

Medical Treatment Guidelines

Washington State Department of Labor and Industries

Work-Related Neurogenic Thoracic Outlet Syndrome: Diagnosis and Treatment*

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**This guideline does not apply to severe or acute traumatic injury of the upper extremities, nor to vascular categories of TOS.*

Work-Related Neurogenic Thoracic Outlet Syndrome: Diagnosis and Treatment

I. INTRODUCTION

This guideline is to be used by physicians, claim managers, occupational nurses, and utilization review staff. The emphasis is on accurate diagnosis and treatment that is curative or rehabilitative (see [WAC 296-20-01002](#) for definitions). An electrodiagnostic worksheet and guideline summary are appended to the end of this document.

This guideline was developed in 2010 by the Washington State's Industrial Insurance Medical Advisory Committee (IIMAC) and its subcommittee on Upper Extremity Entrapment Neuropathies. The subcommittee presented its work to the full IIMAC, and the IIMAC voted with full consensus advising the Washington State Department of Labor & Industries to adopt the guideline. This guideline was based on the weight of the best available clinical and scientific evidence from a systematic review of the literature* and a consensus of expert opinion. One of the Committee's primary goals is to provide standards that ensure high quality of care for injured workers in Washington State.

Thoracic Outlet Syndrome (TOS) is characterized by pain, paresthesias, and weakness in the upper extremity, which may be exacerbated by elevation of the arms or by exaggerated movements of the head and neck. There are three categories of thoracic outlet syndrome: arterial, venous and neurogenic. Arterial and venous thoracic outlet syndromes involve obstruction of the subclavian artery or vein, respectively, as they pass through the thoracic outlet. These vascular categories of TOS should include obvious clinical signs of vascular insufficiency: a cold, pale extremity in the case of arterial TOS, or a swollen, cyanotic extremity in the case of venous TOS. There is a separate surgical guideline for vascular TOS. This guideline focuses solely on non-acute, neurogenic TOS (nTOS).

Work-related nTOS occurs due to compression of the brachial plexus, predominantly affecting its lower trunk, at one of three potential sites. Compression can occur between the anterior and middle scalene muscles (or sometimes through the anterior scalene muscle); beneath the clavicle in the costoclavicular space; or beneath the tendon of the pectoralis minor.¹

The medical literature describes two categories of nTOS: “true” nTOS and “disputed” nTOS. A diagnosis of true nTOS requires electrodiagnostic (EDS) abnormalities showing evidence of brachial plexus injury (see Section III.B.). Disputed nTOS describes cases of nTOS for which EDS abnormalities have not been demonstrated. To avoid confusion that has arisen over these categories, this guideline does not use such terms. Rather, it provides guidance regarding treatment for cases of nTOS that have been confirmed by EDS abnormalities compared with those cases for which the provisional diagnosis has not been confirmed by such studies.

In general, work-relatedness and appropriate symptoms and objective signs must be present for Labor and Industries to accept nTOS on a claim. Electrodiagnostic studies (EDS), including nerve conduction velocity studies (NCVs) and needle electromyography (EMG), should be scheduled immediately to confirm the clinical diagnosis. If time loss extends beyond two weeks or if surgery is requested, completion of EDS is required and does not need prior authorization.

* Evidence was classified using criteria defined by the American Academy of Neurology (see references)

II. ESTABLISHING WORK-RELATEDNESS

Work-related activities may cause or contribute to the development of nTOS.^{2,3} Because simply identifying an association with workplace activities is not, in itself, adequate evidence of a causal relationship, establishing work-relatedness requires all of the following:

1. Exposure: Workplace activities that contribute to or cause nTOS, and
2. Outcome: A diagnosis of nTOS that meets the diagnostic criteria under Section III, and
3. Relationship: Generally accepted scientific evidence, which establishes on a more probable than not basis (greater than 50%) that the workplace activities (exposure) in an individual case contributed to the development or worsening of the condition (outcome).

When the Department receives notification of an occupational disease, the [Occupational Disease & Employment History](#) form is mailed to the worker, employer or attending provider. The form should be completed and returned to the insurer as soon as possible. If the worker's attending provider completes the form, provides a detailed history in the chart note, and gives an opinion on causality, he or she may be paid for this (use billing code 1055M). Additional billing information is available in the [Attending Doctor's Handbook](#).

