



# Postural cues for scapular retraction and depression promote costoclavicular space compression and thoracic outlet syndrome

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## ABSTRACT

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A commonly used postural corrective measure is to pull the shoulders back and down. This corrective measure is most likely based upon the idea that postural acromial protraction is a frequent tendency in neck and shoulder patients, as is excessive clavicular elevation during shoulder movement. However, this corrective measure is based upon logical fallacies, firstly because it will cause scapular depression and downward rotation, which has been associated with scapular dyskinesis (SD), shoulder impingement syndrome (SIS) and neck pain. Secondly, biomechanically it will set the patient in the Halstead's costoclavicular compression ("military brace") test position, which may result in plexopathy and thoracic outlet syndrome (TOS). The corrective measure thus opposes what it is intended to do, as it may exacerbate neck and shoulder problems rather than ameliorating them. Based on the anatomy and evidence, as well as personal clinical experience with 115 TOS patients, it is my impression that the cue in question is harmful and that its usage should be discontinued. Conversely, the patient should be cued to raise his or her scapulae until the superior scapular angles are levelled with the T2 vertebra, and learn to stay there, as this will upwardly rotate the scapulae as well as decompress the costoclavicular space.

**Key words:** Thoracic outlet syndrome; Costoclavicular space syndrome; Scapular posture; Shoulder impingement syndrome; Scapular dyskinesis

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## INTRODUCTION

The notion that proper scapular posture involves pulling the shoulders "back and down" is widely accepted and practiced by several current musculoskeletal practices, as well as by exercise trainers.<sup>1,6</sup> However, pulling the clavicle back and down may compress the costoclavicular space and cause thoracic outlet syndrome: In fact, the Halstead's "military posture" stress test, a provocative test for costoclavicular space (CCS) compression, involves these exact clavicular movements.<sup>7,14</sup> Intentional scapular depression also promotes downward rotation and anterior tilt, which has been associated

with neck pain, scapular dyskinesis (SD), shoulder impingement syndrome (SIS),<sup>15,35</sup> and neck pain.<sup>6, 35,39</sup> The aim of this article is to look at the origin of the "back and down" postural corrective measure, demonstrate its harmful implications, and to provide alternative criteria for assessment and correction.

The notion itself most likely originates from earlier studies, mainly those addressing SD and SIS where it was shown that patients with these afflictions tend to have an anteriorly situated clavicle/acromion in posture,<sup>31,34,40</sup> implying scapular anterior tilt, downward rotation and protraction. Earlier studies have also shown that patients with SIS have a tendency of clavicular elevation as well as scapular

downwards rotation and anterior tilt on the affected side during glenohumeral articulation.<sup>24,29,34,35</sup> It has also been documented that patients with neck pain have a postural tendency of anteriorly positioned clavicles with downward scapular rotation.<sup>6,35,39</sup> Some of these authors recommended postural correctives, which involved scapular retraction.<sup>40,43</sup>

Since the literature suggests that anterior drooping in posture, as well as anterior tilt and downward rotation during shoulder flexion and abduction, one could recommend some degree of scapular retraction as a corrective measure. However, stemming from the powerlifting and fitness communities,<sup>1,5</sup> depression of the scapula was also included in this corrective strategy. As mentioned, scapular retraction and depression may promote CCS compression as well as SD with concomitant SIS. Therefore, it would seem that this corrective intervention is not based upon evidence nor sound biomechanics.

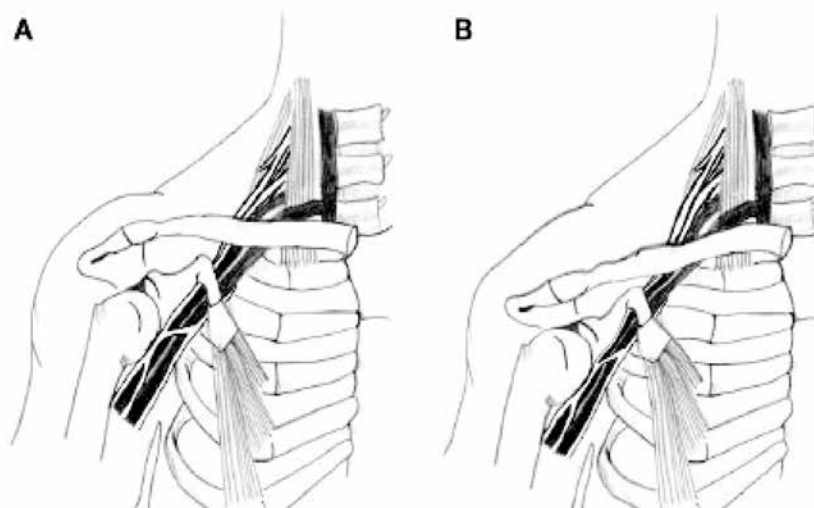
## SCAPULAR RESTING POSTURE

Researchers have estimated that optimal longitudinal resting position of the scapula is when the superior scapular angle is levelled with the T2 spinous process, 0-5° of upwards rotation and approximately 20° of clavicular retraction and 20-25° of upwards clavicular inclination.<sup>6,10,11,14,16,34,44,48</sup>

