APPLIED KINESIOLOGY AND THE

THORACIC OUTLET SYNDROMES

Clive Lindley-Jones B.Ed.(Hons) D.O. M.R.O.

ABSTRACT

A brief outline is given of the history, etiology, diagnosis, examination and treatment options for Thoracic Outlet Syndrome with special reference to the role that Applied Kinesiology can play in enhanced, diagnosis and effective conservative treatment.

INTRODUCTION

The symptoms, sometimes subsequent to trauma, of pain and paresthesia extending from the lateral aspect of the neck into shoulder and arm, paresis in the hand, sometimes with vascular symptoms such as intermittent swelling of the limb with exercise and/or Raynard's phenomenon, all often aggravated by hyperabduction, is one commonly seen by most practitioners using Applied Kinesiology in their practices.

This paper is intended as a review of the classic forms that Thoracic Outlet Syndrome (T.O.S.) can take. It is often the cause of such signs and symptoms mentioned above and so we shall look at the contribution that Applied Kinesiology can make to its successful conservative treatment, in the great majority of cases.

The thoracic outlet is a three dimensional space made up of the anterior scalene muscle anteriorly, the medial scalene muscle posteriorly and the first rib inferiorly

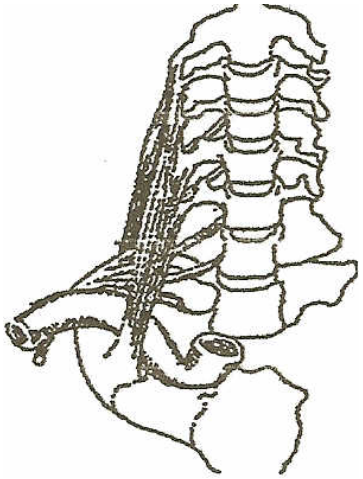

Figure 1. Anterior and medial scalenes with neurovascular bundle passing between. [1]

This condition is known variously as Scalenus Anticus syndrome, Claviculocostal syndrome, cervical rib syndrome or Pectoralis Minor syndrome. Whilst these terms help point to the possible areas of entrapment, as Cole[2] says;*" abnormal conditions in the cervical region of the spine produce symptoms that can hardly be differentiated and any clinical division of the conditions into separate syndromes is purely artificial."* Which is why these different terms are more commonly included in the collective term, Thoracic Outlet Syndromes. Reference to T.O.S., or its more anatomically correct, but more rarely used title, 'Thoracic Inlet Syndrome, is extensively quoted in the literature and has created a plethora of suggested causes and concomitant treatment protocols. However as with so many such clinical entities the literature is full of conflicting views as to the best means of treatment. Its very existence is questioned by some.

[1] Figures1,2,3,4 & 8 are adapted from Walther. D lecture notes on Peripheral nerve Entrapment I.C.A.K-E Annual Meeting London 11.3.89 for which acknowledgement is herby given

[2] Hoag J. Cole W. Bradford Osteopathic Medicine 1969. p391

Howell[3] points out, in her excellent review of the subject that those who doubt the existence of TOS give as evidence:

(1) The lack of "Proof positive," since there is no single diagnostic test or battery of tests;

(2)The failure of surgical intervention to relieve the patient's symptoms consistently, as well as the high percentage of patients whose symptoms consistently reoccur post surgically;

(3)50% to 90% of these patients respond to physical therapy without surgical removal of a muscle and/or rib

Sucher[4] in his two articles on the subject, which, like Howell's, are amongst the best summaries on T.O.S. in the literature, outlines some of the controversy that still exists regarding the diagnosis and treatment of T.O.S. and why some authors[5] use the term "disputed neurogenic T.O.S."

ETIOLOGY

As the various names suggest the one thing that is agreed about this condition is that wherever the exact point of compression may be hypothesised it is a compression of the neurovascular bundle in the region of the thoracic dorsal outlet. Turk[6] divides the possible compression sights up clearly into four regions as they affect the subclavian and axillary artery and vein and the trunks of the brachial plexus as they pass from the neck to the arm.

(1) At the most proximal level the subclavian artery and brachial plexus may be constricted within the interscalene triangle where they emerge between the insertions of the anterior and middle scalene muscles near the first thoracic rib (see Figure 1)

(2) The next level of compression is in the interval between the first rib posteriorly and the clavicle and the Subclavius muscle anteriorly.

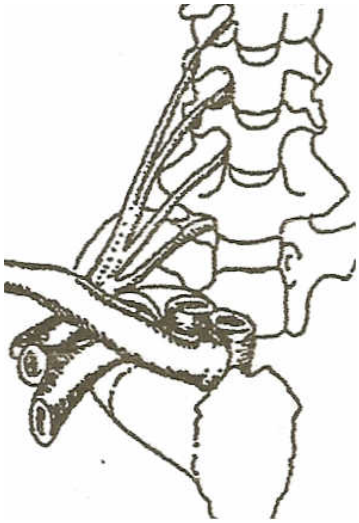

Figure 2. The clavicle, first rib and neurovascular bundle

(3) Then the neurovascular bundle passes beneath an enclosure formed by the coracoid, the pectoralis minor tendon and the costo coracoid membrane.

Figure 3 Pectoralis Minor with the neurovascular bundle passing between it and the coracoid process.

---

[3] Howell J.W. **Evaluation and Management of Thoracic Outlet Syndrome** p152 in Donatelli R. Physical Therapy of the Shoulder Clinics in Physical Theapy Series. 2nd Ed 1991

[4] Sucher B.M. **Thoracic Outlet Syndrome – A Myofascial variant Part 1. Pathology and diagnosis Part 2.** Treatment, Journal of the American Osteopathic Association Vol 90 No 8. August pp.686-704 & September 1990 pp.810-823.

