestingly, most cases appear to have a myofascial etiology with secondary pathologic effects on vascular or neurogenic structures (or both).^{8,9}

It is often difficult to confirm the diagnosis, leading some authors to consider many cases as "disputed neurogenic" TOS^{5,7} or "nonspecific neurogenic" TOS.⁶ No clear-cut diagnostic test has been reported or accepted. Several patients with TOS end up undergoing first rib resection without relief of symptoms, or with development of complications (or both).^{4,7,10}

Part 1 of this article highlights diagnostic criteria and testing that help clarify the problem and direct, as well as monitor, treatment, with particular emphasis on thermography because it is noninvasive and has a high level of sensitivity. Four illustrative case studies are presented as well.

Etiology

In most cases, TOS is probably secondary to scalene or pectoral muscle restriction (or both) with overlying or underlying structural and postural changes.^{2,8,9,11} Cervical ribs or bands

Reprint requests to Benjamin Marc Sucher, DO, 10555 N Tatum Blvd, Suite A-104, Mountain View Center, Paradise Valley, AZ 85253.

Figure 1. *Progressive postural distortion/decompensation with neurovascular compression. A: Normal resting posture. B: Shoulder protraction beginning; sternomastoid muscles are shortening, drawing head anteriorly and inferiorly. C: Advanced deformity with adaptive shortening of scalene and smaller pectoral muscles. Note narrowed costoclavicular space as well (ribs 1 through 5 have been relatively elevated). Neurovascular compression is evident at all three sites.*

are fairly infrequent causes of TOS.^{3,6,12-14} Postural and structural changes probably are predominantly preexisting and complicated or aggravated by stress, trauma, and so forth. The resultant increased tension in the involved muscles subsequently leads to progressive decompensation or strain patterns¹⁵ (Fig 1). Many authors^{16,17} have presented strong claims that the primary problem is vascular, whereas others^{1,18} believe that it is neurogenic. Yet, several authors^{6,14} acknowledge both components and various circumstances in which one component is more involved than the other. However, whether it is a vascular or neurogenic component (or both) that predominates, it should be apparent that the etiology is usually myofascial, as clearly recognized by Travell and Simons.⁹

On close visual (and palpatory) examination of patients, postural effects usually are apparent, and probably predispose affected individuals to compression.^{8,15} Interestingly, several authors^{2,8,10,14,19-21} base their recommendation

for the typical exercise program for TOS on the theory that the shoulder girdle has "dropped" or collapsed, owing to weakness, thereby creating a secondary stress or compression effect on the neurovascular bundle within the thoracic outlet. On careful examination, however, it appears more evident that the shoulder girdle has not really dropped at all, but is literally "pulled" anteriorly and inferiorly, secondary to tight, contracted muscles.

In addition to local compressive effects at the scalene and pectoral sites, it is now obvious that costoclavicular compression is possible in that the clavicle is pulled inferiorly with the shoulder girdle as the ribs are elevated. The first and second ribs are elevated by the scalene muscles, and the third through the fifth ribs are elevated by the smaller pectoral muscles (Fig 1C).

Diagnostic tests

The diagnostic tests currently available for TOS can be separated into two main categories.

ries (or fields): anatomic and physiologic; and two main divisions (or systems): vascular and neurologic.

Anatomic studies include x-ray examination, computed tomography (CT), and magnetic resonance imaging (MRI), and could include angiography/venography as well. Physiologic studies include electromyography and thermography, and could include Doppler blood flow studies with photoplethysmography (Doppler studies).

Vascular studies include Doppler studies, angiography/venography, and thermography. Neurologic studies include electromyography and thermography (Table 1).

Thermography

Examination by thermography is relatively noninvasive and covers most of the foregoing categories/divisions. Therefore, it would seem to be the most widely useful examination, especially as a screening examination. Several authors^{2,18,22,23} have used or have mentioned the use of thermography as an aid in diagnosis of TOS. For patients who have intermittent or positionally related symptoms, stress thermography is recommended.¹⁸ This procedure, which involves adding stress maneuvers to reproduce symptoms and then obtaining additional sets of thermographic images, enhances sensitivity. However, the relative lack of specificity and variations of abnormal that can occur make interpretation of thermography difficult—or at times limited—in diagnosing TOS, depending on the pathology involved. For example, arterial compression could reveal decreased heat emission secondary to decreased blood flow that could involve the entire hand or all the digits, and usually parts of the forearm and arm as well. Increased or decreased heat emission from the cervical, scapular, and suprascapular regions are not uncommon, secondarily to either reflex phenomena or local musculoligamentous changes, because myofascial pain often accompanies or causes TOS. Venous occlusion could reveal the opposite, with relative increase in heat emission in the involved extremity.

Also, even if arterial involvement is primary, reflex autonomic peripheral vasodila-

tion could still yield an increased heat emission distally. In addition, the pattern of increased or decreased heat emission diffusely, distally, could be consistent with reflex sympathetic dystrophy, a differential diagnosis considered with TOS. Reflex sympathetic dystrophy has been reported to coexist with TOS.^{6,22} However, none of the patients described here had dystrophy.