By pulling the scapulae back and down, the only criteria that will be met as regards optimal position, will be retraction. It will also cause depression, anterior tilt (from squeezing the shoulders together,<sup>11</sup> and downward rotation, which completely opposes the corrective's original purpose i.e. to increase upwards rotation and posterior tilt, as well as retraction. Either way, this may cause continuous postural lengthening and inhibition of the trapezius and levator scapulae, often resulting in cervical stiffness,<sup>49</sup> cervical myofascial tenderness and pain,<sup>6,15,37-39</sup> scapular dyskinesia and shoulder impingement syndrome,<sup>6,24-29,34,35,40</sup> and rotator cuff injuries.<sup>30,33</sup>

Although quite troublesome, shoulder and neck pain, etc. are still considered of lesser significance compared to costoclavicular compression syndrome, which normally involves compression of the brachial plexus, subclavian artery & subclavian vein between the clavicle and 1<sup>st</sup> rib, but may also occur against the 2nd rib.<sup>50</sup> Retraction and depression of the scapulae may lead to osseous compression of the costoclavicular space,<sup>7-14</sup> as it quite literally puts the patient's clavicle in continuous Halstead's costoclavicular compression test ("military brace") position, often resulting in thoracic outlet syndrome (TOS).

It is well documented that TOS patients tend to have depressed scapulae, along with anterior tilt,<sup>10-14,44-46,51-60</sup> which further depresses the clavicle. Watson states that scapular depression along with dyskinesia is commonly seen in TOS patients, and that this encourages costoclavicular compression.<sup>11</sup> Elevating the scapulae will decompress the CCS,<sup>58</sup> which is also an orthopedic test called the Cyriax test.<sup>8</sup> This is one major reason why TOS surgery involves resection of the 1st rib; to free the neurovascular bundle within the CCS. It is therefore of paramount importance to ensure that patients do not overly depress their scapulae, to avoid oblivious CCS compression. Figure 1 illustrates how depression and retraction of the clavicle may compress the neurovascular bundle. Contrarily, lifting the clavicle will decompress the neurovascular bundle.



**Figure 1: Costoclavicular space compression (Image source: Watson et al., 2010)**

## **COSTOCLAVICULAR SPACE COMPRESSION**

Compression of the neurovascular bundle (NVB) of the thoracic outlet may lead to plexopathy, muscular atrophy,<sup>61-65</sup> carpal tunnel syndrome (double crush),<sup>65-68</sup> chest pain, ulnar neuropathy,<sup>65-72</sup> dorsal scapular neuralgia<sup>73,74</sup> arm swelling and cyanosis.<sup>75,76</sup> Other symptoms are Raynaud's syndrome,<sup>75,76</sup> positional ischemia or venous insufficiency,<sup>69,72,77-80</sup> which may contribute to intracranial hypertension<sup>83</sup> and migraines in addition it may present as digital sensory loss,<sup>72</sup> hand sweating and coldness,<sup>98-99,107-108</sup> secondary dysautonomia such as atrial fibrillation and vasoconstriction,<sup>81,98-103</sup> deep vein thrombosis (DVT) development,<sup>79,88-93</sup> weakness of the extremities, chest pain and pseudoangina,<sup>69-71,88-90</sup> subclavian artery injuries with subsequent embolus<sup>73,96</sup> which may lead to retrograde thromboembolism with sequela stroke,<sup>91-93,97,98</sup> and more.

Because of the vast array of symptoms that may appear in the sequela of TOS development, it may be some time before these patient presents with conspicuous TOS symptoms and get properly diagnosed. TOS may reveal itself with any of the above-mentioned symptoms. It is well known that these symptoms are not unique,<sup>79,99</sup> and it is hard to diagnose and treat the condition.<sup>78,100-104</sup> This is one of the main reasons why [occult] TOS may be misdiagnosed as carpal tunnel syndrome, ulnar neuralgia, idiopathic chest pain, etc., in its beginning and intermediate stages. Neurographies (MR Neurography imaging (MRN), electromyography (EMG), electroneurography (NCV), somatosensory evoked potentials (SEP) are insufficiently sensitive for detection of TOS, and may only be positive in very advanced stage.<sup>72,105-112</sup> Rousseff et al. (111) states that EMG / NCV is useless for identification of TOS, as 18 out of 20 patients with very obvious TOS symptoms had normal electrodiagnostic results.

