The Thoracic Spine and Rib Cage: Musculoskeletal Evaluation and Treatment

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Chapter 10

Myofascial Considerations in Somatic Dysfunction of the Thorax

Jeffrey J. Ellis and Gregory S. Johnson

Somatic dysfunction is defined as impaired or altered function of related components of the somatic (body framework) system; that is, the skeletal, arthrodial, and myofascial structures and related vascular, lymphatic, and neural elements [1]. The specific identification and timely amelioration of this dysfunction has been the challenge and goal of manual medicine since its inception.

Somatic dysfunction of the thoracic spine and rib cage is ubiquitous in nature with respect to patients complaining of spinal pain and produces a myriad of subjective and objective findings that challenge the clinician to arrive at a concise and accurate diagnosis. In fact, the thoracic vertebral column has been termed "one of the elusive frontiers in spine research" [2]. Recognition of the neuromusculoskeletal and biomechanical attributes and potentials of this region is very helpful in formulating appropriate treatment protocols as well as prognoses. As concepts of vertebral and rib cage motion, both normal and abnormal, are challenged, and even redefined by some [2, 3], increasing attention is being paid to the "driving force" behind this movement—the myofascial system and its potential role in creating and perpetuating somatic dysfunction [4–9].

Long recognized as a potential source of pain in biomechanical somatic dysfunction of the vertebral column [9–15], numerous terms and classifications have been proposed as designates for primary myofascial dysfunction (MFD), including muscular rheumatism [16–18], myalgia [19–21], interstitial myofibrositis [22], myofascial pain syndrome [23], myofaciitis [24–27], and fibrositis [28]. More recently, the myofascial system has received attention because of its causative or primary role in creating biomechanical sources of somatic dysfunction as opposed to its being intrinsically the direct source of pain [5, 6, 29–31].

Myofascial structures are those tissues that are either musculoskeletal (in this case) or connective in makeup and origin. The interrelationship between these tissues and the mechanical dysfunction that can be directly linked to their aberrant function demands the closest attention.

In the thoracic spine and rib cage, structural lesions can be defined through biomechanical nomenclature and include non-neutral type II, FRS (flexed, rotated, and side-bent) or ERS (extended, rotated, and side-bent) dysfunctions of a single vertebral segment; type I, neutral vertebral dysfunctions; and structural, torsional, and respiratory rib cage lesions. The myofascial system frequently provides insight into the identification and causation of underlying somatic dysfunction and presenting symptomatology. Myofascial structures may be observed from four specific perspectives with respect to thoracic spine and rib cage somatic dysfunction (Table 10.1).

First, their diagnostic value should be considered. In the presence of aberrant positional and motion dysfunction of the vertebral column or rib cage, adjacent myofascial structures will often have tissue texture abnormalities. These findings have been de-

Table 10.1. The Role of Myofascial Tissue in the Evaluation, Pathogenesis, and Treatment of Structural Somatic Dysfunction of the Thorax

Diagnostic role
Facilitated segment
Tissue texture abnormality
Primary motion restrictor
Cause of structural dysfunction
Use in MET/PNF
Primary activation force
Source of entrapment/tunnel syndrome
Myofascial/fiber-osseous tunnels

scribed in the osteopathic literature as being consistent with the presence of a "facilitated segment" [1, 32–37]. Korr [34], Mitchell et al. [35], Greenman [1], and others have described the myofascial changes that represent a facilitated segment, including hypertonicity, increased temperature, hyperesthesia, and concomitant fascial dysfunction of the immediate surrounding tissues. These changes are commonly present in the muscles of a single segment of the vertebral column (including the transversospinalis groups located in the anatomic groove between the transverse and spinous process) and of the rib cage (including the levator costae and intercostal muscles). These changes may, however, be analogous to an "idiot light" on a car (e.g., the oil gauge), which when lit, although meriting attention, belies a problem elsewhere. Tissue texture abnormalities in these regions often reflect underlying biomechanical dysfunction and should warrant further investigation. Undue preoccupation with these "lights" and the correlative subjective complaints that accompany biomechanical dysfunction elsewhere, frequently misguide and detract the clinician from formulating an appropriate diagnosis and implementing proper treatment. However, in the presence of a chronic facilitated segment, compensatory myofascial dysfunction may develop, become symptomatic, and warrant treatment in combination with the primary source of dysfunction.