[5] Cuetter A.C. Bartoszek D.M. **The Thoracic Outlet Syndrome: Controversies, over diagnosis, over-treatment and recommendations for management,** Muscle Nerve 1988 11: 66-74

[6] Turk S. Orthopaedics: Principles and their Application 4th Ed. Vol.2. 11984 p.890

(4) Finally, and more rarely given as an area of compression, Turk points to the scissor like encirclement of the axillary artery by the heads of the median nerve as constituting a potential constricting force.

Not unnaturally over time views have changed as to the importance given to the differing merits of the possible sights and causes of compression.

In 1905 Murphy described a case of cervical rib causing compression to the neurovascular bundle. In 1912 Todd[7] by measuring the inclination of the clavicle and first rib in *different* age groups and both sexes and relating this to the descent of the scapular in different circumstances identified scapular ptosis as a significant factor in the production of thoracic outlet syndrome. By 1927 Adson and Coffey[8] had already pointed out that while cervical ribs occurred in approximately 0.5 % of the population; of those only 5% to 10% ever become symptomatic.

The radiograph identification of the presence or absence of a cervical rib is now well understood to prove nothing. Where there is evidence of a cervical rib it may not be causing any problem and where there is none there may be fibrous bands not showing up on X-ray, which could be contributing to the compression. In this case, according to Travell & Simons, there may be an abnormally wide and long C7 transverse process, as long as, or longer than, that of T1.

Not unnaturally, over time, views have changed as to the importance given to the differing merits of the possible sights and causes of compression.

Of those T.O.S. cases that do go to surgery, only about 20%, Leffert[9] found, have osseous abnormalities. While it is obviously well worth palpating and X-raying for the possibility of an extra rib, or really, an unusually extended transverse process, evidence abounds as to the difficulty of linking the diagnosis to this anomaly.

While many have these anomalies few have the symptoms and when they do they can sometimes occur on the normal side. Grieve[10] points out that anomalous ribs can occur as high as C4 and, as he says, *"... the ribs and/or fascial bands must surely have a space occupying effect in some of the subjects, but not all come to clinical notice because of the symptoms reported. When they do, determining whether the rib is causing the patient's difficulties is not easy, as the anomalous structures vary in size, shape and relationship to the neurovascular bundle."*

By 1948, as Telford and Mottershead[11] pointed out, the old ideas of causation in this syndrome were being shown to

[7] Todd T **the Descent of the shoulder after birth** Anat. Anz 14:41 1912 quoted by Leffert D. in Rockwood & Matsen The shoulder Vol. 2 1990 p.768

[8] Leffert R. **Thoracic Outlet Syndrome** in Omer G. Spiner M. Ed.s Management of Peripheral Nerve Problems 1980 p.595

[9] **ibid**

[10] Grieve G.P. Common **Vertebral Joint Problems** 1981 p.131

[11] Telford E.D. & Mottershead S. **Pressure at the cervico-bracial junction** J. Bone Joint Surg. 30 B:249 1948

be less cut and dried than once thought. While the diagnosis of a pressure lesion of the cervical brachial junction was, to their mind, normally simple, at that time they felt the causes of such pressure to be so varied and obscure that, as they put it, *"... in the present state of knowledge it is not possible to forecast the exact cause in all cases. "*

DIAGNOSTIC TESTS

In the 44 years since then advances in diagnostic tests have occurred to make things somewhat easier. Sucher[12] gives a good overview of the anatomic and physiological diagnostic tests now available to us.

These include in the anatomical category; X-ray, C.T., M.R.I. and angiography/venography. In his physiological category he includes electoromyography and thermography as well as Doppler ultrasound and photoplethysmography. Naturally each has its limitations and advantages. Sucher makes a good case for thermography as often the tool of choice in T.O.S. The more invasive and dangerous examinations, such as angiography, are rarely justified in T.O.S. today. While different clinical availability of such tests will affect the practitioners' choice, they can clearly aid in differential diagnosis, build confidence in the diagnosis and provide useful before and after hard evidence in litigation.

Despite all the hardware, making the diagnosis of T.O.S. is notoriously difficult, consequently, over the years a range of provocative maneuver tests have grown up to help the examiner home in on the neurovascular compression, if that is what is causing the symptoms.

Of these tests the Scalenus anticus or Adson's test is probably the best known. But there is also the costo clavicular, hyperabduction or Wright's test and 3-minute elevated arm exercise test. However none of these tests can be taken as path gnomonic. For one thing it would appear that upward of 50% of the asymptomatic population will show a positive result to at least one of these tests as far as a reduction or loss of pulse is concerned. Even when it is remembered that diminished pulse and reproduction of symptoms makes for a positive test result, their predictive value is still in question.

When applied, the specific placing of the patient tested is important. However, it is worth realising that, while 90% of T.O.S. symptoms are neurological, many of these tests are primarily designed to test the effect of position on blood flow in the upper limb being challenged.

[12] Sucher B.M. **op.cit.**pp687-690

SYMPTOMS

Some like Stoddard[13] like to see T.O.S. as typically two syndromes.

One a costo-scalene syndrome in which the predominant symptom is pain in the ulna distribution of the forearm. The other a costo-clavicular syndrome in which, venous congestion and swelling of the hand together with paresthesia of the whole hand are the predominant symptoms.

Cyriax[14] points out that because the pressure may be slight and intermittent nerve conduction does not usually become fully impaired, rather, commonly the nerve may partially recover by night as fast as it is compressed by day with no loss of nerve function supervening. This is because the paresthesia is a release phenomenon. It may come on only when the downward pressure from the day's exertion is ended. Some hours can elapse until the paresthesia fully develops. Commonly it then wakes the patient often in the middle of the night requiring them to hang the arm over the side of the bed or sit or stand up to relieve the discomfort.

