Journal of Bodywork & Movement Therapies (2015) xx, 1–9

Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/jbmt

FASCIA SCIENCE AND CLINICAL APPLICATIONS: APPLIED PHYSIOLOGY

The Torsional Upper Crossed Syndrome: A multi-planar update to Janda's model, with a case series introduction of the mid-pectoral fascial lesion as an associated etiological factor

Craig E. Morris, DC, DACRB, Debra Bonnefin, DC, DACRB, MAppSc*, Caroline Darville, PT

FIRST Health, 19000 Hawthorne Blvd. Suite 302, Torrance, CA 90503, USA

Received 6 June 2014; received in revised form 6 August 2015; accepted 12 August 2015

KEYWORDS
Fascia;
Pectoralis;
Chest wall;
Upper crossed syndrome;
Headache;
Neck pain;
Shoulder pain

Summary The Upper Crossed Syndrome (UCS) was presented by Janda to introduce neuromotor aspects of upper body muscle imbalances, describing sagittal plane postural asymmetries as barriers to recovery from chronic locomotor system pain syndromes. The UCS describes muscle imbalances of key antagonists causing forward postures of the head and shoulders and associated changes in the spinal curves —particularly an increased thoracic kyphosis — as well as changed function in the shoulder girdle. The role of fascial tissue has gained remarkable interest over the past decade, previously emphasizing its anatomic compartmental and binding role, while more recently emphasizing load transfer, sensory and kinetic chain function. The authors introduce the Mid-Pectoral Fascial Lesion (MPFL) as a myofascial disorder, describing 11 ipsilateral chest wall cases. While managing these cases, the authors encountered and subsequently designated the Torsional Upper Crossed Syndrome (TUCS) as a multi-planar addition to Janda's classic sagittal plane model.

This article integrates published updates regarding the role of posture and fascia with the effects of chest wall trauma and a newly described associated postural syndrome as illustrated with this case series. An effective therapeutic approach to release the MPFL is then briefly described.

* Corresponding author. 360 East 1st Street, Tustin, CA 92780, USA. Tel.: +1415 531 4848.
E-mail address: dbonnefin1@gmail.com (D. Bonnefin).

http://dx.doi.org/10.1016/j.jbmt.2015.08.008
1360-8592/

Please cite this article in press as: Morris, C.E., et al., The Torsional Upper Crossed Syndrome: A multi-planar update to Janda's model, with a case series introduction of the mid-pectoral fascial lesion as an associated etiological factor, Journal of Bodywork & Movement Therapies (2015), http://dx.doi.org/10.1016/j.jbmt.2015.08.008

Introduction

The relationship between fascia, posture and health, has been well documented over the past century. AT Still and DD Palmer noted the critical importance of the deep fascia for osteopaths and chiropractors over a century ago (Palmer, 1914; Still, 1902). Mennell and Lewin both recognized Goldthwaite's early 20th Century contribution, emphasizing the importance of posture in relation to health and recovery (Lewin, 1955; Goldthwaite 1945; Mennell, 1920).

Albee introduced the clinical integration of muscle and fascial tissues in 1927 when he described the new disorder of 'myofascitis' (Albee, 1927). Travell, and Rizler, further clarified the anatomical and physiological nature of muscle and fascial tissues by cementing the term 'myofascial' in the literature (Travell and Rinzler, 1953). Nimmo highlighted the reflexive aspects of trigger points (TrP's) while pioneering effective 'direct' (i.e. direct contact of the TrP) manual therapeutic methods (Cohen and Schneider, 1990). Travell continued her groundbreaking myofascial pain and dysfunction work with Simons, culminating in their classic textbooks (Travell et al., 1992, 1998). Rolf highlighted the individual role of the fascial system itself stating, 'Fascia is the organ of posture' (Rolf, 1990). Janda, emphasizing the importance of central and peripheral neural factors in his postural syndromes, described the faciliatory/inhibitory role of muscle imbalances as etiological factors in chronic pain syndromes (Janda, 1968, 1972, 1994). Janda's Upper Crossed Syndrome (UCS) demonstrated how such imbalances influenced postural stability, with both head and shoulders shifted anteriorly (Morris et al., 2006). These 20th century leaders, among others, helped to establish a platform for dramatic escalation of the 21st Century understanding regarding the complex role of the 'neuromyofascial system'.

This century, L. Stecco and colleagues extended and integrated the neuromyofascial system's role in relation to what he calls 'the locomotor apparatus' in both physiological and pathological circumstances (Stecco, 2004). There is now a deeper understanding of the role of fascia in load and force transfer, morphological compartmentalization, and contractile and sensory (proprioceptive and nociceptive) function (Schleip, 2003; Schleip et al., 2007; Stecco, Masiero, et al., 2009; Stecco, 2004; Vleeming et al., 1995).

Investigating fascial aspects of torso and upper extremity functional anatomy, A. Stecco et al., performed chest wall dissections of 6 unembalmed cadavers (Stecco, et al., 2009). They studied the thickness and properties of the pectoral fascia, noting the deep fascia is a thin, laminated, collagenous layer that is intimately connected to the pectoralis major via numerous intramuscular septa. Functioning as a myofascial unit, the deep laminar layer is anchored to the *local* periosteal margins (clavicular, sternal etc.). Additionally, they state that the pectoral fascia acts as an epimysium to the pectoralis major muscle containing muscle spindles that 'allow muscle contractions to be modulated by "peripheral" demands' (Stecco, et al., 2009). The authors suggest this as a possible anatomical contributor for peripheral motor coordination.

These same investigators found that the superficial lamina of the deep pectoral fascia (i.e. 'pectofascial layer') *traverses* the local attachments to communicate directly with *regional* myofascial tissues such as the sternocleidomastoid (i.e. neck region) superiorly, the deltoid, trapezius and latissimus (i.e. shoulder region) laterally, the contralateral pectoralis (chest wall region), and obliquus externus (abdominal region) inferiorly, They also noted that the pectofascial connection could assist with symmetrical counterforces between both contralateral pectoralis groups during bilateral lifting/loading of the upper extremities (Stecco, et al., 2009). This 'trans-regional' architecture can impact motor control along kinetic chains, longitudinally, transversely or obliquely which L. Stecco calls 'slings' (Stecco, 2004). Stecco and Masiero posit that these 'slings' impact function, force and sensory transmission between the trunk, head/neck and all four extremities (Stecco, et al., 2009).