Thermography may reveal a specific neurogenic picture, with the most ideal pattern demonstrating cold fourth and fifth digits (occasionally the third digit), as well as cold medial aspect of the forearm, and possibly cold dorsal aspect of the forearm. However, this somewhat dermatomal pattern could be confused with that of C7 or C8 radiculopathy (especially if shoulder girdle or cervical abnormalities [or both] are seen) or with that of ulnar nerve injury at the elbow.

Electromyography

In addition to helping rule out a pathologic condition at other locations (carpal tunnel, cubital tunnel, cervical root, et cetera), several other reasons exist for electromyographic examination. Even though findings of Doppler studies or thermography might be normal, motor abnormalities could still appear on electromyograms. If the findings of the Doppler studies and thermography are abnormal, it is still important to determine whether any electrical evidence of nerve injury exists, because this finding places the syndrome into a more severe category. Also, subsequent follow-up electromyograms (EMGs) are helpful for assessing the patient's status. Even if thermograms reveal changes consistent with nerve involvement, they cannot provide detailed electrical information that more specifically delineates the pathology, its extent, exact location, and prognosis.

The results of most electromyographic examinations in patients with mild or "nonspecific/disputed" neurogenic TOS are normal.^{6,10,12,13,20,24} The most sensitive indicators for TOS seen on EMGs are seen in the so-called true neurogenic TOS and appear to involve the following: (1) low-amplitude median motor response; (2) low-amplitude ulnar sen-

Table
Diagnostic Procedures for Thoracic Outlet Syndrome

Category (field)		Division (system)	
Anatomic	Physiologic	Vascular	Neurologic
Radiography	Electromyography	Angiography/ venography	Electromyography
Angiography/ venography	Thermography	Thermography	Thermography
Computer tomography	Doppler blood flow studies/photo- plethysmography	Doppler blood flow studies/photo- plethysmography	
Magnetic resonance imaging			

sory response; (3) neurogenic motor unit potentials within the C8/T1 myotomes; and (4) normal median sensory response. The existence of all four of these responses is considered pathognomonic for true neurogenic TOS.²⁵

Some authors believe that F waves^{12,16,18,26} and, more specifically, axillary F-loop latencies,²⁷ are helpful as well. The F wave is elicited distally, although it is representative of impulse conduction from the peripheral portion of the extremity to the spinal cord and back. In the axillary F-loop calculation, the arm and forearm segments are subtracted to provide a conduction time from the axilla to the spinal cord and back, which would therefore represent the time necessary to travel in a loop through the brachial plexus.²⁷ However, F-wave studies are generally less promising, less reliable, and very uncomfortable for the patient.^{1,6,11,12,25} Somatosensory-evoked potentials have also been advocated, but they probably are not reliable or worthwhile either.^{6,28} Although not universally accepted, root stimulation may be helpful.^{12,17} However, most clinicians do agree on the reliability of low-amplitude ulnar sensory responses as being somewhat indicative of TOS, as long as ulnar conduction in the wrist and elbow are within normal limits.^{1,12,14,16,18,25,26}

If there is sensory loss clinically from the medial forearm, medial antebrachial cutaneous nerve studies probably should be done.^{1,29} With full-blown medial cord plexopathy, denervation should be obvious within this distri-

bution, without paraspinous abnormalities, and therefore helpful to confirm the diagnosis. However, this latter condition is quite rare, with usually only mild "nerve irritation" or minor compressive sequelae being present.

Overview of diagnostic tests

It is desirable to see the results of two physiologic tests (Table 1) consistent with, or at least suggestive of, TOS, together with a history and clinical findings positive for the syndrome. Thermographic findings, especially when correlated with results of the clinical examination and findings of Doppler studies, appear to provide objective evidence of TOS. Therefore, thermography may be especially useful in the majority of cases of TOS, which some believe are consistent with the disputed or non-specific neurogenic TOS group.⁵⁻⁷

The EMG should be helpful when there is a question regarding the pattern of abnormality on a thermogram. If the results of these studies point toward a radiculopathy, an MRI (or enhanced CT scan) of the cervical spine will provide the answer. If the pathology looks more like plexopathy (TOS), the Doppler studies could be confirmatory if there is vascular involvement. The Doppler studies should reveal the degree and type of vascular involvement; they may help localize the pathology as well.^{10,30,31} A significant decrease in signal with a bracing maneuver (abduction) would suggest compression at the level of the smaller pectoral muscle, whereas with Adson's maneu-

ver, it would suggest the scalene muscles and, with costoclavicular maneuver, it would implicate a problem between the first rib and the clavicle³⁰ (Fig 1).