Typically the pain and paresthesia occurs on the ulna aspect of the hand and arm. In Cyriax's view the mere discovery of neurological signs at or above the seventh cervical level excludes pressure at the thoracic outlet as the cause. Roos[15], on the other hand, suspects upper plexus involvement in symptoms brought on in lifting or certain movements of the head. While aggravation of symptoms in shoulder depression, movements such as carrying are more likely to be lower plexus involvement.

Traditionally then, the typical picture of the T.O.S. patient has been a middle aged female (3:I female to male) with gradual onset or a history of trauma. This usually involving the head, neck and shoulder area with possible haematoma formation and resultant fibrosis. There may be excessive callus formation after a fracture of the clavicle or even a bifid clavicle, as well as the possibility of the, previously discussed, first rib. Poor posture, possibly brought on by depression or pendulous breasts, may be significant. Inflammatory or malignant disorders in the cervical spine or shoulder must be considered as well as a Pancoast tumour of the lung apex and the possibility of a cervical disc herniation, usually at C5.

Telford and Moottershead [16]show how abnormalities in the insertion of scalene anterior and medius can form V -type or the rare falciform type of insertion which are more likely in scalene anterior syndrome.

Treatment traditionally was normally confined to analgesics, heat and

[13] Stoddard A. **Manual of Osteopathic Practice** 1969 p.214
[14] Cyriax J. **Textbook of Orthopaedic Medicine Vol** 1 Ed8 1982 p.119

[15] Roos D.B. **The place for scalenectomy and first-rib resection in thooracic outlet syndrome** Surgery 92:1077 1982 quoted in Howell **op. ca**. p.166
[16] **Op cit.**

massage, shoulder shrugging and stretching exercises followed, if these failed, with surgery either aimed at decompression at the scalenes or more recently via the axilla to excise the cervical rib or fibrous bands and the first thoracic rib.

The last decade has seen a change in approach generally, possibly due to the disappointing results of surgery and recurrence of symptoms post operatively, as well as a greater awareness of the risks of surgical complication such as pneumothorax, and brachial plexus palsy. What has also changed is a greater awareness of the role of myofascial factors in the overall etiology and an increased sophistication of conservative methods in diagnosing and treating some of the underlying causes of this troublesome complaint

In the sources so far mentioned only Travell, Howell and Sucher have postulated a more dynamic functional cause for much of this, often career destroying, and at times, extremely painful and debilitating condition.

Applied Kinesiology has some of the answers to expand on the original work already done and make the diagnosis and treatment more effective still.

WHOLE BODY EVALUATION

As David Walther[17] so well outlined in his lecture on peripheral nerve entrapment, because of our intense focus on axial somatic dysfunction it is sometimes easy for osteopaths and chiropractors to overlook the peripheral component of nerve entrapment, be it in an osteofibrous tunnel, next to a sharp edge of a bone or through a muscle. Often if the spinal involvement only is adjusted, including any dural torque and the peripheral entrapment is ignored, as Walther reminded us, the patient will reach an improved plateau of health but the condition will not be completely corrected.

Nowhere more so than in T.O.S. does the concept of the "Double Crush Syndrome" or its myofascial equivalent of double entrapment, noted by Travell and Simons[18] serve to aid our understanding. Upton & McComas[19] hypothesised, on the basis of observed anomalies in Carpal Tunnel Syndrome patients that neural function is impaired because single axons, having been compressed in one region, become especially susceptible to damage at another site.

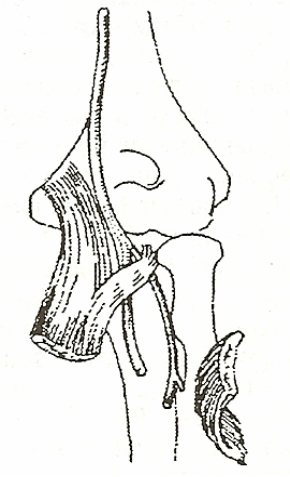

Figure 4 Pronator Teres Syndrome, an example of

[17] Walther D. I.C.A.K - E. Annual Meeting London.l1.3.89

[18] Travell J.G. Simmons D.G. **op cit**. pp.344-366

[19] Upton A.R. McComas A.J. **The Double Crush in Nerve Entrapment Syndromes** The Lancet August 18th 1973 pp.359-361.

As they point out, such an explanation can hardly involve impulse conduction which is an all-or-nothing phenomenon. Their explanation relies on the concept of impaired axoplasmic flow and the, as yet, not fully understood field of trophic substance flow, explored so well elsewhere by Korr.[20] To osteopaths and chiropractors there seems less to be surprised at in the concept that some nerve axonal narrowing might occur at more than one sight and that in some patients, especially those with a history of neck injury, the proximal lesion may have been excessive stretch, rather than compression of the nerve fibres.

What we have to keep in mind here is that anything that may have the effect of reducing the axoplasmic flow can make the nerve in question more susceptible to entrapment. That direct relationship between ischemia and neuropathy should be in our minds hence the importance of anything in the structure of the patient before us that can cause this lowered threshold to entrapment.

The papers on Thoracic Outlet Syndrome that come from the surgeons are understandably excellent in their grasp of the local anatomical factors, but from a conservative management point of view, far too little attention is paid to the overall body modular interaction and the entrapping potential such patterns can create. Often if left untreated these will allow the patient to go straight out and walk the problem back in immediately.