In this same publication, the contributors reported without emphasis that 2 of 6 cadavers demonstrated excessively thickened fascia (2—3 times greater than the other 4 cases) in the mid-pectoral region (Stecco, et al., 2009, p. 260). Is such pectofascial thickening a lesion, perhaps an adhesion or fibrotic scarring? If so, what is the causation and nature of this so-called lesion, and what local and regional consequences would occur in the event of an asymmetrical lesion?

Statement of the problem

The incidence of chest pain due to myofascial dysfunction varies in the literature. In one study, 40% percent of primary care chest pain patients are diagnosed with musculoskeletal chest pain, while another listed musculoskeletal chest pain at 49% (Stochkendahl and Christensen, 2010; Svavarsdottir et al., 1996). Persistent symptoms are common, but unfortunately are often attributed to lack of a thorough and systematic examination once coronary diagnosis has been excluded (Eslick et al., 2003; Stochkendahl and Christensen, 2010). These studies do not take into account a fascial origin or contributor of pain, which may also account for the persistent symptoms noted by these authors.

Women who have been treated for breast cancers with radiation and/or surgery have a risk of developing adhesions, fibrosis and chest wall tenderness (Crawford et al., 1996; Kim and Park, 2004; Lacomba, del Moral, Coperias Zazo, Gerwin and Goni, 2010). Studies vary on rates of developing myofascial pain after breast cancer surgery from 21% to 44% (Cheville and Tchou, 2007; Lacomba et al., 2010). Axillary web syndrome (AWS) has been reported as a sequelae following breast cancer surgery with patients demonstrating tightness of the axilla and chest wall, a protracted shoulder on the side of surgery, decreased shoulder abduction and referred arm pain to the wrist, and associated thoracic kyphosis (Lacomba et al., 2010) (Kepics, 2004; Lacomba et al., 2010). AWS is accompanied by adhesions and the treatment is similar to that of chest wall adhesions (Cheville and Tchou, 2007; Crawford et al., 1996; Kepics, 2004; Smoot et al., 2010).

Other causes of chest wall adhesions have been reported in the literature. Post-surgical adhesions can develop following benign lumpectomies, breast augmentations and

Please cite this article in press as: Morris, C.E., et al., The Torsional Upper Crossed Syndrome: A multi-planar update to Janda's model, with a case series introduction of the mid-pectoral fascial lesion as an associated etiological factor, Journal of Bodywork & Movement Therapies (2015), http://dx.doi.org/10.1016/j.jbmt.2015.08.008

reductions, in addition to pulmonary and cardiovascular conditions (Kim and Park, 2004; Lacomba et al., 2010). Adhesions can also develop due to overuse, leading to pain, long-term, low grade myofascial inflammation and subsequent scarring and fibrosing (Stecco, 2004).

A number of authors from the Czech Republic led by Lewit have investigated adhesions and scar tissue. In 2004, Lewit and Olsanska described the evolution of scar tissue clinical management, with findings that included both palpatory tenderness and restricted mobility (Lewit and Olsanska, 2004). They reported on 51 patients with 'active scars' that responded well to manual therapeutic interventions. They defined 'active' scar tissue as a scar that does not allow for the subsequent layers of tissue to move independently of each other, similar to adhesions. Kobesova et al., further defined active scar tissue to include proprioceptive changes that subsequently causes faulty efferent output, critically compromising postural and normal kinetic chain activity (Kobesova et al., 2007). A key component of the definition of an active scar is ongoing, altered neuromotor activity with movement restrictions or adhesions to underlying layers of muscle and fascia, with the postural and joint compromises occurring secondary to the shortened and/or imbalanced tissues (Kobesova et al., 2007).

Lewit and Olsanska noted that diagnosing these disorders can be challenging because the site of lesion and symptomatic region may be anatomically distant (Lewit and Olsanska, 2004). For example, they reported numerous chest wall surgical cases (11 of 51 cases), with no chest wall symptoms. Instead the main regions of reported pain were the low back and shoulder/arm (14 each), followed by headache (8 cases). Because the lesion site can be distant to the region of pain or other symptoms, a thorough examination, often to an asymptomatic region, is warranted in order to establish the correct diagnosis.

This paper investigates chest wall dysfunction due to restricted fascial tissue among 11 patients, noting the association with postural dysfunction and varying local and distant symptomatic presentations.

Case series presentation

This is a case series report of 11 patients with similar fascial lesions who were treated by the lead author at his private clinic. Patients were treated from 2008 to 2012. A clear explanation was provided to each patient and prior approval obtained for assessment and subsequent therapy. Because Case #1 was the initial patient observed with an mid-pectoral fascial lesion (MPFL) that resulted in a therapeutic resolution, it is discussed in detail in order for the reader to follow the clinical progress and reasoning. The subsequent 10 cases are only summarized. Additionally, Tables 1 and 2 provide a complete overview of symptoms and clinical findings for each case, respectively. The following cases all resulted in the same clinical observation, that of the MPFL.

Case #1: 33-y/o right-hand dominant female physiotherapist with recurrent right shoulder pain over a 2-month period following two separate falls on her right shoulder. She subsequently noted persistent pain with increased lifting, pulling, and end-range motion (flexion abduction and external rotation). Her shoulder was especially provoked while demonstrating resistance exercises with patients. Being a therapist herself, she attempted several self- and assisted (by colleagues) treatment strategies for her shoulder, including physiotherapeutic modalities, stretching of tight pectoral tissues, massage, mobilization of the joints of the spine and upper extremity, strengthening and postural exercises in both open and closed chain strategies. She ultimately consulted the lead author for treatment of these persistent symptoms.

She initially received 2 sessions of in-office therapy that included manipulation of dysfunctional ipsilateral upper quarter joints, ipsilateral periscapular and chest wall (indirect) muscle energy techniques and Dynamic Neuromuscular Stabilization (DNS) reflex stimulation and supportive home exercises. These approaches resulted in short-term benefit only. Therefore, a more thorough examination was performed, which revealed a notable tender restriction of the right chest wall during gliding pectoral fascial palpation from a caudal approach. This lesion was located on the pectofascial wall, apparently adhering to the overlying mammillary/superficial fascial tissue at approximately the level of the 4th – 5th ribs and along the mid-clavicular line. This restriction was somewhat circular and approximately 1 cm in diameter. This lesion is referred to as the (MPFL). A comparison palpatory fascial assessment was performed to the left chest wall that confirmed no such tenderness or restricted fascial gliding. Increased myofascial tone of the entire right-greater-than left chest wall was noted. Because 'indirect' mobilizations to the chest wall had been previously attempted to address this chest wall myofascial hypertonicity, and because of the nature of the palpable adhesion with gliding fascial palpation, direct manual fascial release techniques were deemed clinically warranted. By mutual agreement, direct chest wall MPFL release was performed.