If the findings of the Doppler studies are normal, it may be important to obtain a CT scan or MRI of the axilla, especially if a plain chest x-ray film suggests the possibility of a lesion/mass. It is more difficult to confirm the diagnosis of TOS with anatomic studies, especially because many physicians believe that a positive cut-off with hyperabduction seen on angiograms or venograms (as well as on Doppler studies) can occur in asymptomatic individuals.^{6,12,30} In most cases, it is generally not necessary to perform angiography or venography.^{1,12,21,30}

A recommended initial diagnostic approach includes one screening anatomic study (plain cervical spine and chest x-ray film with apical lordotic views) and one screening physiologic study (thermography, if available; otherwise, electromyography). However, it would still be reasonable to initiate "conservative" treatment (ie, anti-inflammatory agents, antispasmodic agents, physical therapy modalities, manipulation, and stretching exercises) for 2 to 4 weeks before proceeding with any diagnostic tests, especially if the patient's condition is relatively stable. If vascular involvement is suggested clinically, Doppler studies can be added later. If any "hard" objective neurologic abnormality such as loss of reflex or obvious weakness is noted, thermography probably will not be too helpful; instead, an EMG should be obtained. Remember, though, that thermography will indicate abnormality almost immediately, whereas it may take 2 to 4 weeks for pathology to show up on EMGs.

In spite of the foregoing guidelines, however, various conditions exist in clinical situations that will limit which studies can be done. It is not always wise, or possible, to perform all of these diagnostic studies. Each clinician must make a clinical decision as to which examinations have priority.

Report of cases

Case 1

Subsequent to a fall at work in December 1987, a

47-year-old man had pain radiating, on the right side, into his neck, shoulder, anterolateral aspect of the arm, dorsal aspect of his forearm, and the fourth and fifth digits. He also had paresthesia of the entire arm. X-ray films of the chest and scapula showed no abnormality. He was seen by a family physician, who diagnosed his condition as cervicothoracic strain. Treatment consisted of administration of ibuprofen, physical therapy (hot packs, ultrasound, and massage), and manipulation. The man returned to work in February 1988, but continued to have symptoms, even after treatment was discontinued in early May 1988. In September 1988, he was evaluated by an orthopedist, who noted a "positive Adson's maneuver on the right," with paresthesia in the right hand. The diagnosis at this time was mild TOS. Exercises were initiated, resulting in some decrease in symptoms.

The patient was seen by the author in October 1988. Physical examination (which included osteopathic structural examination) revealed local restriction and pain within the right levator, trapezius, scalene, and smaller pectoral muscles. Stress cervical thermography was performed and revealed changes consistent with TOS. The entire right hand (Fig 2, top) had increased heat emission. Additionally, thermograms obtained after stress testing showed increased heat emission in the right lateral aspect of the arm, the dorsal and medial aspects of the forearm, and the ventral lateral aspect of the forearm. Changes consistent with trigger points were also noted in the right suprascapular (trapezius/levator muscle) region.

Treatment was initiated with cyclobenzaprine hydrochloride at bedtime to improve sleep and relax muscles. The patient resumed limited physical therapy, and he began to undergo manipulation with myofascial release treatment. He also started progressive stretching exercises. Myofascial release treatment was initially directed at the suprascapular region, which primarily involved the trapezius, posterior cervical paraspinal, and levator muscles and later included the scalene and pectoral muscles. The patient had progressive improvement, with marked decrease in paresthesia after only four myofascial release treatments and only one treatment directed to the scalene/pectoral muscles. After the fifth treatment (approximately 7 weeks after initiation of treatment), paresthesia was completely relieved. At this point, the patient only occasionally had minimal neck and shoulder pain.

Follow-up thermograms obtained in February 1989 revealed considerable improvement. The extremities were totally symmetric (Fig 2, bottom),

with only some residual increased heat emission from the right suprascapular region, consistent with a trigger area.

Case 1 analysis

The author saw this patient approximately 10 months after an industrial injury with sequelae recently diagnosed as TOS. The patient was performing exercises to relieve the TOS.

Only one additional test was deemed necessary to confirm the orthopedist's diagnosis. After the patient's symptoms cleared, screening thermograms (limited number of sets of images) were obtained again and confirmed objective evidence of resolution of the pathologic condition. These thermograms proved useful in helping to close this Worker's Compensation case.

It was apparent that a myofascial etiology could explain this patient's symptoms; there was objective evidence of myofascial pathology, which, when treated, resulted in resolution of symptoms as well as of the abnormalities noted on diagnostic testing (thermograms). The abnormalities on the thermograms suggested some impairment of venous flow: there was increased heat emission diffusely throughout the entire hand as well as into some of the regions of the forearm and arm. Of course, it is certainly possible that some arterial compression was the predominant feature, with reflex (autonomic) vasodilation and subsequent increase in blood flow into the affected extremity.