Symptoms of nTOS may be exacerbated by certain work-related activities, usually involving elevation or sustained use of the arms. Such activities may include but are not limited to the following⁴:

Lifting overhead	Holding tools or objects above shoulder level
Reaching overhead	Carrying heavy weights

Several occupations have been associated with nTOS. This is **not** an exhaustive list and is meant only as a guide in the consideration of work-relatedness:

Dry wall hanger or plasterer	Assembly line inspector
Welder	Shelf stocker
Beautician	Dental hygienist

III. MAKING THE DIAGNOSIS

A. SYMPTOMS AND SIGNS

A case definition of confirmed nTOS includes appropriate symptoms, objective physical findings ("signs"), and abnormal EDS. A provisional diagnosis of nTOS may be made based upon appropriate symptoms and objective signs, but confirmation of the diagnosis requires abnormal EDS.

Classic symptoms of nTOS include pain, paresthesias, or weakness in the upper extremity. Paresthesias most commonly affect the ring and small fingers.⁵ Symptom severity tends to increase after certain activities and worsens at the end of the day or during sleep.

Signs on examination may include tenderness to palpation over the brachial plexus, the scalene muscles, the trapezius muscles, or the anterior chest wall. Although tenderness may be a useful objective finding, it cannot support the diagnosis of nTOS alone. Advanced cases of nTOS are characterized by objective signs of weakness of the hand, loss of dexterity of the fingers, and atrophy of the affected muscles.

Provocative tests have been described that may help corroborate the diagnosis of nTOS. These tests are based on creating maximal tension on the anatomical sites of constriction. Studies have found a high false-positive rate for these tests in healthy subjects as well as carpal tunnel syndrome patients.⁶ Although they are described for

completeness, the sensitivity and specificity of these tests for nTOS have not been established, and these tests cannot replace confirmatory EDS testing (see Section III.B).

Provocative tests include:

- The elevated arm stress test (EAST or Roos test)- the patient places the affected arm in full abduction and external rotation and then opens and closes the hands slowly for 3 minutes. This test constricts the costoclavicular space. It is considered abnormal if typical symptoms are elicited and the patient cannot sustain this activity for the full 3 minutes.
- The Adson test- the patient extends the neck and rotates the head toward the involved extremity, which is held extended at the side. This test constricts the interscalene triangle. It is considered abnormal if a change in the radial pulse is detected when the patient inhales deeply and holds their breath
- The Wright test- the patient sits or stands with the arm in full abduction and external rotation. This test constricts the costoclavicular space. It is considered abnormal if typical symptoms are elicited and a change in pulse is detected.
- The costoclavicular test- the examiner depresses the patient's shoulder. This test constricts the costoclavicular space and creates tension across the pectoralis minor. It is considered abnormal if typical symptoms are elicited.

Every effort should be made to objectively confirm the diagnosis of nTOS before considering surgery. A differential diagnosis for nTOS includes musculoskeletal disease (e.g. arthritis, tendinitis) of the cervical spine, shoulder girdle or arm, cervical radiculopathy or upper extremity nerve entrapment⁷, idiopathic inflammation of the brachial plexus (aka Parsonage-Turner syndrome), and brachial plexus compression due to an infiltrative process or space-occupying mass (e.g. Pancoast tumor of the lung apex).

B. ELECTRODIAGNOSTIC STUDIES (EDS)

EDS abnormalities are required to objectively confirm the diagnosis of nTOS. Given the uncertainties in diagnostic assessment of nTOS, EDS should be obtained as soon as the diagnosis is considered. EDS may help gauge the severity of injury.⁸⁻¹⁰ Importantly, EDS can help exclude conditions that may mimic nTOS, such as ulnar nerve entrapment or cervical radiculopathy.¹¹ EDS evidence that confirms a diagnosis of nTOS requires:

1. Absent or reduced amplitude (< 12 uV) of the ulnar antidromic sensory nerve action potential (SNAP)

Or

Absent or reduced amplitude (< 10 uV) of the medial antebrachial cutaneous nerve (MABC) antidromic SNAP, with normal amplitude of the MABC SNAP in the contralateral (unaffected) extremity