The only manual assessment and therapeutic access was via the sub-mammillary tissues, since the breast was overlying the noted lesion as described in the Therapeutic Measures section. The MPFL fascial release was performed, followed by spinal and upper extremity manipulations, mobilizations and DNS therapy. The patient was then instructed to perform daily direct self-chest wall mobilizations in addition to her home exercises. These exercises largely consisted of DNS patterns designed to emphasize centrated movements that by definition reestablish muscle balance and optimal joint motion (Frank et al., 2013). Because her right shoulder was forward, postural retraining was critical to restore this postural imbalance. The patient noted improvement following this treatment. She received a total of 7 treatments over a period of 3 weeks and was released from care as asymptomatic. She was instructed to continue her home care program for the foreseeable future.

Further historical investigation into the possible etiological nature of the MPFL was performed. The patient denied direct right chest wall trauma, but did mention that her right breast was slightly larger than the left and that she regularly suffered increased right pre-menstrual breast pain for about two weeks monthly. Part of her pain was from unilateral brassiere cup compression during this

Please cite this article in press as: Morris, C.E., et al., The Torsional Upper Crossed Syndrome: A multi-planar update to Janda's model, with a case series introduction of the mid-pectoral fascial lesion as an associated etiological factor, Journal of Bodywork & Movement Therapies (2015), http://dx.doi.org/10.1016/j.jbmt.2015.08.008

Table 1 Symptoms.

Patient#	HA	Chest wall pain	Breast pain	Neck pain	Mid-back pain	Shoulder pain	Arm dysesthesias	Chest wall tension	Aware head tilt	Aware Ant shoulder
Patient#1						X I				
Patient#2	X I	X I	X I	X I	X	X I	X I			
Patient#3				X B		X I		X I		
Patient#4	X I	X I	X I	X I		X I	X I	X I		
Patient#5	X I		X I	X I		X I				
Patient#6	X I	X I	X I	X I		X I	X I	X I		
Patient#7	X I	X I		X I		X I	X I	X I		
Patient#8		X I		X I		X I	X I			
Patient#9	X I	X		X	X	X I	X I	X I		
Patient#10	X I			X I	X	X I				
Patient#11	X I			X I	X	X I	X I	X I		

HA = headache, I = ipsilateral (Same side of MPFL & Protracted Shoulder), B = Bilateral.

period of hormonally-mediated mammillary engorgement. At times in the past, she had utilized another brassiere with larger cups premenstrually, but admitted to forgetting and using it less as the years passed. Therefore, there is the question of whether long-term, low-grade compressive fascial irritation from tight clothing may have caused this lesion formation, which was then provoked while exercising her patients.

In total 11 patients were diagnosed with an MPFL and accompanying ipsilateral TUCS. Included were 9 females and 2 males. The mean age was 40 with a range from 22 to 55 years of age. Etiologies varied to include: direct trauma (2 cases), indirect trauma to the upper quarter (1 case), post-surgical and post-radiation complications of breast cancer (2 cases), breast augmentation (1 case), motor vehicle collisions (3 cases) and repetitive microtrauma from softball (2 cases). In each case varying types of treatment had been provided, yet failed to offer long-term relief, including: various physiotherapy modalities, manual manipulation and mobilization techniques to the cervical and dorsal spine and upper quarter, DNS therapy, medication, and various exercise programs. In each case, a unilateral TUCS and ipsilateral MPFL was isolated following prior failed trials of manual therapy and exercise. The MPFL was successfully released via manual therapeutic measures in each case and subsequent therapy (manual, modalities and exercises) resulted in significant reductions in symptoms and improved postural findings.

Discussion

This case series illustrates various potential etiologies of the MPFL, several of which are associated with pain or paresthesia. These include seat belt trauma from motor vehicle accidents, blunt trauma, repetitive trauma, clothing-related tissue compression, post-surgical complications, and finally therapeutic radiation. While the other factors are well described in the literature, compressive clothing requires additional clarification. Chest wall compressive clothing, i.e. brassieres, is associated with sternoclavicular pain (De Silva, 1986) pectoral girdle pain, (Ryan, 2000) with negatively impacted autonomic function (Miyatsuji et al., 2002), and shoulder-neck pain associated with large brassiere cup sizes (Oo et al., 2012).

Table 2 Findings.

Patient#	Spinal Joint Dys-F	Ant rib Dys-F	Post rib Dys-F	GHJ protraction	SCJ Dys-F	CSJ Dys-F	ACJ DYS-F	Pec TrP	Head tilt	MPFL
Patient#1	X (C,T)			X I		X I	X I	X I		X I
Patient#2	X (C,T)	X	X	X I	X I	X I		X I	X I	X I
Patient#3	X (C,T)	X	X	X I					X I	X I
Patient#4	X (C,T)	X	X	X I		X I		X I		X I
Patient#5	X (C)	X		X I	X I			X I	X I	X I
Patient#6	X (C,T)	X	X	X I				X I	X I	X I
Patient#7	X (C,T)	X		X I	X I		X I	X I	X I	X I
Patient#8	X (C,T)	X	X	X I						X I
Patient#9	X (C,T)	X		X I		X I	X I	X I	X I	X I
Patient#10	X (C,T)	X	X	X I				X I	X I	X I
Patient#11	X (C,T)	X	X	X I	X I	X I	X I	X I	X I	X I

Dys-F = dysfunction, C = cervical, T = thoracic, Ant = anterior, Post = posterior, GHJ = Glenohumeral joint, SCJ = Sternoclavicular joint, CSJ = Costosternal Joint, ACJ = Acromioclavicular joint, Pec = Pectoralis major muscle, TrP = Trigger point, MPFL = Mid-Pectoral Fascial Lesion, I = Ipsilateral

Please cite this article in press as: Morris, C.E., et al., The Torsional Upper Crossed Syndrome: A multi-planar update to Janda's model, with a case series introduction of the mid-pectoral fascial lesion as an associated etiological factor, Journal of Bodywork & Movement Therapies (2015), http://dx.doi.org/10.1016/j.jbmt.2015.08.008

The MPFL is found at the 'submammary fascial interface' (located between the superficial and deep pectoral fascial layers), which we do not believe has been previously reported in the literature in this manner (i.e. post-radiation and surgical adhesions, as noted above, have been generically reported).

