Thoracic Outlet Syndrome

The Effectiveness Of Tests And Provocations Used In Diagnosis Of Thoracic Outlet Syndrome: A Literature Review

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Abstract:

This literature review investigates several tests for clinical evaluation of Thoracic Outlet Syndrome. Several authors have evaluated provocative tests. Effectiveness and usefulness of the different research studies is reported. A comparison and contrast of the tests is made and discussed. Tests for evaluation of vascular response show a high number of false positive results in the healthy population. Tests for neurologic response have questionable specificity and sensitivity. Tests for evaluation of neural integrity through nervous stretching have high specificity and reliability but a lack of controlled studies. The impact of the available data on clinical decision-making is discussed, after which suggestions for clinical examination of patients with TOS symptoms are made. Combinations of tests for of TOS result in increased specificity and sensitivity, and are recommended for clinical evaluation of this syndrome.
Introduction:

The diagnosis of Thoracic Outlet Syndrome (TOS) remains a highly controversial topic in healthcare. Much of the controversy relates to the patient with complaints of paresthesias, numbness and pain but without any definitive test to identify the cause. Clinical diagnosis of TOS is currently mostly based on provocative positional and compression tests, and on the reproduction of symptoms. Many of the traditional TOS tests are reported to have low specificity and reliability, as well as a high incidence of false-positive and false-negative findings. Electrodiagnostic tests are useful in ruling out other, more distal nerve entrapments. Radiographic test results are frequently normal in the patient population with TOS symptoms. By contrast, clinical testing with vascular TOS tests is frequently abnormal in the asymptomatic population. A number of clinical, radiographic and electrodiagnostic tests have been described in the literature. However, many tests are considered unreliable, and not one test is regarded as the “gold standard”. The symptomatology is complex by nature of the structures involved (1,2,3). TOS consists of a group of impingement syndromes affecting nerves in the brachial plexus and, less commonly, the arteries, veins and lymph vessels in their passage from cervical spine into the axillary region. Historically the vascular component of TOS appears to have been largely overrated. Only in the last decades has the neurogenic etiology been accepted as the dominant factor in this syndrome.

The purpose of this paper is to provide a better understanding of the TOS syndrome. This literature review will describe the anatomy of the thoracic outlet as well as the different dysfunctions of TOS. Provocative tests of the neural integrity, the vascular integrity and neurovascular integrity are categorized and discussed. Research studies investigating the different tests are described and compared. Diagnostic difficulties for the physical therapist in the clinical
diagnosis of TOS are discussed, after which suggestions for clinical examination and treatment of patients with TOS symptoms are made.

**Anatomy of the Thoracic Outlet:**

The thoracic outlet is the upper lid (operculum) of the chest cage, so named because it is the site from which the arterial flow of the thorax exits out. The thoracic outlet is approximately a four-centimeter area with boundaries anteriorly by the manubrium of the sternum, laterally by the first rib and its costal cartilage, and posteriorly by the body of T1 (4,5,6).

This tightly confined area is covered by Sibson’s fascia and surrounded by the scalene muscles, which border the nerve roots and trunks of the brachial plexus on their route to the upper limb. It is further surrounded by the sternocleidomastoideus muscle and the trapezius muscle which serve as the exit site of the subclavian artery, the subclavian vein and the brachial plexus (i.e. the neurovascular bundle). The rigid osseous boundaries, and strong muscular and ligamentous structures, are the underlying anatomical sources of impingement or compression in the thoracic outlet (4,5,6).

The C4, C5, C6, C7, C8 and T1 spinal nerves exit the cervical foramina and form the three trunks of the brachial plexus. These pass in between the anterior scalenus muscle and the medial scalenus muscle. The subclavian artery accompanies the brachial plexus while the subclavian vein is located in front of the anterior scalenus muscle. The trunks of the brachial plexus divide into anterior and posterior divisions, which travel under the clavicle, in the space formed by the first rib and the clavicle (5,6). The cords then form the peripheral nerves into the upper extremity, namely the musculocutaneous nerve, the axillary nerve, the radial nerve, the median nerve and the ulnar nerve (7).
Common underlying musculoskeletal and neurological impairments of Thoracic Outlet Syndrome:

Several anatomical soft tissue structures can be involved in the thoracic outlet syndrome. Arterial, nerve, vein, or lymphatic system could be compressed, resulting in a variety of signs and symptoms. Signs and symptoms of compression of the anatomical structures mentioned above include pain in the upper extremities, at times with radiating character towards the hands and often only into the fifth digit. At times pain is radiating posteriorly to the head. Paresthesias are felt in dermatomes C2 through C8 and T1. A sensation of weakness during and after activity can be experienced. Because of the compression of structures being mostly intermittent, paralysis, thrombosis and emboli are rarely seen (4).

The subclavian artery is compressed in one to two percent of the cases. The passing of the artery through the interscalene triangle places it at risk for compression by the muscle bodies of the scalene whenever there can be muscular hypertrophy. Patients with asthma bronchiale, chronic obstructive pulmonary disease, hyperventilation, and in practitioners of certain sports as weight lifting and bodybuilding are at risk. Abnormally wide insertions or fusions of insertions of both muscles have also been found to cause compression of the subclavian artery. Compression of the subclavian artery gives rise to ischemia, decreased peripheral pulses, coolness, pallor, and possible cyanosis of the upper extremity. Overhead activities will result in increasing complaints, including a temporary “dropping hand”. Signs and symptoms disappear after rest or change of posture, as with elevation of the shoulders. Possible serious complications of compression of the subclavian artery are micro-emboli, ulcerations and gangrene, which are rarely seen (4,5,6).
Running directly beside the subclavian artery, with the exception of passage anterior of the anterior scalenus insertion, and posteriorly to the sternocleidomastoid muscle insertion, is the subclavian vein. Due to the vein’s escape from very tight inter-muscular passage, venous compression syndromes are rare (three to four percent), and involve swelling in the hands whenever the vein is involved. Cyanosis can be present. Patients complain of a “heavy” sensation in the upper extremity, combined with pain. Serious complications of compression of the subclavian vein are, as with arterial compression, micro-emboli, ulcerations of the upper extremity, and gangrene, which is rarely seen (4,5,6).

The third component of the neurovascular bundle, the brachial plexus, follows the same course as the subclavian artery through the interscalene triangle, only slightly more posterior and lateral. It is the most frequently involved structure in thoracic outlet compression (90-95 % of all cases) and produces paresthesias, anesthesias, pain, weakness, and atrophy in the upper extremity as a result of its compression. Motor deficit is seldom significant but compression of the neurovascular bundle may present as isolated intrinsic muscle atrophy of the hand without any pain, at times referred to as a Gilliat-Sumner hand (5,6). The release-phenomenon, as described by James Cyriax in 1978, can be present. Cyriax was the first to recognize that removal of causative pressure resulted in increase of neurovascular symptoms, thus creating the release-phenomenon (8).
Classification of syndromes:

Thoracic outlet compression syndrome is classified according to the three anatomical compressive regions, which are involved. These regions consist of the interscalene triangle, the costoclavicular space and the subcoracoid region, and give the three different syndromes their names.

The **scalenus anticus syndrome** or **Naffziger’s syndrome** involves compression of the brachial plexus and/or the subclavian artery as these structures pass through the interscalene triangle. Both muscles attach to the first rib and serve as a support structure for the brachial plexus. Muscle hypertrophy, accessory muscles and C7 transversomegaly (abnormally enlarged transverse process) usually impose pressure (8,9).