AND

2. Absent or reduced amplitude (< 5 mV) of the median nerve compound motor action potential (CMAP)

Or

Absent or prolonged minimum latency (> 33 msec) of the ulnar F-wave (with or without abnormalities of the median F-wave), and with normal F-waves in the contralateral (unaffected) upper extremity

Or

Needle electromyography (EMG) showing denervation (e.g. fibrillation potentials, positive sharp waves) in at least one muscle supplied by each of two different nerves from the lower trunk of the brachial plexus, with normal EMG of the cervical paraspinal muscles and at least one muscle supplied by a nerve from the middle or upper trunk of the brachial plexus.

AND

To exclude the presence of other focal neuropathies or polyneuropathy as a cause for the abnormalities described above, the following must also be shown:

3. Normal amplitude (≥ 15 uV) of the median nerve antidromic SNAP.

AND

4. Normal conduction velocity (≥ 50 m/s) of the ulnar motor nerve across the elbow.

C. OTHER DIAGNOSTIC TESTS

Arterial or venous vascular studies may be helpful in the diagnosis of suspected arterial or venous TOS. However, these tests have poor specificity for nTOS, and there is no substantial evidence that vascular studies can reliably confirm the diagnosis of nTOS. Therefore, vascular studies conducted as a diagnostic tool for nTOS will not be authorized.

Some have suggested that magnetic resonance imaging (MRI) neurography may be helpful in the diagnosis of nTOS. However, these services will not be authorized for this condition because the clinical utility of these tests has not yet been proven. While the Committee recognizes that these tests may be useful in unusual circumstances where EDS results are normal but there are appropriate clinical symptoms, the Committee believes that at this time the use of these tests is investigational and should be used only in a research setting.

Anterior scalene muscle (ASM) blocks have been used in the evaluation of suspected nTOS.^{12,13} However, this test has poor specificity for nTOS, and there is no substantial evidence that ASM can reliably confirm the diagnosis of nTOS. Therefore, ASM blocks conducted as a diagnostic tool for nTOS will not be authorized.

X-rays of the chest may be useful to evaluate the possibility of an infiltrative process or space-occupying mass (e.g. Pancoast tumor of the lung apex) compressing the brachial plexus.

IV. TREATMENT

Non-surgical therapy may be considered for cases in which a provisional diagnosis of nTOS has been made. Surgical treatment should be provided only for cases in which the diagnosis of nTOS has been confirmed by abnormal EDS. Under these circumstances, the potential benefits of brachial plexus decompression may outweigh the risks of surgery.

A. CONSERVATIVE TREATMENT

Conservative treatment for nTOS has been described in narrative reviews, case reports, and retrospective case series.¹⁴⁻¹⁶ No randomized controlled trials have been conducted to measure the efficacy of conservative treatments for nTOS. No specific method of conservative treatment has been proven to be most effective due to a lack of comparative studies.¹⁴ However, an observational study (n=50), showed that strengthening and stretching exercises reduced pain among 80% of patients after 3 months and among 94% of patients after 6 months¹⁵, and a 2007 systematic review of the available literature concluded that conservative treatment appears to be effective in reducing symptoms, improving function, and facilitating return to work.¹⁴ Examples of conservative treatment include modification of activities that exacerbate symptoms, education, postural exercises, physical therapy, and anti-inflammatory drug therapy.

Because surgical outcomes are poor in many situations, conservative interventions, such as stretching and strengthening exercises, should be considered first. If the initial response to conservative treatment is incomplete, modifying or changing the approach should be considered. If there is no response to conservative treatment within six weeks, or if time loss extends longer than 2 weeks, specialist consultation should be obtained.

Although Botulinum toxin (Botox) injections of the scalene muscles have been reported to relieve nTOS symptoms¹⁷, preliminary results of a randomized trial showed no clear clinical improvement related to this treatment.¹⁸ In addition, it appears that there are substantial technical challenges and potentially severe adverse effects from this procedure. Therefore, Botox injections conducted as a diagnostic tool or for treatment of nTOS will not be authorized.