The authors feel that another noteworthy observation has emanated from these cases: the introduction of a Torsional Upper Crossed Syndrome, a 3-dimensional update to Janda's UCS. Janda described the UCS as a muscle imbalance of key antagonists causing forward postures of the head and shoulders with associated cervical hyperlordosis, thoracic hyperkyphosis and protraction of both shoulders. Altered regional mechanics are associated with these dysfunctions. The UCS has been classically considered to be a sagittal plane dysfunction, as represented in typical published two-dimensional lateral view illustrations (Fig. 1). Furthermore, the musculature imbalance describes the contralateral musculature (i.e. upper trapezii, pectorals, levator scapulae and inhibition/weakness of the lower scapular stabilizers) in the plural, further supporting the understanding that the UCS is an *inferred 2-dimensional model* of symmetrical sagittal plane dysfunction. While it is clear that the protracted shoulder not only deviates from the neutral position in a purely anterior direction, a 3rd dimensional (i.e. from an oblique perspective) illustration has never, from our English literature review, been previously published.

In all cases, the MPFL and TUCS were noted to be on the ipsilateral side (Fig. 2). Overlapping symptom complexes (Table 1) and clinical findings (Table 2) were consistent among the patients and therefore establish the foundation for this case series.

The superficial pectoral fascia, with the enclosed mammillary parenchyma and stroma, attaches strongly to the dermis. There is no direct skeletal muscle attachment to the superficial pectoral fascia. The breast tissue, along with the platysma muscle superiorly, are enveloped within this fascia and bridged together via the supraclavicular superficial fascia (Stecco, et al., 2009). As such, the superficial fascia and inclusive breast tissue glides smoothly over the underlying pectofascial layer, contiguously communicating with the adjoining platysma myofascial tissues superiorly. This linked anatomical relationship may account for the reported increase in postmenopausal large breasts and thoracic pain (Spencer and Briffa, 2013). The authors propose that the fascial loading to the platysma secondary to the inferiorly connected ptotic, heavy breast may influence the formation of the UCS via mechanical and/or neurostimulatory motor mechanisms.

The deep pectoral fascia is dense and adheres strongly to the underlying pectoralis major muscle. The superficial and deep fascial layers are loosely connected, allowing the overlying breast tissue packaged within the superficial fascia to glide over the pectoralis major along this pectomammary bifascial interface. However, the cases

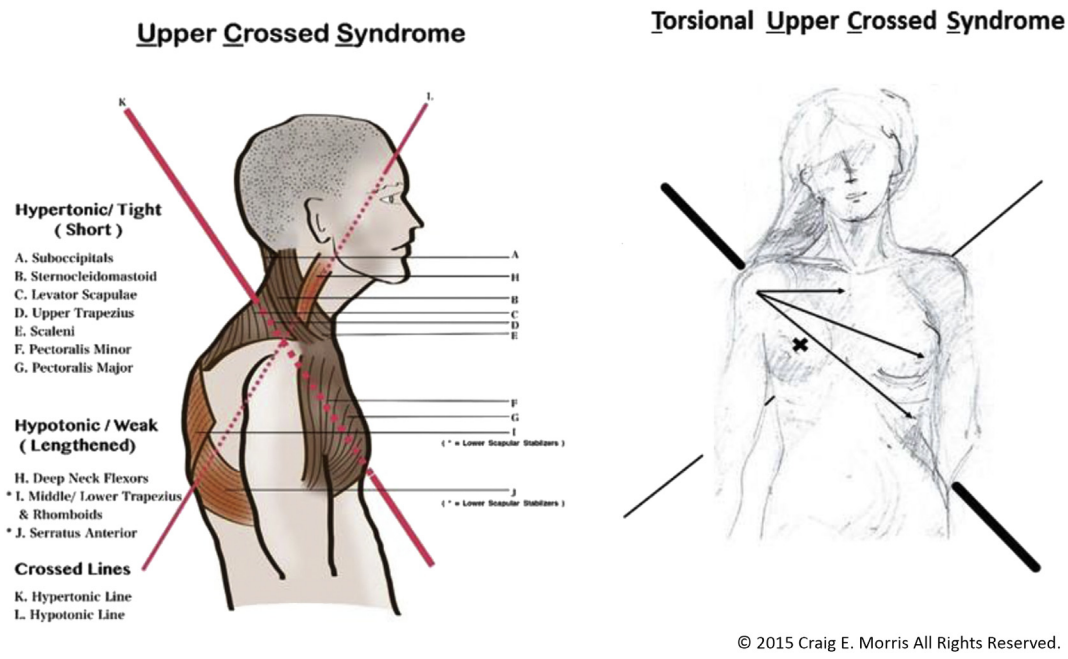


Figure 1 Left: Illustration of UCS from the lateral view, representing crossed lines of hypertonicity/shortening (K) and hypotocinicity/lenthening (L). This represent a 2-dimensional model. Right: Illustration of the TUCS/TVCS: Note the lateral deviation of the head, with hypertonic myofascial tissues and hair falling, to the right side. The right shoulder is relatively forward. The "X" marks the placement of the MPFA. The upper most line represents the humero-sternal myofascial chain as it traverses the hypertonic right pectorals. The second line represents the humero-contracostal myofascial chain than passes over the sternum and extends to the contralateral ribs. The third line represents the humero-infracostal chain that extends to the lower ribs (lpsilateral or contralateral). These chains, together with their inhibited, lengthened contralateral counterparts, define the TUCS. The final line represents the humero-contrapelvic myofascial line, extending to the ASIS. This extends beyond the thorax and is therefore described as the TVCS. TUCS = Torsional Upper Crossed Syndrome TVCS = Torsional Ventral Crossed Syndrome, MPFA = Mid-Pectoral Fascial Syndrome, ASIS = Anterior Superior Iliac Spine.