Second, myofascial structures should be recognized for their potential role as primary motion restrictors in somatic dysfunction [7, 38–40]. In the presence of aberrant myokinematics of the thorax with respect to increased muscle tone, deficient muscle play/accessory mobility, changes in strength or neuromuscular responsiveness, and subsequent alteration in functional excursion/length, motion dysfunction of the related articular structures may result. This diminished functional capacity of the correlative articular structures (thoracic vertebral and rib segments) provides the environment for aberrant arthrokinematics, precipitating the occurrence of a cascade of possible chronic, degenerative changes [41].

In addition, because of the initial myofascial dysfunction, the neuromuscular system commonly functions inefficiently and is unable to provide the proper motor control and intrinsic stabilization necessary to protect and produce efficient function at these related segments [42]. This furthers the degenerative cascade through the repeated trauma of inefficient and improper movement patterns, both segmentally (arthrokinematically) and as an entire kinetic chain (osteokinematically).

Third, myofascial tissues are the operational focus during corrective treatment procedures such as muscle energy technique (MET) and proprioceptive neuromuscular facilitation (PNF) techniques. Lever principles (short and long) are used during METs to localize and normalize articular structures. These techniques, however, require tremendous specificity and rely on the presence of normal kinematics of related myofascial tissues for appropriate localization and to be efficacious. Normalization of MFD of these tissues may be required before their utilization in MET.

Finally, consideration must be given to the role myofascial tissues play in entrapment or tunnel syndromes of the neurovascular structures that pass through or reside in the thorax [13, 43, 44]. As they emerge from their origin in the spinal cord and until they reach their effector organs, nerves must pass through bony, fibrous, osteofibrous, and fibromuscular tunnels, where they risk potential compression, damage, and impairment of their end function [45]. Careful assessment of related myofascial tissues in combination with specifically selected neural tension tests [43, 44] will allow specific identification and isolation of causative myofascial dysfunction.

The purpose of this chapter is to recognize the varied influence and diagnostic information the myofascial tissues of the thorax can provide in managing somatic dysfunction of this region. In particular, the reader will be exposed to:

1. The relevant anatomic and biomechanical attributes of the myofascial structures of the thoracic spine and rib cage.

2. An enumeration and correlation of the specific myofascial structures that characterize nonresponsiveness to treatment, or recurrence in thoracic spine and rib cage articular (position/motion) dysfunctions.
3. An algorithmic methodology for evaluating and enumerating specific myofascial structural dysfunction.
4. An organized framework, methodology, and principles for treating myofascial dysfunction.
5. Relevant case studies that demonstrate the effectiveness of addressing myofascial dysfunction in the presence of movement dysfunction of the thoracic spine and rib cage.

Myofascial Tissue

The body is composed of four primary soft tissues: epithelial, muscle, nerve, and connective [46–48]. The interrelationship of muscle and connective tissue (i.e., myofascial tissue) provides for both normal biomechanics, which support static and dynamic activities (9), and pathomechanics in the dysfunctional state.

Connective tissue chiefly comprises ligaments and tendons (regular or dense) as well as aponeuroses, fascia, synovial membranes, joint capsules, and intrinsic elements of muscle (dense and loose irregular) [47, 48]. Continuous throughout the entire body, the fascial system interconnects with tendons, aponeuroses, ligaments, capsules, peripheral nerves, and intrinsic elements of muscle [49].

Connective Tissue: Fibrous Component

Connective tissue may be organized into two distinct components: fibrous and nonfibrous. The fibrous components consist primarily of collagen and elastin fibers. Irregular and regular connective tissues are distinguished by the periodicity and direction of fibers within each. Irregular connective tissue is recognized by its multidirectional fiber orientation, which provides the necessary strength, in all directions, required for structures such as capsules, aponeuroses, synovial membranes, and fascia. Regular connective tissue is distinguished by a uniplanar or linear fiber orientation, which provides the tensile strength required by ligaments and tendons [46–48, 50].

Connective Tissue: Nonfibrous Component

The nonfibrous portion of connective tissue consists primarily of amorphous ground substance, which is a viscous gel composed of long chains of carbohydrate molecules called mucopolysaccharides, known as glycosaminoglycan (GAG), bound to a protein and water (60–70% of net content). Collectively termed proteoglycan aggregate [48, 51], the nonfibrous portion of connective tissue plays a key role in providing a lubricous state in which collagen and elastin fibers can coexist and thrive. In addition, the proteoglycan aggregate provides appropriate spacing, described as critical fiber distance (CFD), between the fibrous elements and is necessary for normal mobility.