AK provides a rich source of evaluative tools in this area and such techniques as PRYT, Cloacal Synchronisation and Gait Analysis may give further insight. However if we fully screen and I'" all dysfunction throughout the stomatognathic and wider Cranio-sacral system and follow this up with detailed attention to possible secondary peripheral entrapment and then to the meridian system we should find that many of the other problems will already have been fixed except in the more stubborn cases. Walther[21] pointed out in his lecture that very rarely in the literature is any suggestion made to evaluate the pelvis, while it has been his finding that in some cases just treating a category 1 pelvis can take the torsion out of the shoulder girdle to such a degree as to reduce the pain in such problems as T.O.S.[22]or even Frozen Shoulder.[23] This latter condition has at times itself been linked

[20] Korr.I. **Neurochemical and neurotrophic consequences of nerve formation: clinical implications in relation to spinal manipulation (1915)** See The Collected Papers of Irvin M. Korr 1979.

[21] op.cit

[22] Walther D. Synopsis 1988. p.l06. (This. along with tbe present article. appears to be one of the few mentions of T.O.S. so far in AK literature. No doubt more will be forthcoming in Walther's volume on Orthopaedic conditions when that is eventually published.)

[23] For a discussion of Frozen Shoulder see; Lindley.-Jones C.S. Applied Kinesiology and the Frozen shoulder Syndrome I.C.A.K-E. Collected Papers Spring 1991 and Goodbcart.. G. The "frozen sboulder" Syndrome. Collected l'ape<s. pp.S6-58.

with T.O.S.[24]

Other factors to keep in mind while taking the history and when making your examination are such potentially aggravating factor as ileocecal valve problems and the bodies tendency to hold fluid to dilute toxicity, the structural and biochemical changes in pregnancy and lactation, not to mention the stress of lifting and carrying young babies, classic joint degenerative disease such as osteoarthritis, especially secondary to clavicular fractures or shoulder dislocation, diabetes, angina pectoris, rheumatoid arthritis, hypothyroidism, a bruit in the axilla, carpal tunnel syndrome, Pronator teres syndrome, acromegaly as well as the malignancy mentioned above or the herniated cervical disc.

EXAMINATION AND TREATMENT

The history will, in this condition, often give great help in guiding you to your initial hypothesis of T.O.S.

Howell[25] suggests, as frequently the primary complaint is pain, getting the patient to sketch a body diagram of the exact location of the pain/numbness and to highlight the areas that are worst can clarify the diagnosis.

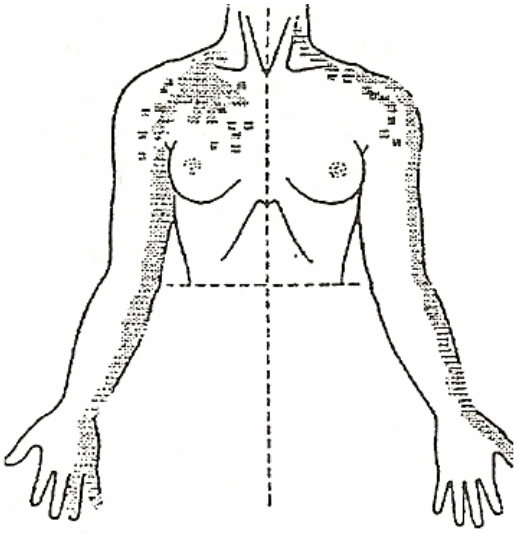

Figure 5 Upper plexus involvement seen on the patients left arm and hand. Lower plexus involvement, shown on the patient's right arm and hand

Typically this might look something like the figure above which shows the lower plexus pattern on the right hand with the upper plexus pattern shown on the left hand.

She also stresses the importance of a systematic plan for evaluation because of the multiple systems which may be involved in T.O.S. This might start with a general upper quarter screening, to rule out a cervical disc and nerve root compression, shoulder dysfunction, and peripheral neuropathies. From here one can move on to postural and respiratory observation.

As we have seen involvement of a unilateral shortened Anterior Scalene muscle is one common finding. Observing, therefore, for the appropriate body language would in this

[24] Hall H. Ed .**Rheumatology vol 1**.1983. p97.
[25] Howell J. op

case mean looking for any sign, on an anterior view, of the head side bent towards and rotated slightly away from the involved side.

While myofascial shortening seems to be most common in this condition, taking the view of hypotonicity often being primary in muscle dysfunction it is obviously always important to look at the antagonists and observe any body language which could suggest weakness. In this example the forward head position *might* also indicate weak cervical extensors. This in turn would make us suspect the possibility of fixations in the sacroiliac which is easy to therapy localise and see if this negates the weak unilateral neck extensors.

The ipsilateral sternocleidomastoid may also be involved along with the upper trapezius. If the upper trapezius is contracted this will effect the slope of the shoulders as well as the height of the scapula causing an elevation on the effected side.

If arm elevation is an exasperating factor in the symptom body picture, body language may well show the shoulder girdle rolled forward, particularly common in those with work related posture, such as; violinists or those working over a bench. With this forward roll of the shoulders there may be an accentuation of the thoracic curve. While this is commonly and rightly put down to a shortening of the pectoralis minor and medial shoulder rotators, this in turn may well be secondary to hypertonicity in the lower trapezius which has allowed the shoulder to role forward in the first place, as well as the elevated scapula and kyphotic dorsal spine. With this may go a hypertonic upper trapezius, which will invariably be tender and in turn raise the shoulder. The weak lower trapezius, of course, may, if bilateral, be associated with dorsolumbar fixations which must be dealt with first of all.

Postural examination can be one of the few clues to pectoralis minor shortening. Shoulder range of motion testing normally will not show this adequately. Observation of the supine patient from the head may show one shoulder more anterior than the other because the coracoid process of the scapula is being pulled inferiorly and anteriorly by the contracted pectoralis minor. Observation of the standing patient laterally may show a protracted shoulder girdle and increased dorsal kyphotic.