A variation of the syndrome described above is called the **cervical rib syndrome**. Accessory ribs of varying size, mono- or bilateral, are found in approximately 0.5 to 1.2 percent of the general population. Also 14 types of cervical bands have been described, increasing the risk of compression. The most frequent is the type III band extending from the neck to the scalene tubercle of the first rib, crossing the concavity of the rib (5). The posterior portion of the brachial plexus formed by mainly C8 and T1 is mostly involved in this syndrome. Symptoms of neuropathy most often involve paresthesia in the fourth and fifth fingers, the lateral side of the hand and possibly the forearm, and decreased sensory appreciation of light touch or pinprick in
the 5th digit. Atrophy, hypertonicity and cramping of the finger flexors, as well as weakness can also be found (4).

The second major site of compression is found between the mobile clavicle and the relatively fixed first rib, and is called the **costoclavicular syndrome**. It may be found in the normal athletic male with massive shoulders, or may be seen in any individual who typically has a backwards, downwards thrust of the shoulders as in a military posture. Fractures of the clavicle producing a large callus, congenitally bifid clavicles, posture, or a thickened and tight clavipectoral fascia may compromise the space between first rib and clavicle thus decreasing the available space for passage of the neurovascular bundle (4,5,6,10). Narrowing of the costoclavicular space places pressure on the subclavian artery and vein, and at times on the brachial plexus. The costoclavicular signs and symptoms include coolness, pallor, weakness, paresthesias, diminished distal pulses and edema in the fingers and dorsum of the hand (4,6).

The third syndrome is called the **hyperabduction syndrome**. The potential area of compression is in the subcoracoid region adjacent to the pectoralis minor muscle, or the space where the neurovascular bundle passes through the retroclavicular space, between the clavicle and the first rib. With full circumduction of the arm, the coracoid process almost forms a fulcrum under which the subclavian vessels and the neurovascular bundle must pass (5,6,10). Compression results in pain, numbness, paresthesias and weakness of the upper extremity (4).

Thoracic outlet compression needs to be differentiated from ulnar and median nerve compression syndromes and cervical radicular signs. Other diagnoses mimicking thoracic outlet syndrome are a Pancoast tumor, complex regional pain syndrome (type I), cervical disc protrusions, cervical degenerative processes, poliomyelitis, tuberculosis, syringomyelitis, inflammatory processes, trauma to the brachial plexus, entrapment neuropathies, M. Raynaud, migraine occipitalis, hyperventilation and M. Paget-Schroetter (4,5,8,9).
Tests and provocations used to evaluate Thoracic Outlet Syndrome:

Multiple tests for clinical evaluation of TOS have been developed since Adson and Caffey reported on cervical rib and scalenus anticus syndrome in 1927 (11). The earlier tests used for provocative testing, described in the literature for the diagnosis of TOS, evaluated vascular integrity. These tests initially were considered positive when detecting a loss or decrease of the radial pulse, and for some tests, with reproduction of symptoms. Several specific maneuvers have been described:

Adson Test or Scalene Maneuver:

The examiner locates the radial pulse. The patient rotates the head toward the tested arm (the scalene muscles will contract) or rotates away from the tested arm (the scalene muscles will stretch and tighten thus compressing in a “scissor-like” manner). A positive test is indicated by a disappearance of the radial pulse.

Allen Maneuver:
The examiner flexes the patient’s elbows to 90 degrees while the shoulder is extended horizontally and rotated externally. The patient then rotates the head away from the side tested. A positive test is indicated by a disappearance of the radial pulse.

**Halstead Maneuver:**

The examiner finds the radial pulse and applies downward traction on the tested extremity while the patient’s neck is hyper extended and the head rotated to the opposite side. Absence or disappearance of the radial pulse indicates a positive test.

**Eden test or costoclavicular test:**

The examiner locates the radial pulse. The patient’s shoulder girdle is depressed and retracted with the patient leaning away from the arm being tested. The patient then breathes in deeply. A positive test is indicated by pain, tingling, and by a disappearance of the radial pulse.

**Wright’s test  (test of the costo-thoraco-pectoral opening):**

The examiner elevates and abducts the patient’s arm while palpating the radial pulse. A determination is made of the speed of, and the angle in the trajectory when symptoms are replicated.

**Hyperabduction maneuver  (test of the costo-thoraco-pectoral opening):**

The arms are elevated and abducted with the elbows bent. The patient is then asked to inhale deeply. The test is positive when the radial pulse is abolished or symptoms are replicated. Novak and McKinnon have adapted this test by keeping the elbow extended and the wrist in neutral as not to provoke cubital or carpal tunnel syndrome (3).
The EAST test (Elevated Arm Stress Test), Abduction External Rotation test (AER test), Stick up test, Hands up test or Roos test:

This test may be a good screening test for all types of thoracic outlet compression. The patient brings the arms up into abduction and external rotation with elbows bend and slightly behind the head. The patient then opens and closes the hands for three minutes. A positive test is indicated if pain, heaviness or profound arm weakness or numbness and tingling of the hand are present.

Not one of the tests described thus far has been accepted as the “gold standard” for TOS, and many of the tests for vascular insufficiency are reported to show high numbers of false-positive and false-negative findings. These tests might be inaccurate and insufficient in many patients with TOS because the vast majority of patients (95%) appear to have complaints relating to brachial plexus nerve compression, with or without compression of the subclavian vessels (3,12,13). Use of the previous tests for symptom reproduction shows better specificity than with testing for vascular integrity. Other tests, which include positional and stretching maneuvers, may be used to elicit and reproduce symptoms in patients with TOS. The following tests are used:

Tinel’s sign:

Tapping with the examiner’s finger over the nerves may produce a tingling sensation within the distribution of the nerve. The test is performed over the common entrapment sites in the upper extremity as the carpal tunnel, the median nerve in the forearm, the cubital tunnel, and
the brachial plexus in the infra- and supraclavicular fossa. This test tends to be positive in later stages of chronic nerve compression.

Pressure Provocative testing (mechanical allodynia):

The examiner applies direct pressure with thumb or finger tips over nervous tissue as the brachial plexus, ulnar nerve and median nerve. The test is considered positive when producing symptoms of paresthesia or numbness in the distribution of the provoked nerve.

Erb’s test or Lasegue of the arm (test for differentiation between radicular and pseudo-radicular symptoms):

The examiner brings the patient’s head into hetero lateral side bending, and the arm into extension and abduction, elbow into extension and pronation, wrist and fingers into flexion. A shooting pain with other symptoms of a herniated nucleus pulposi would indicate radicular symptoms. Burning pain with slowly increasing tingling sensations and other complaints obtained in the history would indicate TOS.

Upper Limb Nerve Tension Testing:

Butler (1) describes tension testing utilizing upper limb nerve tension tests, with emphasis on the upper limb nerve tension test 3 (ULTT 3), which is more sensitive for ulnar nerve irritation. These tension tests involve the increase of tension of nerves, through stretching by means of positioning joints in predetermined positions, to tension nerves from cervical nerve roots to the peripheral nerves in the fingers. The tests are considered positive when reproduction of symptoms is obtained or range of motion is limited. Butler also recommends performance of the slump test in longsitting and sitting to investigate the spinal canal components for adverse neural tension. He also suggests that the possibility of double or multiple crush syndromes should
be investigated, as well as the contribution of the sympathetic nervous system to the symptoms presented (1,14).

Hypomobility of the first rib has also been hypothesized as a possible cause of TOS. Lindgren et al (15) investigated patients with hypomobile first ribs, and indicated that compression of the C8-T1 nerve roots and the stellate ganglion could result in irritation, thus generating TOS symptoms. Lindgren et al described the following test to evaluate this:

**Cervical Rotation Lateral Flexion Test:**

This test is used to detect possible restriction of the first rib, which may lead to TOS symptoms and restricted cervical range of motion. The test is performed by passively and maximally rotating the neutrally positioned cervical spine away from the painful side, after which a passive lateral flexion is performed. The test is considered positive when a bony restriction totally blocks the movement, indicating a hypomobile first rib (15,16,17).