Viscoelastic Properties of Connective Tissue

Collagen fibers are relatively inextensible; however, they have the biomechanical attribute of being able to withstand high tensile forces [47, 48]. Fascia is composed of wavy, multidirectional, multiplanar collagen fibers and elastin fibers and is therefore highly extensible as well as strong. Force, or the load applied to the tissue, is defined as *stress*, whereas the tissue deformation realized is termed *strain* [48, 52]. This stress/strain relationship (Fig. 10.1) depicts the viscoelastic response connective tissue undergoes when force is applied and includes the initial toe region elastic, plastic, and, finally, failure ranges. This collagenous elongation or deformation is known as *creep* and occurs through the process of hysteresis (Fig. 10.2) [52–54].

It is through these physiologic and biomechanical properties that soft-tissue mobilization techniques are purported to demonstrate their efficacy. Collagen has in fact been found to realign, strengthen, and orient its fibroblasts and the resulting newly synthesized fibers in response to the type, amount, duration, and frequency of the stress applied [55–57]. In short, newly deposited collagen fibers become oriented in the direction of stress [58–61]. This process of remodeling in accordance with stresses imposed is known as Wolf's Law of C.T. [55–58] and will be a prime consideration in our attempts to influence the connective tissue system with myofascial mobilization technique.

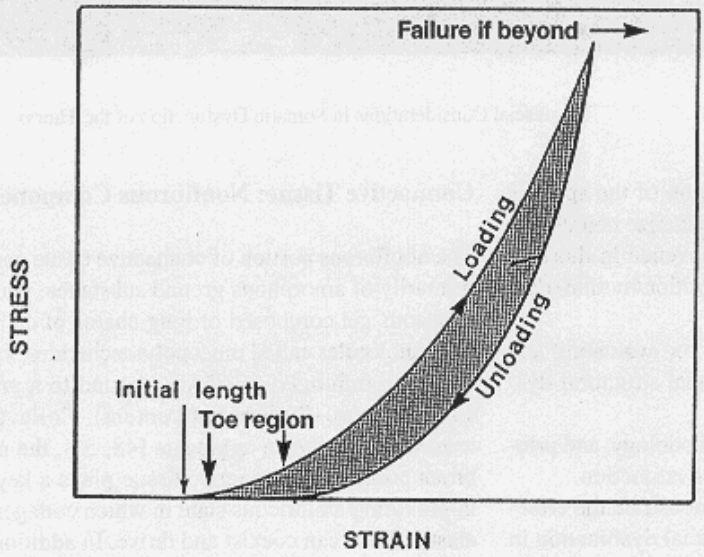


Figure 10.1. Stress/strain curve. This curve demonstrates the viscoelastic behavior and resulting deformation of connective tissue to externally applied stress. (Reprinted with permission from N Bogduk N, LT Twomey. Clinical Anatomy of the Lumbar Spine. New York: Churchill Livingstone, 1987.)

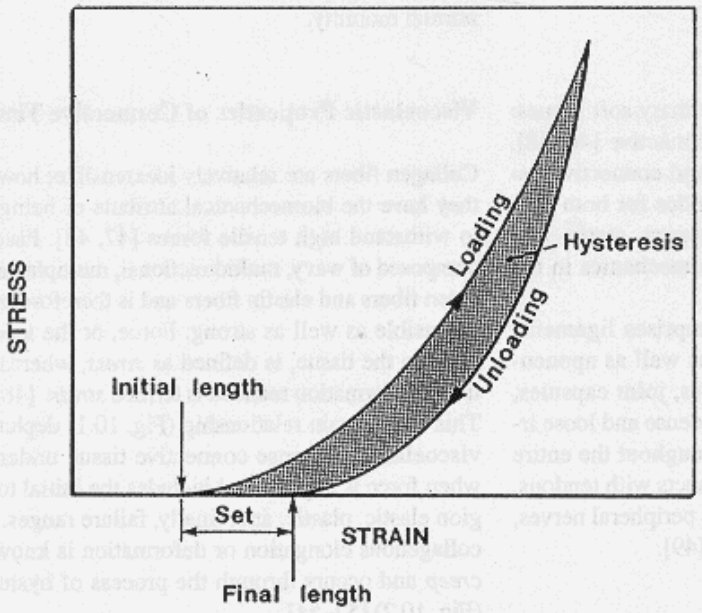


Figure 10.2. Hysteresis. This curve demonstrates tissue deformation with loading and the permanent length changes realized after stress is removed "set." (Reprinted with permission from N Bogduk, LT Twomey. Clinical Anatomy of the Lumbar Spine. New York: Churchill Livingstone, 1987.)