A systematic review conducted by Kwee et al.<sup>112</sup> concludes that MRN is not sensitive nor specific enough for detection of brachial plexus neuropathies nor other peripheral neuropathies. This further complicates the likelihood of these patients (i.e. a victim of the "chronic Halstead's maneuver") to be properly diagnosed and treated.

It is a misfortune for patients to be iatrogenically set into the "back and down" scapular posture, as they may not be diagnosed until many years later due to the diffuse presentation of TOS.

## **CLINICAL PRACTICE & APPROPRIATE CORRECTIVES**

In this section I will provide some criteria for identification of TOS as well as scapular resting dysfunction. Neurogenic TOS is the utmost common variant, which makes up approximately 95% of total TOS incidences.<sup>78,79,113</sup> Because the inferior trunk lies more susceptibly (anteriorly) placed in the CCS, symptoms of C8-T1 (ulnar) neuropathy may appear first. However, the superior trunk (C7) and middle trunk (C5-6) may be affected, especially in more progressed cases of TOS.<sup>65,72</sup> Supraclavicular tenderness (Morley's test) and weakness of the 5<sup>th</sup> finger are sensitive, and relatively specific tests for thoracic outlet syndrome.<sup>69-71,78-79,99,100,113-116</sup> Weakness of the triceps (C7 myotome) is also common. There may also be positional ischemia upon shoulder elevation, demonstrated by a white hand sign,<sup>69-71,116-119</sup> indicating severe compression of the subclavian artery.<sup>59,88,116,120,121</sup> One test alone, e.g. Adson's or Roos' test, may not be specific enough to diagnose TOS, especially because only 5% of TOS incidences are considered to be of vasculogenic dominance.

Compression of the CCS may occur related to posture or intermittently during certain activities. For example, a patient may have a seemingly normal scapular resting height, but still have a tendency of pulling their scapulae back and down during exercise, or have scapular dyskinesis, which may lead to intermittent compression of the neurovascular bundle. It is important to examine the patient's scapular position as well as loaded and unloaded movement pattern in different scenarios. In addition Halstead's CCS test may be used during the examination. A detailed explanation of identification and correction of scapular dyskinesis is outside the scope of this article.

The longitudinal scapular height can be measured by palpating the C7 (vertebra prominens) spinous process, then counting down to the T2 level, and comparing it to the level of the superior scapular angle. Up-/downwards rotation can be measured vertically by evaluating the angle of the medial scapular border, or horizontally, the angle of the scapular spine. The scapula is in downward rotation if the spine is pointing caudally, or if the superior angle is lateral to the inferior angle (sagittal axes). Anterior-/posterior tilting can be evaluated by measuring how far anterior the acromion is in relation to the inferior scapular angle, in the sagittal plane (coronal axis). An inclinometer, which in modern times is available

for download on any smartphone, can also be used reliably to measure scapular angulation.<sup>11,23-26,40,122-128</sup>

The Cyriax release test is another orthopedic test which relieves the CCS by elevating the clavicle.<sup>8</sup> Thus, postural CCS compression may be ameliorated by raising the shoulders slightly,<sup>58</sup> and staying there. This will also upwardly rotate the scapula,<sup>55</sup> which is important for SD and SIS treatment. To correct scapular slouching, ask the patient to lift their acromion until the clavicle is elevated and superior scapular angle is approximately levelled with the T2 vertebra, and the scapula is in mild upwards rotation. The patient must be educated with regards to the etiology of costoclavicular space compression syndrome, so that they become sufficiently motivated to maintain their newly acquired posture. Further, he or she must learn to maintain adequate scapular height during glenohumeral articulation.