One additional consideration is that the entire brachial plexus was irritated and resulted in reflex changes distally, independent of any vascular involvement. Dramatic improvement in symptoms occurred only after both the scalene and pectoral areas were treated by myofascial release technique. Therefore, it can be inferred that the location of the pathology involved both the scalene and pectoral muscles. Of course, it is certainly possible that myofascial release treatment resulted in some improvement in posture, which opened up the space between the clavicle and the first rib, an alternate site of compression. However, this latter site does not appear to be as common a location, and there is somewhat more controversy concerning its involvement.¹⁴

Case 2

A 21-year-old woman was severely jolted and "bounced around," and struck her head against the top of the car in a roll-over motor vehicle accident in March 1989. Subsequently, she had right-sided neck, shoulder, and upper-extremity pain that involved the arm, forearm, and third digit. She also had paresthesia involving the entire upper extremity. Symptoms were intermittent and were noticed most with upper-extremity elevation. Routine x-ray studies revealed no abnormality.

Physical therapy modalities, manipulation, anti-inflammatory medication, and cyclobenzaprine effected partial improvement. Strengthening exercises were subsequently added, but aggravated symptoms. When seen by the author in May 1989, this patient had some clinical evidence of myofascial pain, right shoulder bursitis with tendinitis (biceps), muscle-contraction cephalgia, and TOS. Osteopathic structural examination revealed abnormal shoulder protraction and restricted second through fourth ribs on the right. Doppler studies revealed marked abnormality with grade 4/4 occlusion during Adson's maneuver, costoclavicular stress maneuver, and upper-extremity hyperabduction bracing (stressing the smaller pectoral muscle area). The results of thermography were abnormal, consistent with TOS, with increased heat emission from the right hand (Fig 3, top) and dorsal aspect of the forearm (after stress testing). Additionally, suboccipital changes on the right side were consistent with possible occipital neuritis, and there were changes suggestive of bursitis or tendinitis (or both) of the right shoulder.

The patient was started on a treatment program consisting of physical therapy modalities, anti-inflammatory medication, and an increased dose of cyclobenzaprine at bedtime. She was placed on a vigorous progressive stretching exercise program. Myofascial release treatment was also used, initially directed to the scalene, pectoral, and latissimus muscles. After one myofascial release treatment, the patient noticed essentially complete resolution of paresthesia and pain in the upper extremity. Slight, partial symptoms recurred; exacerbation was thought to be related to biceps tendinitis and occipital neuritis, with secondary exaggeration of postural abnormality (increased trunk flexion with protracted scapula). Methylprednisolone acetate (40 mg/mL, 2 mL total) was injected suboccipitally and about the right shoulder (biceps). As the occipital neuritis and tendinitis resolved, the patient became more responsive to progressive stretching and myofascial release treatment.

Figure 2. Case 1: Thermographic images of hands with color/temperature scale at bottom. Each color represents 1° C, and becomes progressively warmer from left to right, with white being hottest. Top: Images demonstrate pretreatment dorsal (left) and ventral (right) aspects of hands. Notice marked increase in heat emission from right hand with more pink and red versus the cooler (blue and pink) left hand. Bottom: Posttreatment, dorsal (left) and ventral (right) aspects of hands are fairly symmetric, correlating well with clinical improvement.

Figure 3. Case 2: Thermographic images of hands with color/temperature scale at bottom. Each color represents 1° C, and becomes progressively warmer from left to right, with white being hottest. Top: Dorsal (left) and ventral (right) aspects of hands before treatment. Notice increased heat emission from right hand, with more pink and red versus cooler (blue and pink) left hand. Bottom: Posttreatment, dorsal (left) and ventral (right) aspects of hands are symmetric, correlating well with clinical improvement.

Marked improvement was noted, which persisted after the fourth treatment. Other muscles treated included the trapezius, levator, sternomastoid, and the deep anterior/posterior cervical myofascial structures. Local restriction persisted at the fourth rib anteriorly and was released. Follow-up ther-

mograms obtained approximately 6 weeks after the initiation of treatment revealed marked improvement, with completely symmetric upper extremities (Fig 3, bottom). In fact, the only abnormality remaining consisted of mild changes suboccipitally, consistent with trigger areas.

Figure 4. Case 3: Thermographic images of forearms and hands with color/temperature scale at bottom. Each color represents 1° C, and becomes progressively warmer from left to right, with white being hottest. Top: Dorsal aspect of forearms. Bottom: Dorsal (left) and ventral (right) aspects of hands. Notice decreased heat emission from symptomatic dorsal aspect of left forearm with more blue and pink versus warmer (pink and red) dorsal aspect of right forearm. Dorsal aspect of left thumb is also cooler, with more blue versus warmer (pink) right thumb. Ventral aspect of left third digit is warmer, with more pink versus cooler (blue) right third digit.

Figure 5. Case 4: Thermographic images of hands with color/temperature scale at bottom. Each color represents 1° C, and becomes progressively warmer from left to right, with white being hottest. Dorsal aspect of hands (left) and ventral aspect of hands (right). Notice that there is increased heat emission from all digits on dorsal aspect of left hand, with more pink versus cooler (blue) on right hand. Ventral aspect of first three digits of left hand are also warmer, with more pink and blue versus cooler (more blue and light blue) first three digits of right hand.