**Contrasting and comparing the provocative tests:**

Controversy has existed regarding the accuracy of maneuvers for the diagnosis of TOS. This is partially because of the varying definitions for a positive test for TOS. Furthermore, the tests that are deemed positive with detection of a decreased radial pulse are considered unreliable because of the high percentage of positive pulse changes in healthy subjects (18).

Many authors have investigated the reliability, validity, specificity and sensitivity of tests for clinical evaluation of TOS. Reliability is the extent to which repeated measurements of a relatively stable phenomenon are close to each other. Validity is the degree to which the results of a measurement correspond to the true state of the phenomenon being investigated. The sensitivity of a test is the probability of a positive test among patients with the dysfunction or disease. The
specificity is probability of a negative test among patients without the dysfunction or disease. For any tests to be a useful tool in clinical evaluation of TOS, the tests should both reliable and valid (19).

**Testing for vascular integrity:**

Many authors have investigated testing for vascular integrity in healthy subjects. The original intent of tests used for provocative testing, described in the literature for the diagnosis of TOS, evaluated vascular integrity. These tests initially were considered positive when detecting a loss or decrease of the radial pulse, and for some tests, with reproduction of symptoms. Others have investigated tests, combining evaluation of vascular and neural integrity.

Rayan and Jensen (11) assessed the prevalence of positive responses to provocative tests in a healthy population. They investigated the Adson test, the costoclavicular and the hyper abduction maneuvers for vascular and neurologic responses, as well as Tinel’s sign for a neurologic response. Tinel’s sign was positive in 7.5% of the extremities, as well as 2% for the Adson maneuver, 10% for the costoclavicular maneuver and 16.5% for the hyperabduction maneuver for a neurologic response. A vascular response was positively identified in 13.5% of the extremities for the Adson maneuver, 47% for the costoclavicular maneuver and 57% for the hyperabduction maneuver. The authors concluded that the vascular response was greater than the neurologic response in all three tests in a healthy population.

Gergoudis and Barnes (20) found high prevalence of thoracic outlet compression of the subclavian artery in normal individuals. They found significant arterial obstruction in 60% of the subjects, utilizing the Adson, costoclavicular and hyperabduction maneuvers. The Adson maneuver resulted in arterial occlusion (complete obstruction of digital pulse amplitude) in 51% of the subjects and arterial stenosis (more than 75% reduction in digital pulse amplitude) in 2%. The costoclavicular maneuver resulted in arterial occlusion in 14% without any instance of
arterial stenosis. The hyperabduction maneuver resulted in arterial occlusion in 9% of the subjects, and arterial stenosis in 10%. They concluded that provocative tests, as the Adson test, as well as the costoclavicular and hyperabduction maneuvers, used to demonstrate vascular compression, have little importance in evaluating patients with symptoms suggestive of thoracic outlet compression. The authors stated this because of brachial plexus compression and irritation being related to TOS in the majority (98%) of patients. However, they also suggested that the maneuvers investigated might be helpful in assessing patients with vascular complications of TOS.

Plewa and Delinger (21) estimated the incidence of false-positive findings of thoracic outlet syndrome shoulder maneuvers. They tested the Adson test (AT), costoclavicular maneuver (CCM), elevated arm stress test (EAST), and supraclavicular pressure (SCP) in healthy subjects. The AT, CCM, EAST, and SCP resulted in an altered pulse in 11%, 11%, 62%, and 21% of cases, respectively; pain in 0%, 0%, 21%, and 2% of cases, respectively; and paresthesias in 11%, 15%, 36%, and 15% of cases, respectively. These authors concluded that in this study of TOS shoulder maneuvers in healthy subjects, the outcomes of pulse alteration or paresthesias were unreliable in general. However, they also found TOS shoulder maneuvers to have reasonably low false-positive rates with a positive outcome defined as: pain in shoulder, arm or hand during AT, CCM, or SCP; discontinuation of the EAST secondary to pain in the arm; pain in the same arm with 2 or more of the tested maneuvers; or any symptom (pain, paresthesias or altered pulse) in the same arm with 3 or more maneuvers.

Warrens and Heaton (22) described false-positive testing in 64 randomly chosen volunteers. The tests these authors investigated were the costoclavicular test with 27% positive findings, the Adson test with 15% and the hyperabduction test with 14%. Although 17% of the subjects had any symptoms of TOS, 58% of the subjects had a positive result with at least one
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Only 2% were positive for more than two maneuvers. They suggested that the low specificity of the tests investigated, devalued these tests in clinical practice.

Marx et al (19) reviewed literature regarding reliability and validity of commonly used clinical tests for disorders of the upper extremity. They found for the Adson maneuver a specificity of 32% to 87%, for the costoclavicular maneuver a sensitivity of 94% and specificity of 53% to 85%, and for the Allen test specificity of 18% to 43%. Ranges were given to reflect the conflicting findings of the different authors, which is likely due to the fact that the authors chose varying methods to define the presence of TOS. Since true sensitivity and specificity could not accurately be determined based on the available literature, the tests were considered to have insufficient evidence for use in clinical practice.

**Testing for neural integrity through symptom reproduction:**

Positive findings in many other tests include and emphasize symptom reproduction and exacerbation of symptoms of TOS. A few studies have described prospectively the false positive rate of provocative maneuvers in healthy subjects. The EAST test (Elevated Arm Stress Test, Abduction External Rotation test (AER test), Stick up test, Hands up test or Roos test) has been the most frequently investigated test. Other tests investigated are the pressure provocation test, Tinel’s sign and the ULNTT.

Costigan and Wilbourn (23) investigated the sensitivity and specificity of the Elevated Arm Stress Test (EAST test) for evaluation of patients with neurogenic TOS, a type of TOS allegedly presenting with sensory symptoms alone, without muscle wasting or bony abnormalities. They performed testing on 65 patients with clinical and EMG evidence of carpal tunnel syndrome and 24 a-symptomatic controls. The authors found a positive EAST test in 92% of carpal tunnel syndrome patients and in 74% of a-symptomatic controls. They concluded the EAST test not to be specific for TOS. The authors suggested, in patients suspected of neurogenic
TOS, an EMG to be essential to exclude carpal tunnel syndrome before surgical intervention for TOS is considered. They also found carpal tunnel syndrome to be an unlikely differential diagnoses in absence of a positive EAST test.

Barsotti and Chiaroni evaluated the EAST test in 150 normal subjects (24). They found the test to be positive in “nearly all” of the 150 subjects at 2 minutes into the test, because of the onset of interscapular and forearm pain. This had, according to the authors, nothing to do with the usual positional syndrome, but was intolerable. In persons with TOS, the test was positive earlier than in persons without TOS symptoms, namely after 1-2 minutes.

Toomingas et al (25) investigated the results of the Abduction External Rotation (AER) Test among manual and office workers. They found the sensitivity of the AER test to be moderate (33% and 80% respectively) while the specificity was considered good (83% and 90% respectively). A positive association between the AER test and present problems in neck, shoulder, scapula, upper arm including carpal tunnel syndrome at the time of testing was noted.

Toomingas et al (26) in a different study investigated the predictive aspects of the AER test. The AER test was conducted among 137 male industrial and office workers at baseline and after 5 years follow-up, and nerve conduction measurements in the wrist regions were made. Testing after five years of the subjects with “AER signs” resulted in detection of slowed nerve conduction in the wrist area in these subjects. They concluded that the AER test predicted future neck and upper extremity symptoms and signs of nerve compression. According to the investigators, the results of this study gave support to the "double or multiple crush" theory of nerve compression. They found the AER test to be a supplementary tool, valuable in epidemiological and occupational health settings.

Supraclavicular pressure (a pressure provocation test of nerves for mechanical allodynia) was described by Roos as one of the most helpful tests in the diagnosis of TOS (13). It has been the subject of few studies. In a study by Costigan and Wilbourn (23) supraclavicular tenderness
was noted in only 4% of the subjects. Plewa and Delinger (21) found the supraclavicular pressure test rarely painful (2%), with paresthesias noted in 15% of healthy subjects tested.