When feasible, job modifications that reduce the intensity of manual tasks may prevent progression and promote recovery from nTOS.¹⁶ If symptoms persist despite appropriate treatment, permanent job modifications may still

allow the patient to remain at work. Patients do not usually need time off from work activities prior to surgery, unless they present with objective weakness or sensory loss in the upper extremity that limits work activities or poses a substantial safety risk.

B. SURGICAL TREATMENT

Surgical treatment for nTOS has been described in narrative reviews, case reports, and retrospective case series.^{4,19-34} Surgery should include exploration of the brachial plexus throughout its course in the thoracic outlet in order to decompress it by resecting any compressive and/or constrictive structures. These may include any of the three sites of compression mentioned earlier. No specific method of surgical treatment has been proven to be most effective.

Surgical treatment should only be considered if:

1. The patient has met the diagnostic criteria under Section III, and
2. The condition interferes with work or activities of daily living, and
3. The condition does not improve despite conservative treatment.

Without confirmation of nTOS **by both objective clinical findings and abnormal EDS**, surgery will not be authorized.

V. RETURN TO WORK (RTW)

A. EARLY ASSESSMENT

Timeliness of the diagnosis can be a critical factor influencing RTW. Among workers with upper extremity disorders, 7% of workers account for 75% of the long-term disability.³⁵ A large prospective study in the Washington State workers' compensation system identified several important predictors of long-term disability: low expectations of return to work (RTW), no offer of a job accommodation, and high physical demands on the job.³⁶ Identifying and attending to these risk factors when patients have not returned to work within 2-3 weeks of the initial clinical presentation may improve their chances of RTW.

Washington State workers diagnosed accurately and early were far more likely to RTW than workers whose conditions were diagnosed weeks or months later. Early coordination of care with improved timeliness and effective communication with the workplace is also likely to help prevent long-term disability.

A recent quality improvement project in Washington State has demonstrated that delivering medical care according to occupational health best practices similar to those listed in Table 1 can substantially prevent long-term disability. Findings can be viewed at:

<http://www.lni.wa.gov/ClaimsIns/Files/Providers/ohs/CoheSummaryFindings1207.pdf>.

Table 2. Occupational Health Quality Indicators for Neurogenic Thoracic Outlet Syndrome (nTOS)

Clinical care action	Time-frame*
1. Identify physical stressors from both work and non-work activities; 2. Screen for presence of nTOS 3. Determine work-relatedness 4. Recommend ergonomic improvements or other appropriate job modifications	1 st health care visit
Communicate with employer regarding return to work (RTW) using 1. Activity Prescription Form (or comparable RTW form) and/or 2. Phone call to employer	Each visit while work restrictions exist
1. Assess impediments for RTW 2. Request specialist consultation	If > 2 weeks of time-loss occurs or if there is no clinical improvement within 6 weeks of conservative treatment
Specialist consultation	Performed ASAP, within 3 weeks of request
Electrodiagnostic studies	If the diagnosis of nTOS is being considered, schedule studies immediately. These tests are required if time-loss extends beyond 2 weeks, or if surgery is requested.
Surgical decompression	Performed ASAP, within 4-6 weeks of determining need for surgery

*“Time-frame” is anchored in time from 1st provider visit related to nTOS symptoms.

B. RETURNING TO WORK FOLLOWING SURGERY

How soon a patient can return to work depends on the type of surgery performed and when rehabilitation begins. Most patients can return to light duty work within 4-6 weeks and regular duty within 10-12 weeks of surgery.

VI. ELECTRODIAGNOSTIC WORKSHEET

Claim Number: _____

Claimant Name: _____

PURPOSE AND INSTRUCTIONS

The purpose of this worksheet is to help interpret electrodiagnostic studies (EDS) done for an injured worker. The worksheet should be used only when the main purpose of the study is to evaluate neurogenic thoracic outlet syndrome (nTOS). It should accompany but not replace the detailed report normally submitted to the insurer.