Case 2 analysis

When seen by the author, this patient had significant abnormalities, suggestive of some circulatory compromise, revealed on both thermograms (venous) and Doppler studies (arterial). Heat emission was increased throughout the entire right hand, but this increase could be consistent with reflex changes, as seen in the patient in case 1. However, the patient had symptoms for only approximately 2

months. In addition, because of the diagnostic study results (suggesting vascular involvement) and rapid response to initial treatment, it appeared unlikely that she had neurogenic involvement. Therefore, no electromyographic examination was done. Suspected sites of compression included the scalene and pectoral regions for reasons similar to those in case 1. Involvement of these regions is further supported by the results of the Doppler studies,

which showed abnormality with positioning (Adson's and braced abduction maneuvers) that increased compressive forces throughout both sites. In addition, the restriction that involved the second through the fourth ribs would include attachment sites of both the posterior scalene and smaller pectoral muscles.

Interestingly, the patient's symptoms increased after strengthening exercises were initiated, probably because these exercises were performed at the expense of stretching and increasing range of motion. This effect was observed by Joynt³²: "...range can be lost during strengthening and endurance exercise programs if it is not specifically maintained."

Follow-up thermograms near the completion of this patient's treatment program objectively documented resolution of the TOS, with quite dramatic return to symmetry (Fig 3).

Case 3

A 47-year-old woman had pain about the right side of the neck and back as well as headache and upper-extremity paresthesia subsequent to a head-on automobile accident in January 1988. She had been severely jolted, "tossed about," and thrown to the floor. She had no significant improvement after several months' treatment with physical therapy and administration of naproxen and amitriptyline hydrochloride.

This woman was seen by the author in January 1989, at which time structural examination revealed head protrusion anteriorly as in Fig 1C and excessive shoulder protraction. Stress cervical thermograms in February revealed abnormality, consistent with C7 or C6 (or both) nerve irritation. There was decreased heat emission from the dorsal aspect of the left forearm and, after stress, there was decreased heat emission from the dorsal aspect of the left thumb, with increased heat emission from the third digit (Fig 4). Focal increases in heat emission were noted from the right suprascapular region, possibly consistent with musculo-ligamentous injury. Subsequent electromyographic study revealed no abnormality. Findings of Doppler studies were consistent with TOS; there was a grade 4/4 abnormality during Adson's and brace maneuvers (normal blood flow during costoclavicular stress maneuver).

Treatment, which included physical therapy modalities and self-stretching exercises, was essentially ineffective until myofascial release treatment

was used. Myofascial release treatment was directed initially to the scalene muscles and subsequently included the levator, latissimus dorsi, pectoral, and trapezius muscles. The patient noticed significant decrease in upper-extremity symptoms after the third treatment, which included the scalene, levator, and trapezius muscles. After the fifth treatment, which also included the pectoral muscles, she noticed marked improvement. After the sixth treatment, symptoms were almost completely gone and, after the seventh treatment, the patient had no further complaint of upper-extremity symptoms.

Case 3 analysis

This patient's thermograms demonstrated abnormality consistent with cervical nerve irritation within at least one dermatome (but probably two). Because this patient's abnormality appeared to be due more to a neurogenic (and possibly radicular) rather than vascular component, an electromyographic examination was done. Owing to the normal findings on the EMG, Doppler studies were deemed necessary to rule out a vascular component, especially because thoracic outlet stress maneuvers in the clinical examination did reproduce some symptoms.

The findings of the Doppler studies were positive for TOS, and compatible with compressive effects at the level of the brachial plexus, thereby correlating with the findings on the thermograms, which revealed nerve fiber involvement (C7 or C6, or both) and therefore neurogenic TOS. This correlation is more significant if a margin of error for one dermatome level is allowed, thereby involving C7 and C8 nerve fibers (the medial and possibly posterior cord). The normal findings on EMG can lead one to suspect the degree of nerve involvement as only irritative.

Therefore, it could be inferred that the pathologic condition in this patient demonstrated a mixed pattern, with a neurogenic component (based on the thermograms) and a vascular/arterial component (based on the Doppler studies). It can be further inferred that the pathosis included the scalene and pectoral muscles because (1) the Doppler studies were positive with Adson's and brace maneuvers; (2) some improvement occurred after treatment of the

scalene muscles, and marked improvement, after treatment of the pectoral muscles; and (3) the shoulder protraction noted clinically is consistent with a short, restricted smaller pectoral muscle.

Case 4

A 39-year-old woman sustained a whiplash-type injury when her auto was rear-ended in a collision in May 1986. Initial diagnosis was cervical strain. The woman was seen by a chiropractor as well as an orthopedist, and she underwent manipulation combined with physical therapy modalities. Symptoms persisted, and she was seen by an osteopathic physician in late 1987. Treatment included physical therapy, manipulation, massage, and administration of cyclobenzaprine and ibuprofen (Advil). Other medications included naproxen, ibuprofen under the trademarked formulation Motrin, and orphenadrine citrate. Primary symptoms were pain in the left side of the neck and left shoulder with upper-extremity paresthesia and "heaviness." In addition to plain x-ray films, diagnostic testing included a cervical myelogram with CT scan and an electromyographic study, all of which yielded normal findings.