Rayan and Jensen (11) described Tinel’s sign for clinical evaluation of TOS. These authors assessed the prevalence of positive responses to provocative tests in a healthy population. Tinel’s sign was positive in 7.5% of the extremities of healthy subjects. Gillard et al (28) investigated the usefulness of provocative tests in 48 patients with clinical suspicion of TOS. Tinel’s sign showed poor results with sensitivity and specificity of respectively 46% and 56%. Furthermore a positive predictive value of 63% and negative predictive value of 39% was reported.

Table 1 and 2 provide an overview of the tests for clinical evaluation of TOS through compression tests in healthy subjects and subjects with TOS.

<table>
<thead>
<tr>
<th>Test Performed</th>
<th>Author</th>
<th>Vascular Response</th>
<th>Neurologic Response</th>
<th>Positive Response</th>
</tr>
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<tbody>
<tr>
<td>Adson test</td>
<td>Rayan</td>
<td>2%</td>
<td>13.5%</td>
<td>*</td>
</tr>
<tr>
<td></td>
<td>Gergoudis</td>
<td>51% occlusion</td>
<td>*</td>
<td>*</td>
</tr>
<tr>
<td></td>
<td>Warrens</td>
<td>2% stenosis</td>
<td>*</td>
<td>15%</td>
</tr>
<tr>
<td></td>
<td>Plewa</td>
<td>11%</td>
<td>11%</td>
<td>*</td>
</tr>
<tr>
<td></td>
<td>Rayan</td>
<td>47%</td>
<td>10%</td>
<td>*</td>
</tr>
</tbody>
</table>

Table 1.
Contrasting Results of Provocative Tests in Healthy Subjects
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<table>
<thead>
<tr>
<th>Test Performed</th>
<th>Author</th>
<th>Specificity</th>
<th>Sensitivity</th>
<th>Positive and negative predictive values</th>
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</thead>
<tbody>
<tr>
<td>Adson test</td>
<td>Marx</td>
<td>32% to 87%</td>
<td>*</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Gillard</td>
<td>77%</td>
<td>79%</td>
<td>ppv 85% npv 72%</td>
</tr>
<tr>
<td>Costoclavicular maneuver</td>
<td>Marx</td>
<td>53% to 85%</td>
<td>94%</td>
<td>*</td>
</tr>
<tr>
<td>Hyperabduction maneuver (vascular response)</td>
<td>Gillard</td>
<td>90%</td>
<td>52%</td>
<td>ppv 92% npv 47%</td>
</tr>
<tr>
<td>Hyperabduction maneuver (neurologic response)</td>
<td>Gillard</td>
<td>40%</td>
<td>84%</td>
<td>ppv 74% npv 55%</td>
</tr>
<tr>
<td>Tinel’s sign</td>
<td>Gillard</td>
<td>56%</td>
<td>46%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Barsotti</td>
<td>*</td>
<td>100%</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Toomingas</td>
<td>33% to 80%</td>
<td>83% to 90%</td>
<td>*</td>
</tr>
<tr>
<td></td>
<td>Gillard</td>
<td>30%</td>
<td>84%</td>
<td>ppv 68% npv 50%</td>
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<tr>
<td></td>
<td>Marx</td>
<td>18% to 43%</td>
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</tr>
<tr>
<td></td>
<td>Gillard</td>
<td>53%</td>
<td>70%</td>
<td>ppv 72% npv 50%</td>
</tr>
<tr>
<td></td>
<td>Gillard</td>
<td>29%</td>
<td>90%</td>
<td>ppv 69% npv 63%</td>
</tr>
</tbody>
</table>

* No data available

**Table 2.** Contrasting Results of Provocative Tests in Subjects with TOS

**Testing through nerve stretching:**

The majority of patients with TOS, with adverse neural tension, are presenting with neuropathic pain as primary feature. Testing of neural structures utilizing compression for symptom reproduction (Tinel’s sign, pressure provocative testing) as well as nerve stretching for
specific symptoms and mobility (Erb’s test, ULNTT) is performed. The utilization of upper limb neural tension testing (ULNTT) and nerve mobilization (NM) has been gaining popularity (14).

Sandmark and Nisell (27) investigated the validity of five common manual neck pain provoking tests, including the Upper Limb Nerve Tension Test (ULNTT). They determined that the ULNTT had a specificity of 94%, a sensitivity of 77%, a positive predictive value of 85% and a negative predictive value of 91% in 22 out of 75 subjects randomly selected with reported neck pain. In a study of 60 patients with repetitive strain injury, Elvey and associates (1,14) found the ULNTT result positive in 59 of the patients for reproduction of their symptoms.

Range of motion limitations have also been examined in conjunction with upper limb nerve tension testing. Pullos (1,14) used the ULNTT to compare the limitation of elbow motion between normal subjects and patients with neck-arm pain. She found no difference in 100 normal subjects between right and left arms, with an 8-degree difference considered normal. In 25 neck-arm pain patients, 68% had a positive ULNTT with asymmetrical elbow motion, and symptom provocation.

Walsh (14) concluded after reviewing literature that the interrater reliability of the ULNTT still to be questionable, and the intrarater reliability to be strong, although the criteria used in several studies to determine this had not been clearly defined. Reported specificity and sensitivity were high but, according to Walsh, there was a lack of controlled studies from which to draw hard conclusions.

**Testing for mobility of the first rib:**

Some authors have proposed that hypomobility or subluxation of the first rib could contribute to irritation of the C8-T1 nerve roots and the stellate ganglion, thus generating TOS.
symptoms. Lindgren and Leino (17) reported on patients with subluxation of the first rib as a possible mechanism for creating symptoms of TOS. They described 22 cases of both TOS and complex regional pain syndrome, type I. Each case had a hypomobile first rib on the affected side. After isometric exercises of the scalene muscles, the mobility of the first rib was restored and symptoms completely relieved in 13 subjects. They concluded that subluxation of the first rib may irritate the neural network and the stellate ganglion close to the first costotransverse joint.

Lindgren et al (15,16) also investigated the use of testing the combined motion of cervical spine rotation and lateral flexion (CRLF test) for detection of subluxation and restricted movement of the first rib. They found excellent interexaminer reliability. The number of false positive tested ribs was 3 out of 23 subjects; the false negative rate was 2 out of 23 subjects. They suggested inclusion of the test in examination for cervical mobility, as well as for patients presenting with symptoms of brachialgia and TOS.

Testing through combinations of multiple tests:

Several authors have reported on improved specificity and reliability of tests for diagnosis of TOS by combining multiple tests with other areas of the clinical evaluation, as the history, range of motion and muscle tightness. Combinations of tests and specific areas of clinical evaluation have shown significantly improved sensitivity and specificity.

Ribbe and Lindgren (18) studied 315 patients with cervicobrachial symptoms. They found the most reliable symptoms to detect TOS to be: (1) a history of aggravation of symptoms with the arm elevated, (2) a history of paresthesias in C8-Th1, (3) tenderness over the brachial plexus supraclavicularly, (4) a positive abduction and external rotation test (AER test). They found three out of four tests present in 94% of the TOS cases investigated. Thirty-three percent of the time they had false-positive results. The investigators referred to these four tests as the “thoracic outlet syndrome index”. Findings were confirmed by Plewa and Delinger (21). They
reported reasonable specificity when tests resulted in pain in the same arm with 2 or more of the
tested maneuvers, or any symptom in the same arm with 3 or more maneuvers.