Figure 2 shows a 25-year-old patient with chronic brachial, periscapular, chest and neck pain. She had been vigorously pulling her scapula back and down to “relax” her shoulder girdle, inevitably worsening the situation. Selmonosky’s DT was positive, as was Halstead’s CCS maneuver. Both scapulae were situated at the T4 vertebral level; very depressed (Figure 2, left). The left scapula was slightly more depressed than the right one and more caudally rotated as well. There was also bilateral scapular dyskinesia present during movement and loading of the arms. After identifying the scapular depression, the patient was cued to lift her acromion while slightly elevating the



**Figure 2: TOS patient with severely depressed scapulae, before correction**



**Figure 3: TOS patient after correction**

scapulae towards the back of the head, as to promote scapular elevation and upwards rotation with slight retraction, until the scapular angle was parallel with the T2 spinous process. She was told to stay there (Figure 3), and we also practiced moving and loading the arms while maintaining proper scapular height and angulation. The reason for cueing the patient specifically to lift the acromion rather than just the scapula, is that this promotes upwards rotation due to upper trapezius engagement. Some patients may unknowingly lift their scapulae up and forward by engaging the levator scapulae muscle, with sequelar downward scapular rotation. The patient experienced almost immediate remission of her symptoms that were related to loading and articulation of the arms after learning to hold her shoulders up. Also note the trapezius hypertrophy, although the muscle is clearly not over-engaging in lifting the scapulae in the “before” image. This can be misleading, which is why scapular height must be measured rather than ‘eyeballed’.

## DISCUSSION

It has been suggested by several authors that the upper trapezius is overactive in patients with SIS and SD, based on EMG test results during glenohumeral articulation.<sup>17,44,47,129-131</sup> Yet, despite this, a conspicuous pattern of scapular downward rotation, anterior tilting and protraction is demonstrated in the very same patients. Because the UT promotes upwards rotation and posterior tilting, as well as retraction,<sup>15,35,132</sup> the notion that the UT is truly

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over-engaging in scapular movement does not seem likely. Contrary to this, the levator scapuli muscle will cause scapular elevation, protraction, downward rotation and anterior tilting,<sup>132-133</sup> and is, therefore, a culprit more compatible with the evidence, than that of the UT. Moreover, regarding the authenticity of the documented excess UT EMG results, some studies show that hypertrophied muscles with high EMG output on the symptomatic side (in this case, the symptomatic sacroiliac side), was later proven to be significantly weak,<sup>134-136</sup> and that the excessive EMG output signal normalized after increasing the respective muscle's strength. This may suggest that it is not as black and white as the studies which demonstrate UT overactivity suggest; it may still be weak. A weak UT attempting to stabilize and move the scapulae properly, yet fails to do so, may explain why we see increased EMG signals in the UT yet movements which contradict its true involvement, i.e. movements of downward rotation, protraction, depression, which would not be reasonably present if there was legitimate over-engagement of the UT in scapular movement.

Other researchers suggest that in order to maintain proper scapular position during glenohumeral articulation, all of the stabilizers must engage, especially the UT, SA, middle trapezius and lower trapezius.<sup>11,60</sup> It has also been stated that a common error is to over-engage the rhomboid by squeezing the shoulder blades together, as this will promote anterior scapular tilt with concomitant depression of the scapula by the latissimus dorsi muscle.<sup>11</sup>

In accordance with Watson and Mckinnon's papers,<sup>11,60</sup> I have experienced that by setting a patient into the norms that were provided by Sahrman and others,<sup>6,10,11,14,16,34,44,46</sup> one will quickly realize that most patients with neck and shoulder problems tend to have depressed scapulae, and that they need to engage their upper trapezius, not suppress it. A main muscle that retracts and upwardly rotates the scapula, is also the upper trapezius muscle. Most of the negative biomechanical associations (scapular downward rotation, anterior tilt, protraction, depression) with SD, SIS, and TOS, are functionally countered by the upper trapezius, which promote elevation, retraction, posterior tilting and upward rotation. Based on this, and the evidence considered, it would seem quite contraindicated to pull the scapula back and down. It has been demonstrated that scapular elevation has an immediate beneficial effect on cervical pain<sup>137</sup> as well as range of motion.<sup>138</sup> Further, it has been documented that subjects with lower scapular resting position have a tendency of higher pain thresholds in the upper

trapezius.<sup>52-53</sup> Whiplash associated disorders (WAD) patients have also been demonstrated to have low-riding clavicles.<sup>15,37</sup> And, it has been demonstrated that patients with slouched scapulothoracic postures have decreased shoulder abduction ROM and posterior scapular tilting as well as decreased muscle force in glenohumeral abduction above 90°. <sup>49</sup> Finally, it has even been documented that patients with upper extremity deep vein thrombosis have a significantly narrower costoclavicular space in resting posture than that of controls.<sup>94,95</sup> These would all make some solid points against pulling the clavicles back and down as postural means of therapeutic intervention.