When seen by the author in February 1989, the patient had constant pain in the left shoulder and suprascapular region and decreased sensation in the left upper extremity, which included the lateral aspect of the arm to the elbow, most of the forearm, and hand (all digits). She also had loss of motor control distally (she dropped things), and she had tremendous difficulty at her work as a hair dresser; symptoms increased greatly during arm elevation. Examination revealed most pronounced local restriction and pain within the left scalene, smaller pectoral, levator, and trapezius muscles.

Thermograms were obtained and revealed abnormalities consistent with possible TOS. There was a question of C6 or C7 (or both) nerve irritation. Thermograms showed decreased heat emission throughout the left lateral aspect of the arm and increased heat emission throughout the ventral surface of digits 1 through 3 and the dorsal surface of all five digits (Fig 5). Additionally, there were focal increases in heat emission from the right suboccipital and left parascapular region, consistent with trigger areas. Findings of subsequent Doppler studies were abnormal, consistent with TOS. A grade 4/4 obliteration of pulse occurred with Adson's, costoclavicular, and brace maneuvers.

Treatment was initiated with spray (using di-

chlorodifluoromethane 15%, trichloromonofluoromethane 85%, and subsequently chloroethane and stretch, combined with myofascial release treatment. Self-stretching exercises as well as limited physical therapy modalities were also included in the treatment program. Muscles released initially included the scalene, pectoral, trapezius, latissimus, and subsequently the posterior cervical paraspinal and rhomboid muscles. After the third treatment, there was significant improvement: the patient had a marked decrease in hand pain and she was no longer dropping things or having paresthesia. She was treated on three additional occasions and continued to do well, with only some limited, intermittent pain localized about the cervicothoracic region. Attempts were made to obtain follow-up thermograms; however, this patient was reluctant to pursue additional treatment or testing. This case involved litigation, and it is suspected that the patient, motivated by secondary gain, was reluctant to admit to any improvement.

Case 4 analysis

This case is dramatic: the patient had a history of symptoms that persisted for close to 3 years and that were markedly relieved within only three treatments directed to the involved myofascial regions. When seen by the author, the patient had unexplained symptoms and normal myelogram, CT, and EMG findings. Because litigation was involved, it was essential to confirm a diagnosis if, in fact, it was thought that there was an organic cause for her complaints.

The location of pathology inferred from diagnostic testing was somewhat mixed. Thermographic abnormalities suggested C6, C7, and possible C8 nerve involvement. In addition, a vascular component (particularly venous) could have been inferred, in that all five digits demonstrated increased heat emission. However, the fact that the entire hand was not involved (without forearm involvement either) would probably be more suggestive of the aforementioned reflex changes.

Doppler studies revealed significant compressive effect in all stress positions, consistent with probable compromise at the scalene as well as the pectoral sites. This compromise would be consistent with the findings of the clinical examination (restriction and pain in-

volving both muscles) and the treatment response noted after release of both areas as well as of the surrounding involved muscles.

Summary of case analysis

Most of these patients appear to have significant involvement with compressive or constrictive effects at both the scalene and smaller pectoral muscle regions. This involvement is consistent with observations of Travell and Simons⁹; they noted a so-called "double entrapment," which could represent a myofascial equivalent of the "double crush syndrome" described in the electromyographic literature.³³ Thermography was helpful in all four of the cases presented here in confirming and objectifying the diagnosis. Corroboration of the clinical impression with thermograms and Doppler studies greatly increased the diagnostic confidence. In addition, thermograms corroborated clinical improvement in the two patients who had thermograms obtained in posttreatment follow-up. Perhaps, it would be useful, if not interesting, to do posttreatment follow-up Doppler studies as well.

The author did not see most of these patients until after several months or years of ongoing symptoms. In some of the cases, the patient or the referring physician (or both) was interested in surgical treatment (first rib resection) because of a "failed" response to previous therapy. In each of these cases, however, there had been no prior treatment program that had been specifically directed to the involved myofascial regions with either a vigorous, progressive stretching regimen or a specific myofascial release treatment.

Discussion

Diagnosis

Thoracic outlet syndrome can be a painful, debilitating condition, as well as a great source of frustration for physicians and patients. Many patients lose their livelihood because use of the upper extremities is dramatically limited, especially with the hand at or above chest level. Interestingly, TOS appears to develop after even only minor trauma.^{1,2} Unfortunately, the diagnosis is often overlooked, incredibly, for months or years,³ possibly because

of unavailability or unawareness of appropriate diagnostic tests as well as skepticism regarding the existence of the disease process itself. It is probably much more common than previously suspected, especially in a mild form. Woods³ reported that of 1958 patients with soft-tissue cervical injuries, 458 (23%) had TOS diagnosed—"a higher incidence than has been previously reported....in this series, 72% of the patients with TOS were originally diagnosed incorrectly."

In 1968, Urschel and associates¹⁹ highlighted the importance of accurate diagnosis: "Improvement in diagnostic acuity of the thoracic outlet syndrome is imperative to obtaining good therapeutic results." However, at that time, they were advocating the use of either arteriography, venography, or plethysmography.