Gillard et al (28) investigated the usefulness of provocative tests in 48 patients with
clinical suspicion of TOS. They investigated the Adson test, the hyperabduction maneuver, the
Wright test, the Roos test and Tinel’s sign. They found limited sensitivity and specificity of the
tests. Considering all five provocative tests, the tests showed a mean sensitivity and specificity of
respectively 72% (deemed acceptable) and 53% (deemed poor). The Adson test had the best
outcome for sensitivity and specificity with respectively 79% and 76%. Tinel’s sign showed the
worst outcome for sensitivity and specificity with respectively 46% and 56%. Pairs of tests
improved the specificity compared with tests alone; the gain in sensitivity, however, was smaller
then the gain in specificity. Combinations of three, four and five maneuvers involving the Adson
test, the Wright test and hyperabduction maneuver with abolition of radial pulse, the Wright test
with reproduction of symptoms, the Roos test and Tinel’s sign increased specificity but not
sensitivity. The sensitivity and specificity mostly improved when several provocative tests were
used in combination with the best test being the Adson test, particularly when used in
combination with the Roos test (sensitivity 72%, specificity 82%), the hyperabduction maneuver
(sensitivity 72%, specificity 88%), or the Wright test (sensitivity 79%, specificity 76%).

Pascarelli and Hsu (29) investigated work related upper extremity disorders in 485
computer users, musicians and others engaged in repetitive work. They found that 70% of the
persons had clinical evidence of TOS after testing for a positive reaction in two out of five tests.
These tests consisted of the EAST test, Wright’s maneuver, positive supra- and infraclavicular
mechanical allodynia, and scalene tightness with decreased range of motion in the neck.

Roos (13) uses five tests, which he believes are the most reliable for diagnosing
neurogenic TOS: Tinel’s sign (performed over scalene, and supra- and infraclavicular), pressure
provocative testing (supraclavicular), strength testing (triceps, interosseous hand muscles and handgrip testing), hypesthesia (C8, T1 dermatomes) and the EAST test.

**Summary of the tests:**

Results of the tests as described in table 1 and 2 vary strongly. Several reasons for these variations can be identified in the articles investigated. Many of these reasons are identified and described.

**Reasons for variation of tests:**

The definitions for a positive test might have been different in the investigation of tests making a comparison less effective. When based on findings of a diminished radial pulse, many maneuvers are thought to be unreliable, since pulse alterations may be found in high percentages of healthy individuals. Execution of the tests may have been different, as well as the duration of performed tests, which is not always reported. Furthermore, provocative testing can exacerbate upper limb symptoms, even when related to other causes than TOS (28). Positive tests may have identified patients with asymptomatic carpal tunnel syndrome, cervical disc disease or individuals with a predisposition for TOS (21) and have influenced outcomes. Sample size varied strongly, from 22 subjects in the investigation of Lindgren (15) to 485 subjects in the review by Pascarelli and Hsu (29). Also the composition of the samples has influenced the results, since females show a greater response than males. This difference correlates with the clinically observed prevalence of TOS in the general population, probably due to anatomical differences of the chest wall between the two sexes (18). Age composition of the samples may have played a role. The younger age group in the article by Rayan and Jensen (11) had a tendency for a greater vascular response than the older age group. Occupation of the patients has a correlation with positive testing for TOS. Toomingas et al (26) reported that vibration exposure and the vibration
acceleration level experienced during work were associated with a positive AER test.

Furthermore, they found a positive correlation between the AER test and neck and arm pain and, to some extent, carpal tunnel syndrome. This indicates that the presence of minor serial impingements along a peripheral nerve may result in a positive test for TOS and has to be taken into account. Therefore these authors recommended inclusion of testing of more distal sites of possible nerve compression.

Comparing the tests:

Some authors report significantly higher or lower outcomes than others. The investigation of the Adson test by Gergoudis (20) reveals a very high positive response in comparison with other authors. No duration of the test performed was given in the article, which might have influenced the outcome. Marx et al (19) found, in his literature review, a range of specificity of 32% to 87% for the Adson test in patients with TOS, as well as ranges of 53% to 85% and 18% to 43% for respectively the costoclavicular and Allen test. They indicated that the conflicting results were probably due to the fact that the investigators choose varying methods to define the presence of TOS. Rayan (18) reports a high vascular response for the costoclavicular test as well as the hyperabduction maneuver. The composition of his samples for age and gender might have influenced the outcomes in their study. The EAST test shows high false-positive results in healthy subjects, in the literature reviewed, ranging from 57% neurologic response to 100% positive response (19, 23). The duration of the test plays an important part in this test. Roos (30) indicates that the test should be performed for three minutes, which according to several authors results in poor specificity (20, 23, 24). Plewa (21) indicated that shortening the test to 90 seconds might increase the specificity, since many of their healthy subjects did not develop symptoms until after 90 seconds. Many other tests as the Allen maneuver, the Halstead maneuver, the CRLF test and the Wright test have not been extensively reported on.
Results of the tests:

Sensitivity and specificity of the tests for vascular integrity, when used in isolation, are mostly low. False-positive results are high. Tests used in isolation often result in unreliable tests. However, combinations of tests appear to increase specificity and sensitivity, as well as decrease the rate of false-positive results (21). The Adson test, particularly when used in combination with the hyperabduction maneuver, the Roos test and the Wright test show satisfactory specificity and sensitivity (28). Of the tests for neural integrity, the ULNTT appear to have satisfactory outcomes although a lack of controlled studies from which to draw hard conclusions is reported (14). Testing for mobility of the first rib has been the subject of few studies (15,16,17). The CRLF test appears to be a good addition to the “standard “ TOS tests, although further investigation with larger samples is warranted before hard conclusions regarding reliability can be drawn.

Suggestions:

Future investigation of tests used for clinical evaluation should include a sufficient sample size. Depending on the type of investigation, it should represent the proper population investigated, in gender, age and occupation, since this may have significant influence on the outcomes. Degree of pressure or traction given during testing should also be measured, as well as duration of the tests performed, to improve objectivity of the tests.
Diagnostic difficulties:

Several difficulties regarding the anatomy and pathophysiology of the thoracic outlet exist, making the diagnosis of TOS through clinical testing complicated. Experts regarding the topic of TOS have diverging views on the pathogeneses. There is no agreement on whether the basic injury is one of compression or traction.

Several theories regarding the pathogenesis of TOS have been proposed (31). The first theory suggests that, since anatomical anomalies are prevalent in the normal population, they might predispose healthy subjects for symptoms of TOS, as well as to false-positive provocative testing (13,32).

As second theory occupational and postural factors as descending of the shoulder girdle in middle-aged adults has been implicated in the pathophysiology of TOS (33). Normal rib and muscle structures could cause brachial plexus compression and stretching because of muscle disuse atrophy, muscle imbalance, muscle shortening, compensatory muscle overuse, protraction of the shoulders and forward head posture (2, 18).

A third theory proposes that trauma could result in scarring of the brachial plexus. Nerve tissue could adhere to other structures causing decreased range of motion. Bleeding and subsequent fibrosis, contractures and shortening of connective tissue could be the result. Eventually a change in muscle type of the scalene muscles might be seen (31).

Diagnosis of TOS:

Accurate diagnosis of thoracic outlet syndrome is difficult for many reasons. Clinical diagnosis of TOS is presently based predominantly on subjective complaints of the upper
extremity as paresthesia, anesthesia and pain. Many provocative tests, resulting in subjective complaints, reveal low specificity and sensitivity. Objective tests, mostly used by other professionals than Physical Therapists, as X-ray films, computed tomography scans, nerve conduction studies and somatosensory evoked potentials appear most useful for diagnosing moderate to severe cases of vascular or neural compromise (31) as well as for ruling out of other diagnoses (2,3).

Several provocative tests and maneuvers have been described in the literature. The original provocative tests described for the diagnosis of TOS evaluated vascular integrity, noting a loss or decrease of radial pulses with or without the reproduction of symptoms. These tests, which evaluated the effects of positional changes on the radial pulse for diagnosis of TOS, and other tests have proven unreliable because of the high incidence of false-positive findings in healthy subjects. (18,19,20,21,22,23,24). Testing for neurologic response has shown poor to fair specificity and fair to good sensitivity. Only nerve tension tests and testing for mobility of the first rib have shown good specificity and sensitivity. Unfortunately, there is a lack of controlled studies from which to draw hard conclusions (14).