Electrodiagnostic Worksheet for Work-Related Neurogenic Thoracic Outlet Syndrome (nTOS)

Electrodiagnostic criteria for Work-Related nTOS are met if all four boxes are "Yes".	Check the correct box	
	Yes	No
1. Ulnar SNAP* < 12 uV or absent? <div style="text-align: center;">OR</div> ----- Medial antebrachial cutaneous nerve (MABC) SNAP* amplitude < 10 uV or absent, with normal amplitude of the MABC SNAP* in the contralateral (unaffected) extremity? <div style="text-align: center;">AND</div>		
2. Median nerve CMAP amplitude < 5 mV or absent? <div style="text-align: center;">OR</div> ----- Ulnar F-wave (with or without abnormalities of the median F-wave) minimum latency > 33 msec or absent, with normal F-waves in the contralateral (unaffected) upper extremity? <div style="text-align: center;">OR</div> ----- Needle electromyography (EMG) showing denervation (e.g. fibrillation potentials, positive sharp waves) in at least one muscle supplied by each of two different nerves from the lower trunk of the brachial plexus, with normal EMG of the cervical paraspinal muscles and at least one muscle supplied by a nerve from the middle or upper trunk of the brachial plexus? <div style="text-align: center;">AND</div>		
3. Normal amplitude ($\geq 15\mu\text{V}$) of the median nerve SNAP*? <div style="text-align: center;">AND</div>		
4. Normal conduction velocity ($\geq 50 \text{ m/s}$) of the ulnar motor nerve across the elbow?		

*Antidromic

Additional Comments:

Signed

Date

VII. GUIDELINE SUMMARY

Review Criteria for the Diagnosis and Treatment of Work-Related Neurogenic Thoracic Outlet Syndrome (nTOS)				
CLINICAL FINDINGS			CONSERVATIVE TREATMENT	SURGICAL TREATMENT
SUBJECTIVE (Symptoms)	OBJECTIVE (Signs)	DIAGNOSTIC		
Pain, paresthasias, or weakness affecting the upper extremity (most commonly affecting the ring or small finger)	<p>AND</p> <p>Tenderness Scalene Trapezius Anterior chest wall Brachial plexus</p> <p>Weakness</p> <p>Loss of finger dexterity</p> <p>Atrophy</p>	<p>AND</p> <p>Electrodiagnostic studies (EDS) are required to objectively confirm the diagnosis of nTOS.</p> <p><u>EDS criteria are as follows:</u></p> <p>1. Absent or reduced amplitude (< 12 uV) of the ulnar SNAP OR Absent or reduced amplitude (< 10 uV) of the medial antebrachial cutaneous nerve (MABC) SNAP with normal amplitude of the MABC SNAP in the contralateral (unaffected) extremity AND 2. Absent or reduced amplitude (< 5 mV) of the median CMAP OR Absent or prolonged minimum latency (>33 msec) of the ulnar F-wave (with or without abnormalities of the median F-wave), and with normal F-waves in the contralateral (unaffected) upper extremity OR Needle electromyography (EMG) showing denervation (e.g. fibrillation potentials, positive sharp waves) in at least one muscle supplied by each of two different nerves from the lower trunk of the brachial plexus, with normal EMG of the cervical paraspinal muscles and at least one muscle supplied by a nerve from the middle or upper trunk of the brachial plexus AND 3. Normal amplitude ($\geq 15\mu\text{V}$) of the median nerve SNAP AND 4. Normal conduction velocity ($\geq 50\text{m/s}$) of the ulnar motor nerve across the elbow</p>	<p>Modify job activities that exacerbate symptoms</p> <p>AND/OR</p> <p>Physical therapy with strengthening and stretching, postural exercises</p> <p>AND/OR</p> <p>Anti-inflammatory drug therapy</p>	<p>Surgical treatment should only be considered if:</p> <p>1. The patient has met the diagnostic criteria under Section III</p> <p>AND</p> <p>2. The condition interferes with work or activities of daily living</p> <p>AND</p> <p>3. The condition does not improve despite conservative treatment</p> <p>Without confirmation of brachial plexus compression by both objective clinical findings and abnormal EDS, surgery will not be authorized.</p>

References

Evidence was classified using criteria defined by the American Academy of Neurology[†]