From an osteopathic medicine standpoint, some physicians may find it useful to examine the mobility of the first through the fifth ribs. Because the scalene muscles attach to the first and second ribs and the smaller pectoral muscle attaches to the third, fourth, and fifth ribs, involvement of any of these muscles would be expected to cause limited exhalation excursion (restricted into inhalation) of the related ribs. In addition, findings noted on Doppler studies that implicate specific muscle involvement may correlate with specific rib restriction to further substantiate a scalene or pectoral etiology (or both) of TOS.

Treatment

Several authors^{7,11,14,19,22} have mentioned that the first line of treatment of TOS should be conservative. Several have described exercises for stretching the scalene and pectoral muscles^{8,9,18} as well as use of anti-inflammatory medication, muscle relaxants, and physical therapy.^{3,11,18,19,21,22} Some authors have observed that the prognosis is good, although relapse is common.¹⁸ There is no doubt that some individuals probably need to continue at least a low level of stretching exercises indefinitely. Simple recurrence of mild upper-extremity symptoms is not necessarily an indication for the need for surgical treatment. Many authors^{1,11,13,20,21} have said that conservative treatment should be used unless there is "sig-

(continued on page 703)

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BRIEF SUMMARY

Contraindications: Severe LV dysfunction (see *Warnings*), hypotension (systolic pressure < 90 mm Hg) or cardiogenic shock, sick sinus syndrome (if no pacemaker is present), 2nd- or 3rd-degree AV block (if no pacemaker is present), atrial flutter/fibrillation with an accessory bypass tract (eg, WPW or LGL syndromes), hypersensitivity to verapamil.

Warnings: Verapamil should be avoided in patients with severe LV dysfunction (eg, ejection fraction < 30%) or moderate to severe symptoms of cardiac failure and in patients with any degree of ventricular dysfunction if they are receiving a beta-blocker. Control milder heart failure with optimum digitalization and/or diuretics before Calan SR is used. Verapamil may occasionally produce hypotension. Elevations of liver enzymes have been reported. Several cases have been demonstrated to be produced by verapamil. Periodic monitoring of liver function in patients on verapamil is prudent. Some patients with paroxysmal and/or chronic atrial flutter/fibrillation and an accessory AV pathway (eg, WPW or LGL syndromes) have developed an increased antegrade conduction across the accessory pathway bypassing the AV node, producing a very rapid ventricular response or ventricular fibrillation after receiving I.V. verapamil (or digitalis). Because of this risk, oral verapamil is contraindicated in such patients. AV block may occur (2nd- and 3rd-degree, 0.8%). Development of marked 1st-degree block or progression to 2nd- or 3rd-degree block requires reduction in dosage or, rarely, discontinuation and institution of appropriate therapy. Sinus bradycardia, 2nd-degree AV block, sinus arrest, pulmonary edema and/or severe hypotension were seen in some critically ill patients with hypertrophic cardiomyopathy who were treated with verapamil.

Precautions: Verapamil should be given cautiously to patients with impaired hepatic function (in severe dysfunction use about 30% of the normal dose) or impaired renal function, and patients should be monitored for abnormal prolongation of the PR interval or other signs of overdosage. Verapamil may decrease neuromuscular transmission in patients with Duchenne's muscular dystrophy and may prolong recovery from the neuromuscular blocking agent vecuronium. It may be necessary to decrease verapamil dosage in patients with attenuated neuromuscular transmission. Combined therapy with beta-adrenergic blockers and verapamil may result in additive negative effects on heart rate, atrioventricular conduction and/or cardiac contractility; there have been reports of excessive bradycardia and AV block, including complete heart block. The risks of such combined therapy may outweigh the benefits. The combination should be used only with caution and close monitoring. Decreased metoprolol clearance may occur with combined use. Chronic verapamil treatment can increase serum digoxin levels by 50% to 75% during the first week of therapy, which can result in digitalis toxicity. In patients with hepatic cirrhosis, verapamil may reduce total body clearance and extrarenal clearance of digoxin. The digoxin dose should be reduced when verapamil is given, and the patient carefully monitored. Verapamil will usually have an additive effect in patients receiving blood-pressure-lowering agents. Disopyramide should not be given within 48 hours before or 24 hours after verapamil administration.

Concomitant use of flecainide and verapamil may have additive effects on myocardial contractility, AV conduction, and repolarization. Combined verapamil and quinidine therapy in patients with hypertrophic cardiomyopathy should be avoided, since significant hypotension may result. Concomitant use of lithium and verapamil may result in a lowering of serum lithium levels and increased sensitivity to lithium. Patients receiving both drugs must be monitored carefully. Verapamil may increase carbamazepine concentrations during combined use. Rifampin may reduce verapamil bioavailability. Phenobarbital may increase verapamil clearance. Verapamil may increase serum levels of cyclosporin. Concomitant use of inhalation anesthetics and calcium antagonists needs careful titration to avoid excessive cardiovascular depression. Verapamil may potentiate the activity of neuromuscular blocking agents (curare-like and depolarizing); dosage reduction may be required. Adequate animal carcinogenicity studies have not been performed. One study in rats did not suggest a tumorigenic potential, and verapamil was not mutagenic in the Ames test. Pregnancy Category C. There are no adequate and well-controlled studies in pregnant women. This drug should be used during pregnancy, labor, and delivery only if clearly needed. Verapamil is excreted in breast milk; therefore, nursing should be discontinued during verapamil use.