**Influence of anatomical abnormalities and posture on testing:**

Juvonen et al (32) investigated the rate of anomalies at the thoracic outlet in the general population. They found normal bilateral thoracic anatomy in 5 out of 50 cadavers (10%) during 98 dissections. They concluded that congenital anatomic abnormalities are frequent in the normal population. They also suggested that the abnormalities would predispose subjects to TOS after stress, related to occupation and cervical injuries Roos initially described 7 different types of congenital bands and ligaments, and finding these in 33% of the general population. Roos later increased this number after finding 7 more anomalies to 14 (13,31,32).
Occupational factors have been hypothesized to be a factor in development of TOS. Hairdressers, painters, construction and industrial workers, nursing staff, switchboard operators, and other workers performing repetitive manual work (rather than heavy work) have an increased prevalence of TOS. Juvonen et al reported 60-80% of the normal population to have positive findings in clinical tests aimed at irritating the neurovascular bundle in the thoracic outlet. They suggested that the presence of abnormal fibromuscular bands often resulted in positive findings in during provocative tests for TOS. This would predispose subjects to TOS after certain stress, possibly related to occupation or cervico-brachial injury (32).

Machleder (34) indicated that anatomical abnormalities represent stages in a spectrum of developmental variation of the neurovascular and musculoskeletal structures at the base of the neck. He concluded that the abnormalities could become significant in settings of unusual physical requirements, repetitive stress, or injury. He indicated that repetitive mechanical stress on the job could eventually expose anatomical weaknesses in the thoracic outlet.

Several authors describe congenital abnormalities, which could contribute to the pathogenesis of TOS. Examples are cervical ribs, abnormal thoracic ribs, and scalene muscle- and insertion abnormalities. Table 3 shows a classification Gruber made of the cervical rib anomalies in 1842.

### Table 3. Gruber Classification of Cervical Rib Anomalies:

<table>
<thead>
<tr>
<th>Anomaly Type</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type I</td>
<td>large, hypertrophic transverse process of C7</td>
</tr>
<tr>
<td>Type II</td>
<td>rudimentary rib, but with free extremity and no connection to the first rib</td>
</tr>
<tr>
<td>Type III</td>
<td>incomplete rib connected to the first thoracic rib by a fibrous band</td>
</tr>
<tr>
<td>Type IV</td>
<td>complete cervical rib fusing with the first rib or connecting with it by a cartilaginous pseudo-articulation</td>
</tr>
</tbody>
</table>
Cervical ribs occur in 0.5-1.5% of the general population; they are three times more frequent on the left side. Presence of cervical ribs in a patient with upper extremity symptoms would support a diagnosis of TOS (5,36).

**Vascular versus neurological TOS in relationship to testing:**

Another important reason for the low specificity and sensitivity of the “original” TOS tests may be the fact that 90-95% of TOS symptoms is attributed to compression or stretching of the brachial plexus at the thoracic outlet, while vascular compression symptoms are thought to be present in about 5% of the patients with TOS (35).

Roos indicated that, because tests evaluating the effect of positional changes on radial pulse have proven unreliable, examination of neurologic symptoms is pertinent for establishment of the diagnosis of TOS (18). He reported that symptoms of TOS are related to brachial plexus compression and irritation rather than to vascular compression, and found this in 98% of the cases investigated (30).

Swift and Nichols reported that some patients with TOS had droopy shoulder syndrome, and suggested that the symptoms resulted from stretching of the brachial plexus (33).

Nakatsuhi et al (12) proposed that the symptoms of TOS might be related to increased tension of the brachial plexus and surrounding vasculature due to muscular imbalance and the resultant downward traction. Ide et al (12) investigated the mechanism of nerve irritation in thoracic outlet syndrome. One of their findings was that the provocation of TOS through pulling the arms down and back was often not a result of compression, but of stretching of the brachial plexus between distal and proximal fixed points. Symptoms were aggravated by downward traction of the arm (traction on the brachial plexus), and relieved by pulling the arms upward (relaxing the brachial plexus). They distinguished three subsets of patients with neurogenic TOS: those with only compression irritation of the brachial plexus (type I TOS, 18%), those with a combination of
stretching and compression irritation to the brachial plexus (type II TOS, 74%), and those with only stretching irritation of the brachial plexus (type III TOS, 8%). The three subsets were found to have characteristic responses to provocative maneuvers, with traction rather than compression, producing signs and symptoms in a majority of the patients with type II and type III TOS.

Disputed neurologic TOS and testing:

Wilbourn (31) divided TOS into four distinct subgroups: arterial vascular, venous vascular, true neurologic and disputed neurologic TOS (disputed N-TOS). Several other names for disputed N-TOS have been proposed including “assumed N-TOS”, “aspecific N-TOS”, “nonspecific N-TOS”, “symptomatic N-TOS”, “TOS without objective findings”, and “cervical-brachial neuralgia”. The first three subgroups are uncontroversial. They share several features including presentation with very low incidence, a characteristic presentation, and one or more reliable, confirming laboratory procedures. The fourth subgroup of disputed N-TOS is unclear in etiology. Several theories regarding the pathogenesis of disputed N-TOS have been proposed. They can be divided into three categories: (1) congenital anomalies, (2) postural factors and (3) traumatic injury.

Pascarelli and Hsu stated that “neurogenic TOS is a clinical diagnosis and as such, is a victim of the tendency to discount physical findings in favor of laboratory tests, which in case of disputed N-TOS are mostly negative” (29).

Chronic nerve compression and its influence on testing:

A further explanation for the difficulty of diagnosing TOS might be the intermittent nature of the dysfunction. Novak et al (2) describe the presence of chronic nerve compression with possible histological changes. These can progress from changes in the blood nerve barrier function, to connective tissue thickening, to local followed by diffuse changes of segmental demyelination, and finally to Wallerian degeneration with nerve fiber loss. Patient presentation
reflects these patho-histological findings. In the early stages, the patient may present symptom
free and will describe symptoms only when provoked with the extremity in certain positions.
Physical findings will be limited to positive provocative tests (pressure or positional). In moderate
degrees of nerve compression, changes in vibration and pressure thresholds and muscle weakness
may be apparent. In severe nerve compression, loss of nerve fibers will be present, combined with
atrophy and decreased two-point discrimination. Sensory complaints parallel the changes,
beginning with intermittent paresthesia in certain positions. Progression into persistent
paresthesia and finally constant numbness may occur. When pain is prevalent, patients may avoid
aggravating positions and modify activities to minimize discomfort, and thus avoid progressing
into the moderate and severe stages. These patients present with positive positional and pressure
provocative tests, but rarely with positive objective test abnormalities (2,31).

**Double crush phenomenon and testing for TOS:**

Another confusing entity could be the presence of the double crush phenomenon. Upton
and McComas postulated their double crush phenomenon theory (1,2,18) in 1973, suggesting that
minor serial impingements along a peripheral nerve could have an additive effect and result in
neuropathy. The basis of the distal neuropathy was considered to be altered axoplasmatic flow.
The phenomenon is applicable to TOS in the thoracic inlet and outlet, as well as to the more distal
sites of nerve compression, as compression of a number of sites may contribute to the patient’s
symptoms, distally and proximally through retrograde pain radiation. In a study by Costigan and
Wilbourn (23) supraclavicular tenderness was noted in only 4% of the subjects. However, they
also found a positive supraclavicular pressure test in 23% of with confirmed carpal tunnel
syndrome. Therefore the authors recommended including of more distal sites of possible nerve
compression to be tested, as the cubital and carpal tunnel, in examination of the brachial plexus in
patients with symptoms of TOS (2,38). Toomingas et al (26) recommend that prevention,
evaluation, and management of neck and upper extremity nerve compression diseases should attend to all probable locations of such compression.