1. Watson LA, Pizzari T, Balster S. Thoracic outlet syndrome part 1: clinical manifestations, differentiation, and treatment pathways. *Manual Therapy* 2009;14:586-595. *Narrative Review*
2. Sanders RJ, Hammond SL. Etiology and pathology. *Hand Clin* 2004;20(1):23-6. *Narrative Review*
3. Pascarelli EF, Hsu YP. Understanding work-related upper extremity disorders: clinical findings in 485 computer users, musicians, and others. *J Occup Rehabil* 2001;11(1):1-21. *IV*
4. Landry GJ, Moneta GL, Taylor LM, Jr., Edwards JM, Porter JM. Long-term functional outcome of neurogenic thoracic outlet syndrome in surgically and conservatively treated patients. *J Vasc Surg* 2001;33(2):312-7; discussion 317-9. *IV*
5. Brantigan CO, Roos DB. Diagnosing thoracic outlet syndrome. *Hand Clin* 2004;20:27-36. *Narrative Review*
6. Nord KM, Kapoor P, Fisher J, Thomas G, Sundaram A, Scott K, Kothari MJ. False positive rate of thoracic outlet syndrome diagnostic maneuvers. *Electromyogr Clin Neurophysiol* 2008;48(2):67-74. *III*
7. Seror P. Symptoms of thoracic outlet syndrome in women with carpal tunnel syndrome. *Clin Neurophysiol* 2005;116(10):2324-9. *IV*
8. Machanic BI, Sanders RJ. Medial antebrachial cutaneous nerve measurements to diagnose neurogenic thoracic outlet syndrome. *Ann Vasc Surg* 2008;22(2):248-54. *III*
9. 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(10):2316-22. *IV*
10. Tolson TD. EMG for thoracic outlet syndrome. *Hand Clin* 2004;20:37-42. *Narrative Review*
11. Rousseff R, Tzvetanov P, Valkov I. Utility (or futility?) of electrodiagnosis in thoracic outlet syndrome. *Electromyogr Clin Neurophysiol* 2005;45(3):131-3. *IV*
12. Torriani M, Gupta R, Donahue DM. Sonographically guided anesthetic injection of anterior scalene muscle for investigation of neurogenic thoracic outlet syndrome. *Skeletal Radiol* 2009;38:1083-1087. *IV*
13. Jordan SE, Machleder HI. Diagnosis of thoracic outlet syndrome using electrophysiologically guided anterior scalene blocks. *Ann Vasc Surg* 1998;12(3):260-4. *IV*
14. Vanti C, Natalini L, Romeo A, Tosarelli D, Pillastrini P. Conservative treatment of thoracic outlet syndrome. *Eura Medicophys* 2007;43:55-70. *Systematic Review*
15. Hanif S, Tassadaq N, Rathore MF, Rashid P, Ahmed N, Niazi F. Role of therapeutic exercises in neurogenic thoracic outlet syndrome. *J Ayub Med Coll Abbottabad* 2007;19(4):85-8. *III*
16. Crosby CA, Wehbe MA. Conservative treatment for thoracic outlet syndrome. *Hand Clin* 2004;20:43-49. *Narrative Review*
17. Jordan SE, Ahn SS, Gelabert HA. Combining ultrasonography and electromyography for botulinum chemodenervation treatment of thoracic outlet syndrome: comparison with fluoroscopy and electromyography guidance. *Pain Physician* 2007;10(4):541-6. *IV*

[†] Edlund W, Gronseth G, So Y, Franklin G. Clinical Practice Guideline Process Manual. American Academy of Neurology 2004 . (www.aan.com).