Beardall[26] however has suggested an alternative way to test pectoralis minor. This involves the supine patient flexing the arm on the shoulder 90 degrees, completely externally rotating the humerus, and keeping the elbow straight.

From there the arm is drawn across the chest towards the hip for the upper division and the knee for the lower division with the shoulder drawn

[26] Beardall A. **Clinical Kinesiology Vol.4. Shoulder Arm and Hand** pp.51-52

anterior and depressed 20 degrees. I have found using this method that some pectoralis minor muscles, if this is truly what is being tested, do show up being weak when other methods of testing fail to show this.

Travell's charts mapping the areas of referred pain from myofascial trigger points are useful guides to remember in examining patients with a possible T.O.S. She has also developed tests to determine whether pain is the result of active myofascial trigger points. These include;

1. THE SCALENE CRAMP TEST.

Figure 6 The subject rotates the head towards the side of pain, pulls the chin down towards the clavicle by flexion of the head. The trigger point will be aggravated by forced contraction of the muscle and the pain will be referred.

2. THE SCALENE RELIEF TEST.

If the subject is experiencing too much pain to perceive a difference with the previous test this is an alternative. The patient places the forearm against the forehead while raising and pulling the shoulder forward. This is designed to relieve pressure caused by the clavicle on active trigger points in the scalene muscles. When held for several minutes relief from pain should occur.

Inspection should always be made for vascular changes. A simple measurement that can be made is to compare the circumference of both upper limbs and inspection of the superficial veins in the arms and on the chest.

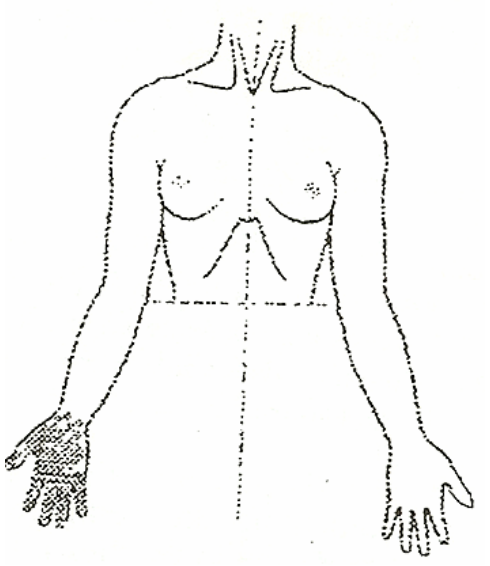

Figure 7 Compression due to Thoracic Outlet Syndrome whether of the cervical no or costo clavicular origin could manifest in hand in this way. Look for evidence of ischemia in one hand (e.g. coldness. discolouration, trophic changes). Clearly bilateral changes are more suggestive of classic Raynaud's disease.

Changes here could also be as a result of such things as chronic liver degeneration when the role of triglycerides and cholesterol needs to be considered in the patient's health.

If recurrent thoracic or cervical subluxations are found with dysfunction in the levator scapulae and rhomboids suspect dorsal scapula nerve entrapment in the scalenes. There will usually be tenderness in the lower part of the medial scalene muscle and pain radiating into the rhomboids. (See figure 8 over page)

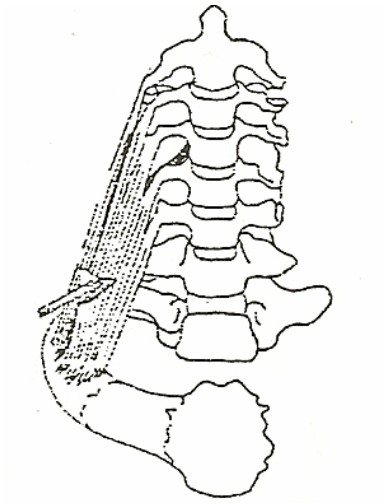

Figure 8 Showing possible sites for the entrapment of the Dorsal Scapula Nerve. Entrapment here can often cause recurrent thoracic subluxations due to its effect on the rhomboids and cervical subluxations via levator scapulae.

One great advance that Applied Kinesiology has offered us is greater accuracy in getting more of the whole picture by a detailed use of postural analysis and careful in-depth use of manual muscle testing.

For example, as mentioned above in the case of pectoralis minor syndrome, we could expect to find a tender coracoid process (also a possible active neurolymphatic reflex to supraspinatus). From there we could then go on to examine for a dorsolumbar fixation from a weak lower trapezius which may have been instrumental in allowing the contracted pec. minor in the first place. But we can then go on to check for any bilaterally weak Pectoralis major clavicular muscle which might be masking the D/L fixation in the first place.

A contracted pec. minor may also be contributing to lymphatic congestion affecting the immune system and possibly creating lung problems, and a tendency to frequent colds. If we have checked for retrograde lymphatic problems it is also worth remembering Goodheart's observation that bruxism is sometimes associated with this condition, and so we move our attention to the stomatognathic system. In this way our hunt for the trail of dysfunction can take us all over the body in less time than it takes to write about it.

Almost nowhere else in the literature is any mention given to a detailed evaluation of the state of synergists and antagonists and their proprioceptive function. To give her credit, Howell[27] gives the most detailed non-AK approach to manual muscle testing. While only osteopaths Barral and Mercier[28] have noticed that it is not only the thoracic inlet changes that can influence Adson's and Sotto-Hall's test. In one experiment a dramatic increase in the right radial pulse as measured by Doppler was observed following manipulation of the liver! Howell[29], who writes as a physical therapist, rightly observes that the theory of myofascial pain has not been

[27] Howell J. **op. cit.** p171
[28] Barral J-P. Mercier P. **Visceral Manipulation** 1988 p.50
[29] **Ibid**. p170

adequately explored in T.O.S. patients. However Sucher's[30] articles in 1990, have done an excellent job in rectifying this lack.