Between January 1st 2017 and June 20th 2018, I evaluated 115 TOS patients for the co-presence of scapular depression, at my clinic in Oslo (Norway). They were diagnosed based on the criteria provided by Selmonosky's diagnostic triad (DT) which involves supraclavicular tenderness (Morley's sign) as well as relative weakness of the fifth finger, with or without a white hand sign.<sup>69,70,71,116</sup> I classified scapular depression as having the superior scapular angle situated more than two finger widths below the T2 vertebra (approximately 2 cm). The survey revealed that 100% of the patients had scapular depression on the symptomatic side, cf. Sahrman and colleagues' norms. Most of these had been told to pull their scapulae back and down to correct their posture by their musculoskeletal therapist, and became considerably worse after following these cues, as a result. Five of these even had TOS surgery to decompress the CCS, but were still told to pull their shoulders back and down by their therapist, although TOS surgery clearly aims to increase the costoclavicular interval. The latter patients had severely depressed scapulae on the afflicted side, resulting in compression of brachial plexus between the clavicle and the 2nd rib. It is my impression, although clearly well-intended, that being cued to pull "back and down" obviously and iatrogenically exacerbated the situation for these patients.

Because of the consensus that scapula has a tendency to protract, rotate forward (anterior tilt) and down (downward rotation) in patients with SD, SIS and neck pain, there may be some warrant in increasing scapular retraction alone. However, pulling back and down will cause the scapula to retract, depress and downwardly rotate, and is not compatible with any the criteria provided by the evidence, which states to increase retraction, upward rotation and posterior tilt. Increasing depression, downward rotation and anterior tilting may not only promote scapular dyskinesis and shoulder impingement syndrome, but

also encroachment of the costoclavicular space and sequellar TOS.

The notion that proper scapular posture is obtained by pulling the shoulders back and down, is most likely based on only a few EMG studies, which show high upper trapezius output, as well as “pirate tales” from fitness and powerlifting communities. However, because patients with SIS and SD also demonstrate downward rotation, protraction, and depression, and, because the UT prevents these, it is unlikely to be truly over active nor over-engaged in moving the scapulae in a pathological manner. Retraction and depression of the scapulae may cause compression of the CCS as it mimics the Halstead’s CCS compression test. CCS compression implies osseous compression of the neurovascular bundle, which may lead to many diffuse and seemingly unrelated secondary problems, whose etiology may prove difficult to identify. Scapular depression has also been uniformly identified in TOS patients. There are also reports that scapular depression has been associated with increased myofascial pain in the cervical musculature, restricted range of motion, WAD, upper extremity DVT development, and more. Therefore, often well-intended yet misunderstood cueing of pulling the shoulders “back and down”, may set the patient on a dark journey with many diffuse symptoms and few answers. Because unreliable diagnostic value of neurographic examinations, and because relatively few practitioners are versed in recognizing the signs of TOS, especially in its beginning-intermediate stages, there is likelihood that the patient will continue to pull their shoulders back and down and not suspect

this as the etiology of their newfound problems. This can lead to longstanding problems for the patient and it may take a long time before his or her symptoms are finally identified as related to costoclavicular space compression.

## LIMITATIONS

The survey part of this manuscript is based on patients who, on their own initiative have visited my unsubsidized private clinical practice. They may or may not fully represent the general patient population within the NHS.

## CONCLUSION

In conclusion, it is of utmost importance to evaluate the patient’s scapular resting position based on the evidence, rather than generically cueing him or her to pull their scapulae back and down. If the patient presently has low-riding scapulae, and is cued further into depression, an iatrogenic sequela of maladies may develop as result. Pulling the shoulders “back and down” is a logical fallacy, which does not result in what it is thought to do, is not compatible with the evidence, the anatomy, biomechanics nor with common sense. Although the cue itself, and I reiterate, is clearly originating from well-meaning therapists, it is my impression that the SIS and SD studies have been gravely misinterpreted, and that the “back and down” cue should be abolished once and for all.

**Conflict of interest:** Nil

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★★★☆☆

## MY MOST MEMORABLE PATIENT

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It is human nature to seek rewards, some are necessary for survival and for worldly comforts,



these are earning money and amassing wealth,



satisfaction and is beyond any description, this later reward is exemplified with monitoring and

follow up of a little girl's growing up,(opportunity to get this type of reward often knocks at the the door of medical professionals provided we record the progress of our patients over a long period ).

This girl was born with most of her intestine and liver out side her abdomen this is an emergency and untreated, is life threatening. We replaced her intestine back

and gradually repaired her tummy. Her hernias were also repaired in early childhood. Years moved on and three days ago grateful mother brought this fifteen years old young lady of class 9th just to pay their personal tributes. Here one can also see the similarity of abdominal scar then and now !



It is now possible to improve this scar, but girl is quite happy to keep it as it is ! We should pray that Lord almighty may give us more such opportunities frequently.

★★★☆☆