18. Travlos A. Treatment of thoracic outlet syndrome with botulinum toxin Injection: a double-blind, randomized controlled trial. . University of British Columbia, 2007. Available at: <http://clinicaltrials.gov/ct2/show/study/NCT00444886?view=results>. Not yet published.
19. Povlsen B, Belzberg A, Hansson T, Dorsi M. Treatment for thoracic outlet syndrome (review). Cochrane Database of Systematic Reviews 2010(1):Art. No.: CD007218. DOI: 10.1002/14651858.CD007218.pub2. *III*
20. Chang DC, Rotellini-Coltvet LA, Mukherjee D, De Leon R, Freischlag JA. Surgical intervention for thoracic outlet syndrome improves patient's quality of life. J Vasc Surg 2009;49(3):630-5; discussion 635-7. *IV*
21. Chang DC, Lidor AO, Matsen SL, Freischlag JA. Reported in-hospital complications following rib resections for neurogenic thoracic outlet syndrome. Ann Vasc Surg 2007;21(5):564-70. *Observational Study*
22. Abdellaoui A, Atwan M, Reid F, Wilson P. Endoscopic assisted transaxillary first rib resection. Interact Cardiovasc Thorac Surg 2007;6(5):644-6. *IV*
23. Colli BO, Carlotti CG, Jr., Assirati JA, Jr., Marques W, Jr. Neurogenic thoracic outlet syndromes: a comparison of true and nonspecific syndromes after surgical treatment. Surg Neurol 2006;65(3):262-71; discussion 271-2. *IV*
24. Krishnan KG, Pinzer T, Schackert G. The transaxillary approach in the treatment of thoracic outlet syndrome: a neurosurgical appraisal. Zentralbl Neurochir 2005;66(4):180-9. *IV*
25. Altobelli GG, Kudo T, Haas BT, Chandra FA, Moy JL, Ahn SS. Thoracic outlet syndrome: pattern of clinical success after operative decompression. J Vasc Surg 2005;42(1):122-8. *IV*
26. Sanders RJ, Hammond SL. Supraclavicular first rib resection and total scalenectomy: technique and results. Hand Clin 2004;20(1):61-70. *Narrative Review*
27. Samarasam I, Sadhu D, Agarwal S, Nayak S. Surgical management of thoracic outlet syndrome: a 10-year experience. ANZ J Surg 2004;74(6):450-4. *IV*
28. Nannapaneni R, Marks SM. Neurogenic thoracic outlet syndrome. Br J Neurosurg 2003;17(2):144-8. *IV*
29. Maxey TS, Reece TB, Ellman PI, Tribble CG, Harthun N, Kron IL, Kern JA. Safety and efficacy of the supraclavicular approach to thoracic outlet decompression. Ann Thorac Surg 2003;76(2):396-9; discussion 399-400. *IV*
30. Bhattacharya V, Hansrani M, Wyatt MG, Lambert D, Jones NA. Outcome following surgery for thoracic outlet syndrome. Eur J Vasc Endovasc Surg 2003;26(2):170-5. *IV*
31. Balci AE, Balci TA, Cakir O, Eren S, Eren MN. Surgical treatment of thoracic outlet syndrome: effect and results of surgery. Ann Thorac Surg 2003;75(4):1091-6; discussion 1096. *IV*
32. Sharp WJ, Nowak LR, Zamani T, Kresowik TF, Hoballah JJ, Ballinger BA, Corson JD. Long-term follow-up and patient satisfaction after surgery for thoracic outlet syndrome. Ann Vasc Surg 2001;15(1):32-6. *IV*
33. Athanassiadi K, Kalavrouziotis G, Karydakis K, Bellenis I. Treatment of thoracic outlet syndrome: long-term results. World J Surg 2001;25(5):553-7. *IV*
34. Franklin GM, Fulton-Kehoe D, Bradley C, Smith-Weller T. Outcome of surgery for thoracic outlet syndrome in Washington state workers' compensation. Neurology 2000;54(6):1252-7. *III*
35. Hashemi L, Webster B, Clance E, Courtney T. Length of disability and cost of work-related musculoskeletal disorders of the upper extremity. . J Occup Environ Med 1998;40:261-269. *Descriptive Study*

36. Turner J, Franklin G, Fulton-Kehoe D. Early predictors of chronic work disability associated with carpal tunnel syndrome: a longitudinal workers' compensation cohort study. *Am J Ind Med* 2007;50:489-500. *II*