Upledger[31] points out the complexity of the anatomy of the thoracic inlet, as he says, "*the potential for reduction of fluid flow, restriction of fascial mobility and osseous somatic dysfunction or restricted motion at the thoracic inlet is immense.*"

Elsewhere[32] he gives a more detailed analysis of the fascia of this area of which there is not space to go into here.

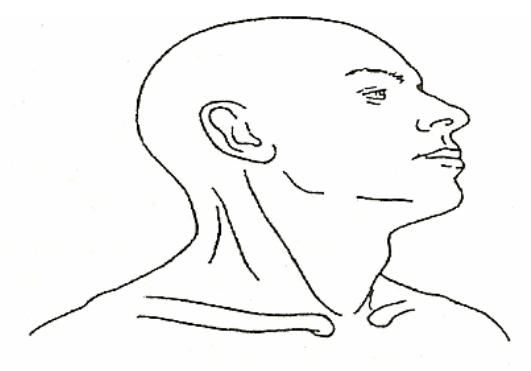

Figure 8 The Thoracic Inlet

Suffice to note that, as Upledger[33] shows, the investing fascia of the area is really part of one continuous fascial sheet so that hyper tonus of any one of the a number of these muscles affects the fascia in such a way as to result in an imbalanced, hypertonic and restricted condition of the whole thoracic inlet.

It is worth noting here that the prevertebral fascia, which attaches to the base of the skull, descending downwards ultimately to blend with the fascia of the trapezius muscle, does not ensheath the subclavian or axillary veins and therefore does not cause venous congestion directly.[34]

Cranial osteopathic unwinding of thoracic inlet is worth employing here, which can often give greater insight into the nature of the restrictions focused in this area. This involves an AP contact over the inlet, inferiorly between the spinous process of C7 and the upper few thoracic vertebra and superiorly over the anterior superior thoracic wall to take in the sternoclavicular joints and the supra sternal notch and upper Costachondral region. A light (1-2kg) pressure is induced until any shearing or torsional motion in the tissues is perceived. This is then followed but not allowed to return in the direction it came. The motion is gently followed in whatever other path it goes in until resolution is felt.

Hutchin[35] has made a very useful observation in relations to this area in her findings regarding the fixation patterns observed in the manubrio-sternal joint and its 'Lovett brother; relationship to the xiphisternum. Given its importance in relation to the thoracic outlet, posture and respiration, it is not difficult to see

[30] Sucher **op. cit**
[31] Upledger J.E. **Cranialsacral Therapy** 1983. p.54
[32] Upledger J.E. **Cranial sacral Therapy II: Beyond the Dura** 1987.p115-129
[33] **Op. cit** 1983 p.53
[34] Cailliet R. **Shoulder Pain** Ed. 2 1981. p97
[35] Hutchin, A. **Fixation of the Manubriao-Sternal Joint and its Lovett Brother Relationship to the Xiphi-Sternal Joint** I.C.A.K.-E Collected Papers Spring 1991. pp.128-130

how a fixation here at the sternal joints, which often presents with bilateral weakness in subscapularis, could have important impact on normal motion in the area and negatively effect the thoracic outlet.

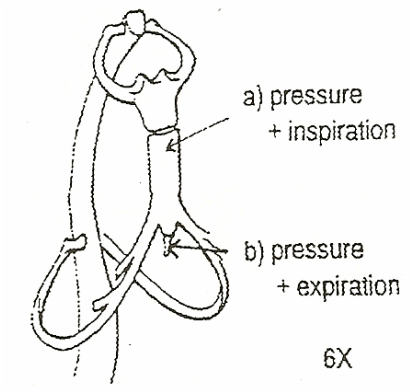

Figure 10 The manubrium can be fixed in both inspiration and expiration. For treatment of this condition see article (footnote 35)

A further advantage of Hutchins's discovery is to awaken us to this fixation's propensity to, at times, mask a sphenobasilar compression pattern.

If it is true that. when the cranial mechanism is compromised the body is forced to increase diaphragmatic-costal respiration as a back up, as Klapper[36] reminds us, then it is not difficult to see how fixation at the manubrio and xiphi-sternal joints might, in fact, be masking the all important sphenobasilar compression lesion.[37] Given its high priority as a primary structural lesion effecting so much else, finding it masked or recurrent through such a fixation, may be crucial to further unraveling the thoracic inlet fully. This can also be said for the other common primary cranial lesions introduced to AK from Sutherland's work by Smith.[38]

Howell[39] has made a useful aide memoire for us in searching for myofascial trigger points, which may often be the cause of a lot of the pain in T.O.S.

Using Roo's concept of upper and lower plexus involvement she divides them as follows.

Muscles that refer myofascial pain to mimic thoracic inlet syndrome Upper and Lower Plexus symptoms patterns.	
Upper Plexus	**Lower Plexus**
Scalene anterior	Pectoralis minor
Subclavius	P.M.S.
Levator Scapulae	Upper trapezius
P.M.C.	Triceps med. Head
S.C.M.	Subscapularis
Infraspinatus	Pectoralis minor*
Multifidius	
Scalene anterior*	
Medial scalene*	
Subclavius *	

Figure 11.

*** Muscles classically implicated in compression of the brachial plexus and subclavian vessels, which results in upper and lower plexus symptom patterns.**

While not mentioned here by Howell, we should not forget the omohyoid muscle. It could cause some problem as it comes up from its attachment on the

[36] Klapper G. **Identification and Correction of the Primary Cranial Lesions** Proceedings of the Summer Meeting fo the I.C.AK-U.S. vol1 1991-2 p.88

[37] For the first introduction to the diagnosis and treatement of this vital concept of Sutherlands into Applied Kinesiology see Smith C.R. **The Role of the Osteopathy in Applied Kinesiology** Collected Papers of the I.C.A.K.-E Spring 1988 (Klapper's article seems rather later on the scene with this useful addition!)