Please cite this article in press as: Morris, C.E., et al., The Torsional Upper Crossed Syndrome: A multi-planar update to Janda's model, with a case series introduction of the mid-pectoral fascial lesion as an associated etiological factor, Journal of Bodywork & Movement Therapies (2015), http://dx.doi.org/10.1016/j.jbmt.2015.08.008

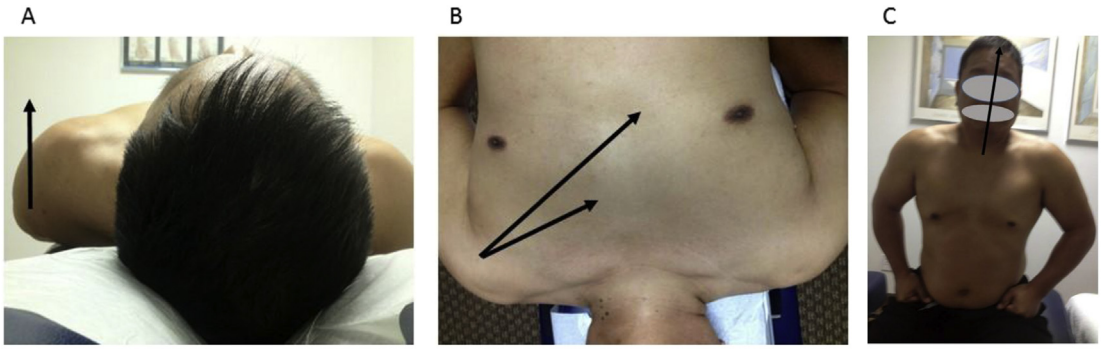


Figure 2 Patient #7. A: Cephalad view taken of the supine patient. Note that the left shoulder is anterior relative to the right, while the left pectoral muscle appears hypertonic. B: AP view taken of supine patient demonstrates hypertonic and forward left chest/shoulder in comparison to the right. the patient was unaware of any of these asymmetries.

presented here indicate the presence of a lesion adhering these two fascial surfaces, restricting such gliding.

Stecco, Macchi et al., point out that when the fascia is altered from trauma or overuse the mechanoreceptors become active even at rest, which may result in myofascial pain syndromes (Stecco, et al., 2009). Myofascial trigger points are commonly observed in the upper quarter, including the pectoralis major and minor, which can provoke local or referred pain and paresthesia of the ipsilateral shoulder and upper extremity (Greenman, 1996; Travell et al., 1998). Sternalis muscle trigger points may also be present provoking pain and/or paresthesia locally over the sternum (Travell et al., 1998).

Palpatory evaluation of each of these patients demonstrated two key diagnostic findings of the MPFL: unilateral pectofascial tenderness and restricted gliding in multiple planes of the superficial loose fascial layer over the underlying deep pectoral fascial layer. This method of assessment is termed by the authors the *pectofascial glide assessment*.

For male patients, especially with minimal to no chest wall adipose tissue, direct mid-pectoral palpation was clinically optimal. For female patients, and male patients with excess pectoral adipose tissue and/or tenderness, the superficial fascial tissue bulk, as well as potential tenderness and loose areolar morphology interfered with direct, lateral, medial or cephalad palpatory reliability regarding the MPFL. Therefore, optimal access to the lower pectoral fascia along the mid-clavicular line occurred from caudal access, allowing direct pectofascial palpation to the adhesion site.

Marked tenderness was consistently noted at the 4th—5th ribs, along the mid-clavicular line. It is one to two ribs cephalad to the infra-mammillary fold, which approximates the 6th rib level, but can vary anatomically in location (Muntan et al., 2000). The MPFL is found to be approximately 0.5—1 cm in size. Because of the tenderness and restricted fascial glide, this is described as a 'lesion' here, although there is reason to describe it as an adhesion. It is important to perform bilateral chest wall assessment, in an attempt to confirm the diagnosis as reliably as possible. In the case of the asymmetrical adhesion, there was significantly less or no restricted fascial gliding and/or tenderness present on the patient's contralateral mid pectoral region. Finally, ipsilateral greater than contralateral palpable costosternal dysfunction was consistently noted.

Other common examination findings were hypertonicity in the ipsilateral pectoralis minor and major musculature, sternocleidomastoid, scalenes, upper trapezius and levator scapulae muscles.

Marked tenderness was also noted at the tip of the transverse process of C2 on the ipsilateral side. The ipsilateral shoulder was protracted in comparison to the contralateral side. When integrated with the ipsilateral head tilt, the oblique line of hypertonic myofascial tissues completes and defines the TUCS (Fig. 1).

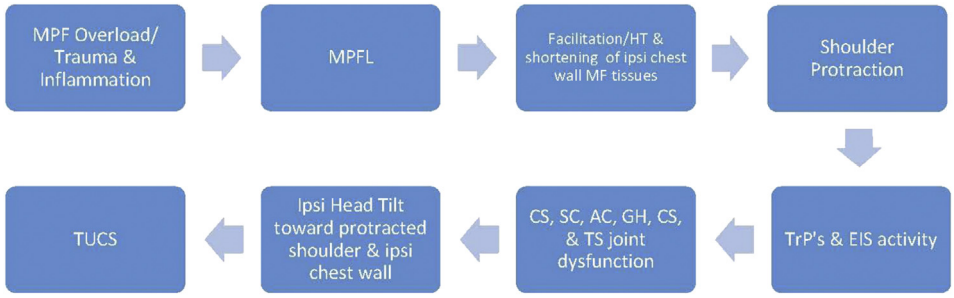
In addition to Janda's description of the UCS, others have described a similar postural presentation as 'Forward Head and Rounded Shoulder Posture' (FHRSP). Thigpen et al., noted that consistent criteria defining the FHRSP has not been established (Thigpen et al., 2010). They found that this posture has been associated with altered scapular kinematics and muscle activity, even in the absence of pain (Thigpen et al., 2010). Other regional disorders have been reported that may provide overlapping conditions and provide additional etiological possibilities. Kalke et al., reported on the 'sternoclavicular syndrome', finding that pain and tenderness at times extended to the costosternal junctions inferiorly (Kalke et al., 2001). In a case study of 58 patients, they found 40 to have unilateral symptoms, with ipsilateral shoulder and/or arm pain to be common complaints.

A theoretical cascade

The cases presented here demonstrate that the initiating cause and order of dysfunction can vary. Nevertheless, a theoretical continuum of dysfunction is proposed in Fig. 3.

