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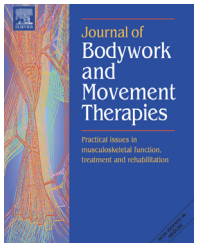

The Torsional Upper Crossed Syndrome: A multi-planar update to Janda's model, with a case series introduction of the mid-pectoral fascial lesion as an associated etiological factor

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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.

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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