Adverse Reactions: Constipation (7.3%), dizziness (3.3%), nausea (2.7%), hypotension (2.5%), headache (2.2%), edema (1.9%), CHF, pulmonary edema (1.8%), fatigue (1.7%), dyspnea (1.4%), bradycardia: HR < 50/min (1.4%), AV block: total 1°, 2°, 3° (1.2%), 2° and 3° (0.8%), rash (1.2%), flushing (0.6%), elevated liver enzymes. The following reactions, reported in 1.0% or less of patients, occurred under conditions where a causal relationship is uncertain: angina pectoris, atrioventricular dissociation, chest pain, claudication, myocardial infarction, palpitatory purpura (vasculitis), syncope, diarrhea, dry mouth, gastrointestinal distress, gingival hyperplasia, ecchymosis or bruising, cerebrovascular accident, confusion, equilibrium disorders, insomnia, muscle cramps, paresthesia, psychotic symptoms, shakiness, somnolence, arthralgia and rheumatism, hair loss, hyperkeratosis, macules, sweating, urticaria, Stevens-Johnson syndrome, erythema multiforme, blurred vision, gynecomastia, increased urination, spotty menstruation, impotence.

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- Data on file, G.D. Searle & Co.

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nificant vascular compromise or motor loss^{21(p597)} observed, or as long as the patient is improving. Therefore, it would appear that in order for the so-called conservative treatment to be given a fair trial, it should be directed as specifically as possible to the involved structures.²⁰ Treatment should be aggressive and persistent, as tolerated.

The specific treatment protocol involves a stretch and release of the involved muscles as well as some of the surrounding myofascial elements, thus allowing the shoulder girdle to elevate and "set back" to its normal anatomic position. The rapidity with which the patients described here responded to treatment appears to bear out the effectiveness of such a protocol. All patients had essentially complete relief of upper-extremity symptoms within one to four treatments directed to the scalene and pectoral muscles.

After the first few treatments, as upper-extremity symptoms decrease, residual pain often exists about the cervicothoracic region. It is almost as if the condition has reverted to a simple cervicothoracic strain syndrome, with tenderness or trigger points (or both) in those related muscles (eg, trapezius, levator, rhomboid). These more superficial muscles are usually involved in most common strain syndromes. It would appear that after a simple strain (perhaps secondary to mild trauma), the superficial muscles gradually and progressively tighten, drawing the head and neck anteriorly-inferiorly as the shoulder girdle protracts (Fig 1). This muscle tightening and shoulder protraction sets the position to allow the deeper muscles to shorten adaptively, further narrowing the neurovascular space (scalene and pectoral sites), thereby resulting in upper-extremity symptoms. As the deeper muscles shorten, they often activate trigger points within them as well, further increasing local and referred pain.

Early vigorous treatment (including stretching and release) might abort the whole process leading to TOS. However, each individual patient has his or her own relative state of muscle tension and predisposing posture/structure before the trauma or aggravating situation occurs. This predisposition, compounded by the

pain of acute trauma, often results in rapid progression of muscle shortening and subsequent development of TOS, which becomes difficult to treat. In addition, many practitioners are unaware of specific aggressive nonsurgical treatment techniques.

Conclusion

Thoracic outlet syndrome is a relatively common problem. It appears to develop after trauma as a result of progressive increase in myofascial tension throughout the cervicothoracic region with subsequent postural or structural changes. It may be appropriate to refer to many cases as "functional thoracic outlet syndrome," because the actual constellation of upper-extremity symptoms that define TOS should be regarded as temporary, easily reversed by simple corrective muscle stretch and release, with postural improvement.

Thermography appears to be a sensitive, non-invasive test that can help confirm the diagnosis and identify the pathology involved in TOS. It is a valuable initial screening examination because it provides physiologic data regarding both the vascular and neurologic status. A thermogram showing abnormality can direct the physician to use more aggressive stretching and physical therapy modalities as well as specific direct release techniques, if the latter techniques are available. Follow-up thermograms are useful for monitoring effects of treatment, which is especially helpful in cases involving litigation, secondary gain, or questionable motivation of the patient. When the thermogram reverts to normal or relative symmetry, treatment usually should be tapered, discontinued, or altered, depending on the clinical situation.

Part 2 of this article will appear in the September issue of JAOA. It will focus on a novel treatment method that is rapidly and powerfully effective and will review theoretical considerations behind the treatment. It will also describe self-stretching exercise technique.

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American Osteopathic Association

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