**The role of posture as a cause of Thoracic Outlet Syndrome:**

This chapter describes the possible influence of posture on the pathogeneses of TOS. Several problems are described including the role of normal and abnormal development of posture, muscle imbalance and its effect on posture, neural involvement and its effect on posture, and predisposing functional activities and work duties, all relating to TOS.

**Development of posture relating to TOS:**

During normal development of the human shoulder girdle, the scapula descends from a relatively high position at birth to a lowered position in adulthood. These relationships are influenced by hypertrophy and atrophy of musculature as well as chronic postural positions. The scapulae are entirely suspended by musculature. Especially the rhomboid and the levator scapulae muscles are therefore important in considering pathology and possible treatment (8).

Many problems in the thoracic outlet are congenital rather than acquired. Because of the (changing) configuration of the shoulder girdle throughout life, true thoracic outlet compression is rarely found before puberty. Several factors influencing posture and leading to increasing angulation of the neurovascular structures may result in symptoms, as previously described (8). The scapula tends to descend more in females then in males, partly explaining a greater incidence of thoracic outlet syndrome in women then in men. Increasing age and decreasing mobility as well as changes in the shoulder girdle, in combination with excessive body weight and breast hypertrophy, appear predisposing factors for symptoms to become manifest (4,8).
Muscle imbalance and posture relating to TOS:

Muscle imbalance or muscle strain can be a major source of symptoms in patients with thoracic outlet compression. With persistent positional changes, musculature is able to change its resting length. This may have a significant functional impact because of weakening of musculature. The typical “slumped” rolled-forward posture of the shoulders and neck are comfortable, but is a potentially damaging posture for the scapular and neck musculature (2).

Janda describes the effects of myofascial imbalances on postural equilibrium extensively. His principles include the relationship of postural versus phasic musculature and their correlation with agonist/antagonist muscle groups; postural musculature having a predisposition to react to dysfunction with shortening and tightening, and phasic musculature reacting with weakening. His description of the “Oberkreuz- and Unterkreuz-syndrome” describes the vital role of agonist/antagonist relationships in postural problems involving the spine and extremities (2,13). Sucher and Heath (1993) describe several structural and postural considerations for TOS. They indicate that thoracic outlet compression rarely is confined to one “primary” dysfunction. The localized dysfunction at the thoracic outlet may be one aspect or a regional component of a global musculoskeletal or systemic viscero-somatic dysfunction. (14).

In osteopathic literature mechanical linkage in TOS is described through decompensation in the frontal plane, as with leg length discrepancies. This would result in development of trigger points or somatic dysfunction. “Thoracic distress” and shoulder pain have been noted with this decompensation, as well as the perpetuation of shoulder girdle dysfunction leading to the symptoms of thoracic outlet compression. Sagittal plane decompensation through excessive
hyper- or hypolordosis can result in compensatory protraction of the shoulder girdle thus encouraging the “thoracic outlet posture” (14).

Robin Mckenzie has developed a concept of treatment, describing the presence of pain and decreased mobility, in conjunction with forward head posture, protracted shoulders, decreased lumbar lordosis, and a bent and stooped posture. He relates three predisposing factors related to TOS: poor sitting posture, increased frequency of flexion in the cervico-thoracic spine and loss of cervical lordosis and mobility (15).

**Neural involvement and posture relating to TOS:**

Neural involvement has been proposed to play a significant role in TOS. Hyper-excited or “facilitated” segments in the spine may result in an exaggerated response to even mild stimuli from remote sites. Trigger points can play a role through perpetuating and maintaining TOS symptoms. Sympathetic nerve involvement might further influence symptoms, as well as peripheral nociceptor branches from the upper thoracic region, which are believed to project to the brachial plexus (14).

Double or multiple crush syndromes have been suggested to play a role in TOS. The alteration of axoplasmatic transport at proximal level would account for the increased susceptibility at other sites, distally, along the periferal nerves. In the case of brachial nerve compression multiple sites of compression contribute to patient symptoms. High incidences of cubital and carpal tunnel syndrome are associated with nerve root compression and alterations of axoplasmatic flow resulting in subsequent pathologic changes. Identification and treatment of all sites of compression is needed for successful treatment of TOS (1,2).

**Functional activities and posture relating to TOS:**
Muscle swelling from trauma, exercise or hypertrophy may initiate the syndrome.

Wearing of heavy backpacks, which these days appear to be normal in schools, will pull the shoulders back and down. Also individuals who have acquired a backwards and downwards posture of the scapulae as in military posture may develop symptoms (3,8).

Athletes (swimmers, volleyball players, tennis players, baseball pitchers) and individuals with high development of the trapezius muscle and other neck musculature are reported to run the highest probability of developing symptoms of thoracic outlet compression (8).

Individuals who perform work duties requiring prolonged overhead activities (painters, electricians, plasterers, mechanics) are at risk. Workers using static work postures (assembly line workers, cash register operators, students, needle workers) may accentuate forward head posture, depressed scapulae and development of symptoms (4,5). Abnormal static and repetitive work postures can have three effects. First, work postures and positions can result in increased nerve tension. Examples would be increase of brachial plexus tension during overhead reaching, and elbow flexion, increasing tension in the ulnar nerve. Second, certain work postures can maintain muscles in abnormally shortened positions, resetting them to new length. This may result in pain upon stretching of muscles, as well as compression of neurovascular structures if being crossed by shortened and tight musculature. An example would be head-forward scapular abducted positions resulting in tightness of the scalene and/or pectoralis minor muscle, compressing the brachial plexus. Third, abnormal work postures will result in some muscles being placed in shortened positions and others in lengthened positions, placing both at mechanical disadvantage.

With weakness of one set of muscles and overuse of another, the result will be muscle imbalance. This can evolve in forward head position, increase of thoracic kyphosis and scapular abduction.

For these reasons addition of ergonomic assessment of the workplace to a treatment plan might be necessary to remove etiology (4,5,13).
Examination of the patient with suspected TOS should include several areas of evaluation. It should include history including questionnaires, clinical investigation of TOS, and investigation of other problems. This may include cervical ribs, peripheral neuropathy and other pathology, which has to be excluded.

**History:**

The patient’s history of the complaints may assist in diagnosing a dysfunction. Neurogenic problems can result in complaints of paresthesias and numbness, especially in the ulnar distribution of the forearm, and could be related to nerve compression in the region of the brachial plexus. Pain questionnaires may assist in determining the impact of the patient’s dysfunction on his life. Overhead activities and prolonged abduction as well as traction (lifting groceries) will exacerbate the upper extremity complaints. At times, patients may report an injury or accident with a slowly progressive onset of upper extremity pain. Arterial compression will often result in coldness of the hands, and Raynaud’s phenomenon. Venous abnormalities will present with edema and venous congestion. However, the majority of patients with TOS will present with neurogenic symptoms.

**Clinical testing:**
Sensory testing for vibration, stationary and moving touch, as well as two point discrimination may be helpful to determine the degree of nerve compression. Posture could be an important component of TOS as well as muscle imbalance in the neck, shoulder and upper back. Mobility testing of several joints may be important (3). Testing of the mobility of the first rib (CRLF test), cervicothoracic junction, acromioclavicular joints, the sternoclavicular joints and the scapulothoracic joints should be performed. Depending on the patient’s history, vascular testing and/or testing for neural integrity should be performed. The Adson test, particularly when used in combination with the hyperabduction maneuver and the Wright test show satisfactory specificity and sensitivity and are probably the best tests for vascular integrity. Use of more tests may increase the specificity for TOS. Neural integrity can be investigated with the ULNTT for investigation of the brachial plexus and the peripheral nerves.