The restricted fascial layers of the MPFL can provoke afferent stimulation to affect local fascio-pectoral hypertonicity. According to Stecco et al., the deep lamina of the deep pectoral fascia extends to the ipsilateral aspects of the costosternal region of the chest wall, which may explain the tenderness found at the sternocostal and costal margins in these cases. We propose increased insertional irritation in addition to myofascial irritation and trigger

Please cite this article in press as: Morris, C.E., et al., The Torsional Upper Crossed Syndrome: A multi-planar update to Janda's model, with a case series introduction of the mid-pectoral fascial lesion as an associated etiological factor, Journal of Bodywork & Movement Therapies (2015), http://dx.doi.org/10.1016/j.jbmt.2015.08.008



MPF = Mid-pectoral fascia, MPFL = Mid-pectoral fascial lesion, HT = hypertonicity, Ipsi = ipsilateral, MF = Myofascial, Chest wall myofascial tissues = pectoralis major, minor, intercostal muscles and sternalis, TrP = trigger point, EIA =, Enthesopathic Insertional Activity, CS = Costosternal articulations, SC = sternoclavicular joints, Acromioclavicular joint, GH = Glenohumeral joint, CS = Cervical spine, TS = Thoracic Spine, TUCS = Torsional Upper Crossed Syndrome.

Figure 3 Proposed cascade of dysfunction leading to MPFL and altered mechanics of the upper quarter.

point activity of intercostal myofascial tissues as etiological factors of this local tenderness, pain and referred symptomatology. In addition, associated dysfunction of the costosternal joints can interfere with optimal thoracic motion and provoke local pain and further contribute to overall myofascial dysfunction of the chest wall (Greenman, 1996).

Proposed mechanisms for the regional reactions are explained next. The superficial lamina of the deep pectoral fascia passes over the clavicle and integrates with the regional tissues via the occipital and cervical myofascial chains resulting in increased enthesopathic tension to the ipsilateral cervical spine and skull. The resultant asymmetrical joint loading and dysfunction can lead to altered 3-dimensional mechanics of head tilt, anterior spinal translation, and asymmetrical motion and dysesthesias to the head and the upper quarter.

Blau, in his 2005 summary, emphasized the extracranial etiological nature of tension headaches "arising not from muscular, but rather fascial coverings and tendons, or their insertions into the periostium, or from relevant joints" (Blau, 2005). Blau stated such regions include the temporomandibular joints, the cervical region (i.e. cervicogenic headaches) and the chest wall (Blau, 2005). According to Blau, asymmetrical tension-type headaches, were noted among 8 of these 11 patients. Bogduk and Marsland discussed the etiological aspects of cervicogenic headache, emphasizing the "biomechanical vulnerability" of the C2-3 zygapophyseal joints (Bogduk and Marsland, 1986). They noted this transitional spinal crossroad linking the head-rotating (i.e. axial plane) joints above and flexion-extension (i.e. sagittal plane) joints below. (Bogduk N, Marsland A. Almost three decades later, Bogduk cited the C2-3 zygapophyseal joints as the most likely source of cervicogenic headache, with lateral C1-2 joints and C3-4 zygapopheses the next most likely sites (Bogduk, 2014). These critical points can assist one to better visualize the headache component of the TUCS, with fascial chains from the chest and cervical regions causing torsional ipsilateral neck and head deviation, tensioning of the upper cervical periostia and asymmetrically loading the upper cervical joints and more direct influences from the commonly associated increased posterior myofascial chain activity around the cervicothoracic region found in UCS in an attempt to compensate for the pectoral (and other myofascia) over-activity.

Furthermore, ipsilateral shoulder protraction results in altered shoulder mechanics including scapular instability, glenohumeral impingement and expanding regions of myofascial trigger point activity (Page, 2011; Thigpen et al., 2010; Travell et al., 1998). With the inclusion of the head/neck region asymmetry, the ipsilateral chest wall and shoulder protraction, the full TUCS is established.

Therapeutic Measures

Indirect fascial release methods consistently proved insufficient to release the MPFL. This is logical since the superficial pectoral fascia encompasses the mammillary tissues and has no direct skeletal muscle attachment. As such, an indirect fascial release contacting the origin and insertion of the underlying pectoralis major myofascia would fail to release the adherent superficial fascia since it would simply move with it at the adhesion site. Therefore, direct release methods, designed to shift or separate the two fascial surfaces, were utilized with successful results. Success in these cases was based upon normalization of chest fascial motion symmetrically, reduction/normalization of the forward shoulder, reduction of the UCS and reduction of reported pain.

Of the 11 cases, the number of sessions required for release was between 3 and 16 sessions, with an average of 8.7 sessions. The four main determinants of the number of in-office sessions were chronicity of MPFL, compliance with home self-mobilization exercises, avoidance of tight brassieres/clothing compressing the breast/chest wall, and skill in performing self-mobilization exercises as detailed below.

Please cite this article in press as: Morris, C.E., et al., The Torsional Upper Crossed Syndrome: A multi-planar update to Janda's model, with a case series introduction of the mid-pectoral fascial lesion as an associated etiological factor, Journal of Bodywork & Movement Therapies (2015), http://dx.doi.org/10.1016/j.jbmt.2015.08.008

For MPFL release of the two male patients, supine position was most effective. Direct skin contact with the pisiform/proximal hypothenar region of the ipsilateral extremity (i.e. left chest wall and left hand) over the MPFL is performed. As the superficial tissue/slack is removed the contact hand moves toward the opposite shoulder until a hard barrier of resistance is met. The clinician then releases a little of the tension, while maintaining the same downward pressure. Then, a sudden, light thrust, designed to mobilize the adhesion, is performed repetitively between 3 and 5 times (after the initial barrier of pectoral fascial tissue resistance is palapated). The fact that this mild thrusting technique provided better results than light mobilization methods described for fascial adhesions (Stecco, 2004) leads us to suspect that a fibrotic component of the MPFL may be present.

The same maneuver is then performed to the opposite chest wall, with only one or two thrusts if there is little or no contralateral lesion, to allow the patient to feel the difference between the presence and absence of adhesion. Fascial releases tend to be more effective if released in multiple planes of motion, so the same procedure is then performed at 90° from the initial release line, using the opposite hand applied to the same MPFL. Then, the opposite chest wall is targeted with the thrust again directed toward the opposite shoulder.

MPFL release was easily performed on the male patients, but required infra-mammillary methods for the females. For females, the loose nature of the mammary tissue makes supine fascial release impractical, as the goal is to mobilize the overlying superficial areolar fascia from the underlying deep pectoral fascia. Therefore, a seated position allows the superficial areolar fascial/mammillary tissue to be tensed by gravity so that the two target fascial layers can be differentiated and then released from one another. A hand contact, utilizing the interphalangeal web between the first & second digit as a type of 'functional spade' can provide an effective shearing-type release. As with male patients, 3—5 light thrusts directed at an angle toward the opposite shoulder is performed bilaterally and then performed toward the ipsilateral shoulder. The patient is then instructed to perform the maneuver once daily at home.