Definitions for Classification of Evidence

Rating of Therapeutic Article	Rating of Diagnostic Article	Rating of Prognostic Article	Rating of Screening Article
<p>Class I: Prospective, randomized, controlled clinical trial with masked outcome assessment, in a representative population. The following are required:</p> <ul style="list-style-type: none"> a) primary outcome(s) clearly defined b) exclusion/inclusion criteria clearly defined c) adequate accounting for drop-outs and cross-overs with numbers sufficiently low to have minimal potential for bias d) relevant baseline characteristics are presented and substantially equivalent among treatment groups or there is appropriate statistical adjustment for differences. 	<p>Class I: Evidence provided by a prospective study in a broad spectrum of persons with the suspected condition, using a reference (gold) standard for case definition, where test is applied in a blinded evaluation, and enabling the assessment of appropriate tests of diagnostic accuracy. All patients undergoing the diagnostic test have the presence or absence of the disease determined.</p>	<p>Class I: Evidence provided by a prospective study of a broad spectrum of persons who may be at risk for developing the outcome (e.g. target disease, work status). The study measures the predictive ability using an independent gold standard for case definition. The predictor is measured in an evaluation that is masked to clinical presentation and, the outcome is measured in an evaluation that is masked to the presence of the predictor. All patients have the predictor and outcome variables measured.</p>	<p>Class I. A statistical, population based sample of patients studied at a uniform point in time (usually early) during the course of the condition. All patients undergo the intervention of interest. The outcome, if not objective, is determined in an evaluation that is masked to the patients' clinical presentations.</p>
<p>Class II: Prospective matched group cohort study in a representative population with masked outcome assessment that meets a-d above OR a RCT in a representative population that lacks one criteria a-d.</p>	<p>Class II: Evidence provided by a prospective study of a narrow spectrum of persons with the suspected condition, or a well designed retrospective study of a broad spectrum of persons with an established condition (by "gold standard") compared to a broad spectrum of controls, where test is applied in a blinded evaluation, and enabling the assessment of appropriate tests of diagnostic accuracy.</p>	<p>Class II: Evidence provided by a prospective study of a narrow spectrum of persons at risk for having the condition, or by a retrospective study of a broad spectrum of persons with the condition compared to a broad spectrum of controls. The study measures the prognostic accuracy of the risk factor using an acceptable independent gold standard for case definition. The risk factor is measured in an evaluation that is masked to the outcome.</p>	<p>Class II. A statistical, non-referral clinic-based sample of patients studied at a uniform point in time (usually early) during the course of the condition. Most patients undergo the intervention of interest. The outcome, if not objective, is determined in an evaluation that is masked to the patients' clinical presentation.</p>
<p>Class III: All other controlled trials (including well-defined natural history controls or patients serving as own controls) in a representative population, where outcome is independently assessed, or independently derived by objective outcome measurement.**</p>	<p>Class III: Evidence provided by a retrospective study where either persons with the established condition or controls are of a narrow spectrum, and where the reference standard, if not objective, is applied by someone other than the person that performed the test.</p>	<p>Class III: Evidence provided by a retrospective study where either the persons with the condition or the controls are of a narrow spectrum. The study measures the predictive ability using an acceptable independent gold standard for case definition. The outcome, if not objective, is determined by someone other than the person who measured the predictor.</p>	<p>Class III. A sample of patients studied during the course of the condition. Some patients undergo the intervention of interest. The outcome, if not objective, is determined in an evaluation by someone other than the treating physician.</p>
<p>Class IV: Evidence from uncontrolled studies, case series, case reports, or expert opinion.</p>	<p>Class IV: Any design where test is not applied in an independent evaluation OR evidence provided by expert opinion alone or in descriptive case series (without controls).</p>	<p>Class IV: Any design where the predictor is not applied in an independent evaluation OR evidence provided by expert opinion or case series without controls.</p>	<p>Class IV. Expert opinion, case reports or any study not meeting criteria for class I to III.</p>

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IIMAC Committee Members

Gregory T. Carter MD MS- Chair
Dianna Chamblin MD
G.A. DeAndrea MD MBA
Jordan Firestone, MD PhD MPH
Andrew Friedman MD
Walter Franklin Krenzel III MD
Robert G.R. Lang MD

Subcommittee Clinical Experts

Michel Kliot MD
Mark H. Meissner MD
Lawrence R. Robinson MD
Thomas E. Trumble MD
Michael D. Weiss MD

Consultants:

Terrell Kjerulf MD
Ken O'Bara MD

Department staff who helped develop and prepare this guideline include:

Gary M. Franklin MD, MPH, Medical Director
Lee Glass MD, JD, Associate Medical Director
Simone P. Javaher BSN, MPA, Occupational Nurse Consultant
Reshma N. Kearney MPH, Epidemiologist