Other investigation:

Cervical ribs occur in 0.5-1.5% of the general population, and are three times more frequent on the left side. These may be palpated or may be seen in radiographic investigation. Presence of cervical ribs in a patient with upper extremity symptoms would support a diagnosis of TOS (5,36). A request for radiographic investigation might be warranted. Other relevant pathology should be excluded. Evaluation of more distal sites of possible nerve compression should be tested. Compression of nervous tissue, especially in the cubital and carpal tunnel, may result in complaints mimicking the symptoms of TOS in the upper extremity (3). Objective testing may exclude pathology or could diagnose moderate to severe vascular or neurogenic TOS. Evaluation of coexistent distal nerve compression should be performed (18,37).
Relevance of physical therapy in treatment of TOS / treatment options:

The following chapter will discuss effectiveness of physical therapy intervention in treatment of TOS, as well as provide a summary of several treatment options, available to the physical therapist.

Physical therapy plays an important role in conservative treatment of TOS. Lindgren (1997) reports in his study that 88% of the patients were satisfied with the outcome of their treatment, and that the ranges of motion of the cervical spine and upper thoracic aperture had normalized in 8 of 10 patients (16). Novak (1995, 2002) reports success with conservative treatment of TOS ranging from 50% - 90%. The achievement of poor results was related to obesity, workers' compensation and associated carpal or cubital tunnel syndrome (2,16,17).

Physical therapy treatment of TOS requires treatment of the impairments causing the brachial plexus compression and other levels of nerve compression, and treatment of muscle imbalance in the cervico-scapular region. It also requires treatment of other conditions present, but not clearly directly related to the TOS, such as foraminal compression, cervical disc disease,
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rotator cuff tendonitis and epicondylitis (2). Attention to only one component of the TOS problem will not result in total relief of symptoms.

The following treatment options can assist in treatment of TOS:

**Patient Education:**

Patient education is a vital component of a comprehensive treatment plan for TOS, and patients with highly irritable conditions must begin modifying their activities to spend less time in irritating positions. Sleeping postures should be addressed since patients frequently wake up with exacerbated symptoms. Patients awakening with pain, paresthesias, numbness, headaches and neck stiffness are possibly aggravating their symptoms in the cervico-scapular region. Change of neck posture through use of different pillows or cervical supports may be prescribed. Advise or referral for weight reduction and breast support may be needed if necessary (2).

**Treatment of head posture and cervical mobility:**

Head posture and cervical mobility should be examined to determine the presence of forward head posture, for which McKenzie propagated a treatment approach. He indicates that forward head posture may result in three predisposing factors related to TOS: poor sitting posture, increased frequency of flexion in the cervico-thoracic spine and loss of cervical lordosis and mobility. Evaluation often reveals decreased cervical retraction. Stretching exercises are started for improvement of upper cervical spine mobility, and are advanced towards cervical spine extension, side bending, rotation and flexion (15). Manipulation techniques may also be used to improve cervical mobility and to decrease dysfunction or aberrant motion. Decrease of forward head posture may further influence shoulder mobility by relieving restrictions into abduction and external rotation, thus decreasing TOS symptoms (18).
Muscle re-education and stretching:

Janda describes the muscle imbalance in the cervico-thoracic region, with other authors adding to his work. The group of muscles considered postural agonists, prone to tightening and shortening, consists of the upper trapezius, sternocleidomastoideus, levator scapulae, pectoralis major and minor, and cervical erector spinae musculature (suboccipital musculature). The group of muscles considered the phasic antagonist group, prone to weakening, consists of the latissimus dorsi, mid and lower trapezius, rhomboids and anterior cervical musculature (2). Because patients with TOS usually present with tightness in the postural musculature, stretching of the tight musculature will be effective. Stretches are most effective with low force and long duration. The stretching should be performed multiple times a day, progressively stretching a little further each time, to create and maintain true elongation of the muscle. Discomfort after stretching should subside within seconds to minutes although persistent pain for 24 hours has been reported, probably due to breaking up of adhesions or irritation of neurovascular structures (3,19).

Soft Tissue Manipulation:

Myofascial release and soft tissue manipulation may assist with decrease of pain and improvement of mobility. This is accomplished by treatment of trigger points, freeing up of restrictions in musculature, fascia and joints, and through increase of circulation. Three approaches can be distinguished, namely the autonomic approach (Connective Tissue Massage, Hoffa massage), the mechanical approach (Rolfing®, Tragering®), as well as use of movement approaches (Alexander®, Feldenkrais®) for postural education (13).

Neural mobilization:

Neural tissue stretching should be included in treatment since neural connective tissue can become short and tight with abnormal postures. Butler describes several techniques for
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evaluation and treatment of the peripheral nerves, spinal canal components and sympathetic fibers involved in TOS (1).

**Strengthening exercises:**

Strengthening exercises are not started until patients have achieved adequate pain-free range of motion. These should be aimed towards strengthening of the phasic muscles, consisting of the latissimus dorsi, mid and lower trapezius, rhomboids and anterior cervical musculature, as well as the serratus anterior muscle. Improvement of strength will result in decreased hyperactivity of the postural musculature thus breaking the cycle of muscular imbalance in cervico-thoracic region. Strengthening should be directed at both power and endurance, necessitating the aerobic conditioning of the patient. Often poor aerobic condition is accompanied by poor posture. This results in overuse of accessory respiratory musculature, in particular the scalene, the sternocleidomastoideus and trapezius muscles. Diaphragmatic breathing exercises will result in a more efficient breathing pattern and thereby decrease use of accessory respiratory musculature. Postural correction in a more upright posture will decrease flexion and improve chest expansion with inspiration (2).

**Modalities:**

Modalities as heat, laser, transcutaneous electrical stimulation, ultrasound, diathermia, cold, or massage are not necessary for successful treatment of the patient with TOS. They can however facilitate treatment by decreasing pain and/or preparing connective tissue for further manipulation by increasing or decreasing extensibility (2,3,14). Influence of analgesics/anti-inflammatories and muscle relaxants during treatment should be considered because these medications have often been prescribed to the patient, to improve ability to sleep and function (19).
**Postural education:**

Postural instruction starts with patient education about symptom-exacerbating postures and symptom-relieving postures. Positions during work, at home and during sleeping should be modified for long-term relief (2). Movement approaches as the Alexander® and Feldenkrais® techniques for postural education and improved postural awareness may also prove beneficial (13).

**Conclusion:**

Many provocative tests have been described for the diagnosis of TOS. This paper categorizes the most commonly used tests. It also compares and contrasts information regarding reliability, validity, sensitivity and specificity in available literature of tests and provocations used to evaluate Thoracic Outlet Syndrome. Most tests either investigate vascular integrity, through decrease or obliteration of the radial pulse, or neural integrity through exacerbation and reproduction of symptoms, through compression and stretching of nervous tissue. Individually, the tests that use compression show, as investigated by several authors, results ranging from poor to fair. When these tests are used in combination the Adson test, particularly when used in combination with the hyperabduction maneuver, the Roos test and the Wright test show improved specificity and sensitivity. Increase of the number of tests may increase the reliability. The ULNTT as test for neural integrity through stretching appears to have satisfactory outcomes although a lack of controlled studies from which to draw hard conclusions is reported. Testing of
mobility of the first rib joint is highlighted in this paper through the CRLF test and shows, as far as investigated, excellent reliability.

The diagnosis of thoracic outlet syndrome remains mostly a clinical decision. The diagnosis appears best made by obtaining the patients history. Then a combination of appropriate tests for TOS, resulting in an increase of specificity and sensitivity of these tests over individually performed tests, should be performed.

Examination and management of TOS should be directed towards altering all the aggravating factors and sites of compression that result in increased symptomatology, since treatment of one site or factor will likely result in partial relief (2,3, 37,38).

References:


19. Marx RG, Bombardier C, Wright JG. What do we know about the reliability and validity of physical examination tests used to examine the upper extremity? J Hand Surg. 1999 Jan; 24(1):