In each case, the patient's adhesions were released and the tone of the ipsilateral pectoral muscle tone was normalized. In some cases, the adhesion released quickly within a few sessions, others required more sessions. In more resistant cases, the clinician tended to perform more clinical releases due to having a better mechanical advantage than the patient to more effectively release the adhesion. The length of time for resolution was found to be associated with the size and chronicity of the lesion, since adhesive scar tissue reportedly becomes more fibrotic and toughened over time (Stecco, 2004, p. 80). Additional indirect manual fascial release techniques were performed extending across the chest wall with respect to the entire myofascial plane, as myofascia can become chronically shortened due to the anterior shoulder position. These techniques were performed with the goal of returning the shoulder back to a neutral position along with chest stretches to further lengthen the shortened myofascial chest wall tissues. Mobilization techniques to the costosternal articulations helped to restore chest wall motion and additional neuromotor and myofascial manual techniques and exercises were provided (the description of these methods is beyond the scope of this paper) to establish improved postural shoulder neutrality. Once a more neutral shoulder position was established, asymmetrical myofascial load to the cervicodorsal spine was reduced. With a more neutral posture achieved we were able to employ additional effective rehabilitative strategies.

Summary

This case series brings to light the presence of the MPFL and its potential impact on the chest wall, including the local musculature, fascia and joints. The increasing understanding of the fascial system's role as part of the 'locomotor apparatus' role in sensorimotor and load transfer activities along kinetic chains also helps one to better understand the postural torsional consequences of the asymmetrical anterior shoulder, a consequence related to axial sagittal collapse. As the shoulder rolls forward, the cervicodorsal spine is loaded and traverses anteriorly and asymmetrically toward the unilateral forward shoulder. This ipsilateral postural syndrome involving pain and paresthesia to the entire upper quarter can ensue secondary to the loading of the myofascial tissues, altered mechanics, and stresses to the joints and describes the TUCS.

This was a retrospective, observational study and not a clinical trial. The authors of this paper feel that these observations warrant further research to include prospective studies with clearly defined outcome measures. Histological studies are needed to further understand the nature of this lesion in addition to research of etiological factors and incidence rates among various subgroups. Finally, we recommend further investigation of how the TUCS, affects, interacts or is affected by conditions such as scoliosis and other spinal deformities of varying etiologies.

Informed consent

Written informed consent has been obtained from all patients to use their anonymous data to discuss their cases or publish their pictures.

Conflict of interest statement

The authors declare there is no conflict of interest.

References

Albee, F., 1927. Myofascitis. A pathological explanation of many apparently dissimilar conditions. Am. J. Surg. 3, 525—533.

Blau, J.N., 2005. Feeling and seeing headaches. J. Headache Pain 6 (1), 10—19. http://dx.doi.org/10.1007/s10194-005-0143-8.

Bogduk, N., 2014. The neck and headaches. Neurol. Clin. 32 (2), 471—487. http://dx.doi.org/10.1016/j.ncl.2013.11.005.

Bogduk, N., Marsland, A., 1986. On the concept of third occipital headache. J. Neurol. Neurosurg. Psychiatry 49 (7), 775—780.

Cheville, A.L., Tchou, J., 2007. Barriers to rehabilitation following surgery for primary breast cancer. J. Surg. Oncol. 95 (5), 409—418. http://dx.doi.org/10.1002/jso.20782.

Please cite this article in press as: Morris, C.E., et al., The Torsional Upper Crossed Syndrome: A multi-planar update to Janda's model, with a case series introduction of the mid-pectoral fascial lesion as an associated etiological factor, Journal of Bodywork & Movement Therapies (2015), http://dx.doi.org/10.1016/j.jbmt.2015.08.008