[38] Smith C.R. **Vertical and Lateral Sphenobasilar Strains (subluxations)** I.C.A.K.-E Collected Papers Spring 1989 pp.25.26

[39] Howell **op. cit** p.171

cranial border of the scapula near the coracoid process to the attachment by the central tendon on the clavicle and from there superiorly onto the hyoid.

When tense, it can be mistaken for the upper trapezius or scalenes and can prevent full stretch of these muscles. The omohyoid also acts synergistically with the upper trapezius and levator scapulae in elevating the shoulder girdle off the chest wall.

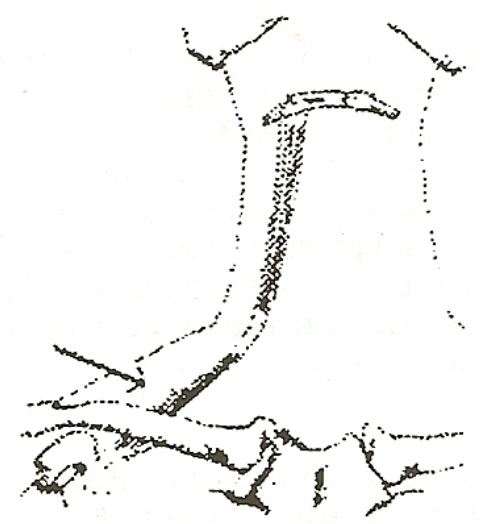

Figure 12 Omohyoid

As Walther[40] has outlined, when a muscle that tests strong in the clear is stretched and then re-tested, it tests equally strong or stronger because of facilitation by myotactic reflex. However a positive muscle stretch reaction is found when a previously strong muscle is stretched and then tests weaker. This disharmony is normally associated with the associated fascia or with trigger points, those small hypersensitive spots from which impulses bombard the central nervous system and give rise to familiar referred pain.

Due to their role in pumping blood and lymph there is an active relationship between neurovascular and neurolymphatic reflexes and the positive muscle stretch reaction. When there is a need for fascial release, these reflexes do not usually have a positive therapy localisation however, when therapy localised whilst the muscle is being stretched, the positive stretch reaction is abolished. It has been noticed that when the muscle and its fascia are treated there is often an improved function in the associated organ or gland. Conversely, improved results in myofascial problems may be obtained if the associated reflexes are also stimulated.

Fascial release treatment is well known in AK and should normally provide little problem for the experienced practitioner. However, in T.O.S. cases, pain or restriction of movement of the shoulder can prove some handicap.

Sucher[41] goes into great detail in the second of his papers as to the importance of good patient education and therefore compliance, in effective management of myofascial release. This is because both active stretch by the patient at home and passive stretch and myofascial release by the practitioner can be painful in the early stages with a structure that has been contracted for some time.

Possibly, if one could treat the patient early, especially after trauma, some

[40] Walther D. **Synopsis** 1988.p.169

[41] Sucher B. **op.cit Part 2** pp.820 -823

T.O.S. cases might be able to be aborted. The larger, more superficial, muscles, such as the rhomboids, trapezius and levator, may well be the first to contract after a simple cervicothoracic strain. With this come gradual tightening and protraction of the shoulder with the deep smaller muscles contracting and developing trigger points and fascial shortening adaptively.

Whether one employs classic Travell spray and stretch;

(Stretch is the ACTION, spray is DISTRACTION[42])

Or Sucher's adaptation of this, regular patient stretching at home between treatments should be encouraged. This will most likely be painful at first, but should leave no residual pain when it is discontinued. If the T.O.S. has been of some months or years duration, a vicious circle may have been established. This feedback loop starts with shortening and contracture leading to pain, leading to lack of use and guarding and restricted movement, which in turn leads to further shortening, resulting, in what Sucher[43] outlines as, an abnormal muscle engram. He points out how hard that is to change. This is for two reasons:

First, because it is difficult to change the muscle length because of the recoil tendency of contracted connective tissue. This needs to be overcome by encouraging the process of 'plastic deformation or creep' via passive and active stretch and other myofascial treatment.

Second, unless it is sustained, deep and frequent, the new reprogramming will not be enough to change the old myofascial-spinal-cerebral engram.

The majority of symptoms T.O.S. patients present with are neurological. Of these, sensory rather than motor symptoms predominate. One way of mapping out your progress during treatment may be the use of Tinel's sign. Some authorities suggest that, while *local* pain produced by pressure applied to a nerve is a sign of nerve irritation, pressure on the nerve resulting in tingling felt in the *distribution* of the nerve is a sign of axon regeneration.

Other evaluations used at times include mapping of paresthesia with Semmes Weinstein monofilaments, skin temperature mapping, volumetric and circumferential measurements and cold recover time.

However, to those schooled in manual muscle testing the few upper limb motor signs sometimes present will come more naturally.

If the compression is of some duration or of considerable severity some of the muscles supplied by the lower trunk may be atrophied.

---

[42] Travell & Simons **op. cit** p.64

[43] **Ibid.** p67

Turk[44] has observed the most commonly involved muscles to be interossei and the lumbricals with less commonly the thenar muscles, especially the abductor pollicis brevis and opponens pollicis.

NUTRITION

Nutritional deficiency may be important when Raynaud's phenomenon is a particular part of the problem. Check for dry eyes and moth and dry flaky skin which can be a sign of essential fatty acid deficiency.