Cohen, J., Schneider, M., 1990. Receptor-tonus technique: an overview. Chiropr. Tech. 2 (1), 13–16.
Crawford, J.S., Simpson, J., Crawford, P., 1996. Myofascial release provides symptomatic relief from chest wall tenderness occasionally seen following lumpectomy and radiation in breast cancer patients. Int. J. Radiat. Oncol. Biol. Phys. 34 (5), 1188–1189.
De Silva, M., 1986. The costoclavicular syndrome: a 'new cause'. Ann. Rheumatic Dis. 45 (11), 916–920. http://dx.doi.org/10.1136/ard.45.11.916.
Eslick, G.D., Jones, M.P., Talley, N.J., 2003. Non-cardiac chest pain: prevalence, risk factors, impact and consulting — a population-based study. Alimentary Pharmacol. Ther. 17 (9), 1115–1124. http://dx.doi.org/10.1046/j.1365-2036.2003.01557.x.
Frank, C., Kobesova, A., Kolar, P., 2013. Dynamic neuromuscular stabilization & sports rehabilitation. Int. J. Sports Phys. Ther. 8 (1), 62–73.
Goldthwaite, J., 1945. Bodymechanics. JB Lippincott, Philadelphia.
Greenman, P., 1996. Rib Cage Principles of Manual Medicine, second ed. Lippincott Williams & Wilkins, Baltimore, pp. 137–278.
Janda, V., 1968. The significance of muscular faulty posture as pathogenetic factor of vertebral disorders. Arch. Phys. Ther. (Leipz) 20 (2), 113–116.
Janda, V., 1972. What is the typical upright posture in man? Cas. Lek. Cesk 111 (32), 748–750.
Janda, V., 1994. Muscles and cervicogenic pain syndromes. In: Grant, R. (Ed.), Physical Therapy of the Cervical and Thoracic Spine. Churchill-Livingstone, New York, pp. 195–216.
Kalke, S., Perera, S.D., Patel, N.D., Gordon, T.E., Dasgupta, B., 2001. The sternoclavicular syndrome: experience from a district general hospital and results of a national postal survey. Rheumatol. Oxf. 40 (2), 170–177.
Kepics, J., 2004. Physical therapy treatment of axillary web syndrome. Rehabil. Oncol. 22, 21–22.
Kim, Sun Mi, Park, Jeong Mi, 2004. "Normal and abnormal US findings at the Mastectomy Site 1." Radiographics 24 (2), 357–365.
Kobesova, A., Morris, C.E., Lewit, K., Safarova, M., 2007. Twenty-year-old pathogenic "active" postsurgical scar: a case study of a patient with persistent right lower quadrant pain. J. Manip. Physiol. Ther. 30 (3), 234–238. http://dx.doi.org/10.1016/j.jmpt.2007.01.005.
Lacomba, M.T., del Moral, O., Coperias Zazo, J., et al., 2010. Incidence of myofascial pain syndrome in breast Cancer surgery: a prospective study. Clin. J. Pain 26 (4), 320–325, 310.1097/AJP.1090b1013e3181c4904a.
Lewin, P., 1955. The Back and its Disc Syndromes. Lea & Febiger, London.
Lewit, K., Olsanska, S., 2004. Clinical importance of active scars: abnormal scars as a cause of myofascial pain. J. Manip. Physiological Ther. 27 (6), 399.
Mennell, J., 1920. Massage: It's Principles and Practice. J & A Churchill, London.
Miyatsuji, A., Matsumoto, T., Mitarai, S., et al., 2002. Effects of clothing pressure caused by different types of brassieres on autonomic nervous system activity evaluated by heart rate variability power spectral analysis. J. Physiol. Anthropol. Appl. Hum. Sci. 21 (1), 67–74.
Morris, C.E., Greenman, P.E., Bullock, M.I., et al., 2006. Vladimir Janda, MD, DSc: tribute to a master of rehabilitation. Spine (Phila Pa 1976) 31 (9), 1060–1064. http://dx.doi.org/10.1097/01.brs.0000214879.93102.4e.
Muntan, C.D., Sundine, M.J., Rink, R.D., et al., 2000. Inframammary fold: a histologic reappraisal. Plast. Reconstr. Surg. 105 (2), 549–556 discussion 557.
Oo, M., Myint, Z., Sakakibara, T., Kasai, Y., 2012. Relationship between brassiere cup size and shoulder-neck pain in women. Open Orthop. J. 6, 140–142. http://dx.doi.org/10.2174/1874325001206010140.
Page, P., 2011. Shoulder muscle imbalance and subacromial impingement syndrome in overhead athletes. Int. J. Sports Phys. Ther. 6 (1), 51–58.
Palmer, D., 1914. The Chiropractor. Beacon Light Printing, Los Angeles.
Rolf, I., 1990. Rofing and Physical Realities, Reprint ed. Healing Arts Press, Vermont.
Ryan, E.L., 2000. Pectoral girdle myalgia in women: a 5-year study in a clinical setting. Clin. J. Pain 16 (4), 298–303.
Schleip, R., 2003. Fascial plasticity — a new neurobiological explanation. JBMT 7 (1), 11–19.
Schleip, R., Vleeming, A., Lehmann-Horn, F., Klingler, W., 2007. Letter to the Editor concerning "A hypothesis of chronic back pain: ligament subfailure injuries lead to muscle control dysfunction" (M. Panjabi). Eur. Spine J. 16 (10), 1733–1735. http://dx.doi.org/10.1007/s00586-006-0298-2 author reply 1736.
Smoot, B., Wong, J., Cooper, B., et al., 2010. Upper extremity impairments in women with or without lymphedema following breast cancer treatment. J. Cancer Surviv 4 (2), 167–178. http://dx.doi.org/10.1007/s11764-010-0118-x.
Spencer, L., Briffa, K., 2013. Breast size, thoracic kyphosis & thoracic spine pain – association & relevance of bra fitting in post-menopausal women: a correlational study. Chiropr. Man. Ther. 21 (1), 20. http://dx.doi.org/10.1186/2045-709X-21-20.
Stecco, A., Macchi, V., Stecco, C., et al., 2009a. Anatomical study of myofascial continuity in the anterior region of the upper limb. J. Bodyw. Mov. Ther. 13 (1), 53–62. http://dx.doi.org/10.1016/j.jbmt.2007.04.009.
Stecco, A., Masiero, S., Macchi, V., et al., 2009b. The pectoral fascia: anatomical and histological study. J. Bodyw. Mov. Ther. 13 (3), 255–261. http://dx.doi.org/10.1016/j.jbmt.2008.04.036.
Stecco, L., 2004. Fascial manipulation of Musculoskeltal pain. In: Day, J.A. (Ed.).
Still, A., 1902. Philosophy and Mechanical Principles of Osteopathy. Hudson-Kimberly Publishing Company, Kansas City, MO.
Stochkendahl, M.J., Christensen, H.W., 2010. Chest pain in focal musculoskeletal disorders. Med. Clin. North Am. 94 (2), 259–273. http://dx.doi.org/10.1016/j.mcna.2010.01.007. S0025-7125(10)00008-8 [pii].
Svavarsdottir, A.E., Jonasson, M.R., Gudmundsson, G.H., et al., 1996. Chest pain in family practice. Diagnosis and long-term outcome in a community setting. Can. Fam. Physician 42, 1122–1128.
Thigpen, C.A., Padua, D.A., Michener, L.A., et al., 2010. Head and shoulder posture affect scapular mechanics and muscle activity in overhead tasks. J. Electromyogr. Kinesiol 20 (4), 701–709. http://dx.doi.org/10.1016/j.jelekin.2009.12.003.
Travell, J., Rinzler, S., 1953. The myofascial genesis of pain. Postgrad. Med. J. 11, 425–434.
Travell, J., Simons, D.G., Simons, L., 1992. Myofascial Pain and Dysfunction; the Trigger Point Manual: the Lower Extremity, Vol. 2. Williams & Wilkins, Lippincott.
Travell, J., Simons, D.G., Simons, L., 1998. Myofascial Pain and Dysfunction; the Trigger Point Manual: Upper Half of Body, , second ed.Vol. 1. Williams & Wilkins, Lippincott.
Vleeming, A., Pool-Goudzwaard, A.L., Stoeckart, R., et al., 1995. The posterior layer of the thoracolumbar fascia. Its function in load transfer from spine to legs. Spine (Phila Pa 1976) 20 (7), 753–758.

Please cite this article in press as: Morris, C.E., et al., The Torsional Upper Crossed Syndrome: A multi-planar update to Janda's model, with a case series introduction of the mid-pectoral fascial lesion as an associated etiological factor, Journal of Bodywork & Movement Therapies (2015), http://dx.doi.org/10.1016/j.jbmt.2015.08.008