Marsden[45] observed that work by Belch and colleagues[46] found that using evening primrose oil in a double blind trial on Raynaud's disease patients reduced the symptoms of Raynaud's attack helping to reduce blood viscosity by lowering the levels of thromboxanes in the blood. Thromboxanes, while being useful in encouraging the blood to clot on injury, can have the effect of reducing circulation and cause the spasm associated with Raynaud's phenomenon.

Dietary changes that might help could include the use of GLA, increased pure water, reduction of poor dietary intake with more whole food rich in essential fatty acids such as the liberal use of extra virgin olive oil or better still, linseed oil, walnuts, hazelnuts and chestnuts. A good intake of EFA-rich fish like salmon, sardines and mackerel with vitamin E as synergistic support for this increased essential fatty acid intake, may help, in part, to reduce some of the Raynaud's phenomena and possibly the ischemia and therefore enhance the chances of good axonal transport so reducing risks of double crush entrapment. Linseed or Flax seed oil (the Latin name for flax is 'Linum usitatissimum', which means 'most useful') **is** fast being recognised as the world's richest natural food source of linolenic acid as well as phosphatides, lecithin and the precursors to PGE 3. As such it is rapidly superseding even the more popular evening primrose oil as the essential fatty acid of choice.[47]

CASE HISTORY

As so often happens when one focuses on something, very soon one's practice starts to fill with the very subject of one's enquiries. In part this no doubt is due to raised awareness picking up the problems one had previously failed to diagnose correctly. However, it is hard not to see some more subtle plan at work, directing those in need of help to one's doorstep.

This has certainly been the case with T.O.S. and my practice. Since I began to ruminate about it for this article, numerous patients seem to have sprung out of the wood work, walking examples of the commonness of this painful condition.

The case of Mrs. P., a 67 year old

[44] Turk S. op.cit. p.901

[45] Marsden K. **Raynaurd's Disease : a case history** Int J. Comp Med Dec 1991 p.19

[46] Belch J. Shaw B. O'Dowd A. Saniabadi a. Lieberman P. Sturrock R. & Forbes C **Thrombosis Haem.** Vol. 54 pp. 490-494

[47] Erdmann R. & Jones M. **Fats, Nutrition and Health** 1990 p.103

retired Head teacher, is typical. She presented with right arm pain and dysfunction of five years duration. The pain was difficult to fully pin down. She felt it started in the shoulder region and extended to the right hand into the 1-3 fingers. On certain movements she would get pain into the biceps and down into the forearm and fingers, often with decreased power in her grip, leading to a tendency to drop things. To gain relief she had at times resorted to hanging her arm out of the bed because of the pain.

She noticed it was increasingly difficult to manage certain movements such as swimming, driving, knitting, writing etc.

The problem seemed to have started subsequent to a fall five years ago onto her right shoulder and had been aggravated more six months before coming to see me, by two further falls, when again her right shoulder was involved.

Typically she began to think that nothing could be done and that she would have to accept these considerable restrictions on her life as the price of advancing years. As she later confided to me, she only sought help to placate the promptings of her friend, another former patient.

On examination, she showed numerous hypotonic muscles in and around the shoulder and a range of typical related problems.

Upper trapezius, supraspinatus, pectoralis minor, triceps, psoas, the scalenes, pronator teres (ulna division) omohyoid, flexor carpi radialis longus and brevis, and coracobrachialis, all showed up as hypotonic over the weeks of treatment, along with the dorsal scapular nerve trio of rhomboids, levator scapula and serratus anterior.

Cranial faults compounded the problem as would be expected, with sphenobasilar compression and vertical and lateral shear patterns present. As you might expect, the kidney meridian was low, causing upper trapezius and psoas weakness, destabilising both ends of the spine. Double crush-like patterns were present in the elbow, caused by the weakness of flexor carpi radialis longus and brevis, leading to pain on the lateral epicondyle, while pronator teres syndrome was evident in the forearm.

Lung meridian involvement was affecting the deltoids, serratus anterior and coracobrachialis. Notably, with the latter, the patient volunteered the comment, that she "had to tell the arm to brush my hair".

Good improvement was seen after four treatments, attending to the above in the normal manner. The patient also undertook home stretching exercises. It was not until after several more treatments, however, that the, now obvious fact of her hypertonic pectoralis minor, became clear. This was due to a spleen meridian imbalance, causing a weak right lower trapezius allowing the pectoralis minor to be less effectively opposed in its anterior draw

on the scapula.

Further imbalance in the neck was caused by right neck extensor weakness secondary to a right sacro-iliac fixation. Usually the last part of the puzzle to go involves releasing the neurovascular bundle sufficiently to allow the patient to, once again, carry heavy weights or do back stroke while swimming, or any other backward extension of the arm. However, as a keen knitter and swimmer, as well as a woman with a good sense of humor, Mrs. P. proudly told me recently that, not only could she do both these occupations again, but she could now even do them simultaneously!

CONCLUSION

Our job then, is to so change the tissues and their neuronal feedback that the old pattern has no chance to assert itself, but rather the body has been given back to itself in its pre-contracted neurovascular bundle compression state.

To this end the skilled AK practitioner should be able to demonstrate to the patient how the pain is aggravated and how to reduce it. As well as this, if patient myofascial stretching is felt to be required, then careful coaching of the patient is needed. This is for two reasons.

First, because a poorly done stretch produces poor results.

Second, because, in a condition of longstanding pain, demonstrating out the patient that the discomfort in stretching these contracted structures can be a 'good' and productive pain, is the only way to get willing co-operation and confidence in the treatment

Finally, in cases where there is a large postural component, postural re-education may need the skills of a teacher trained in the Alexander technique to ensure long-term freedom from recidivism.