Effects of Immobilization

The effects of immobilization of articular capsules have been well documented [48, 50, 62–64], and the information provided may be extrapolated for use in observing what occurs to other connective tissues including fascia. The chief effects of immobilization are summarized in Table 10.2.

These changes equate to stiffer, harder tissues, a reduction in muscle play and functional elongation, and an overall diminution in fascial mobility secondary to biomechanical aberrancies. Myofascial mobilization efforts are directed at normalizing these aberrancies while keeping in mind the histologic and biochemical reasons for this presentation.

Table 10.2. Effects of Immobilization on Connective Tissue

Reduction in glycosaminoglycan (especially hyaluronic acid, which maintains a high affinity for H_2O
Subsequent reduction in H_2O
Thixotropic state according to dehydration
Increased tissue rigidity and stiffness
Production of abnormal cross-link formation
Decreased and altered spacing between collagen fibers (decreased critical fiber resistance)
Diminished fiber glide
Fatty infiltrate within spaces, which potentially matures to become scar tissue
Random orientation and deposition of newly synthesized collagen fibers

Muscle

Three types of muscles are found in the human body: cardiac muscle; smooth (nonstriated or involuntary) muscle, which lines the hollow internal organs; and skeletal (striated or voluntary) muscle, which attaches to the skeleton via tendons, causes movement to occur and accounts for 40–45% of total body weight [52].

Performing both static and dynamic activities, skeletal muscle, which will be our focus, consists of two basic components: muscle fibers (the contractile component), and connective tissue (the noncontractile component) [52]. The fiber, the structural unit of skeletal muscle, is virtually sheathed in connective tissue, first as a single fiber covered with loose endomysium, then as various sized bundles or fascicles covered with dense perimysium, and finally as muscle bellies covered with epimysium [47].

An intimate relationship exists between the contractile and noncontractile elements of skeletal muscle. The contractile portion provides force production, relaxation, and extensibility, whereas the noncontractile portions provide for space, the lubrication of the contractile elements, and the elasticity required by muscle for appropriate broadening, lengthening, and muscle play.

Numerous authors have written about the histologic and biomechanical effects of trauma and immobilization in the form of microscopic and macroscopic changes to the myofascial unit [63, 65–67]. These dysfunctions include alteration of tone (secondary to impaired peripheral and central innervation); hypertonicity with localized trigger points (contraction) [14]; diminution of motor recruitment and control [39, 41, 68]; a decrease in functional excursion/muscle length secondary to sarcomere loss [62, 66, 67, 69, 70]; diminished accessory motion of connective tissue elements (contracture); and decreased muscle play. It is these microscopic and macroscopic changes that combine to create abnormal tension vectors and levers that alter the normal homeostatic balance that exists in the biokinetic chain.

Myofascial Dysfunction: Contracture/Contraction/Cohesion-Congestion (Fluidochemical)

In the consideration of motion dysfunction of the thoracic spine and rib cage, as well as the entire vertebral column, careful attention must be placed on defining the causative or contributory barriers to normal movement. Three theoretical models for the manifestation of MFD are contracture, contraction, and cohesion-congestion. Elements of each of these categories are presented in Table 10.3.

Table 10.3. Theoretical Models for the Manifestation of Myofascial Dysfunction

Contracture	Contraction	Cohesion-Congestion
Inert/noncontractile	Muscle contraction	Fluidochemical
Capsular/fascial fibrotic	Muscle holding	Edematous/dehydrated
Abrupt/firm end-feel	Hypertonicity/spasm	Boggy/stiff/reactive
Chronic	Acute/distress	Acute/chronic

Contracture

Contracture describes those myofascial structures (particularly connective tissues) that have undergone some degree of fibrotic alteration that makes their end-feel stiffer, harder, and less resilient or elastic, with associated shortening [5, 14, 58]. This may be secondary to various and numerous biomechanical and biochemical causes but yields as its end result specific characteristics including microscopic cross-linking of collagen fibers with diminution of fiber glide; alterations in tissue creep capacities [71]; alterations in tissue layer mobility, resulting in adhesion to underlying myofascial or osseous structures [29]; a change in mobility of intramuscular septum; and posttraumatic scarring. This definition of contracture should be distinguished from other works that classify "contracture" as the sustained intrinsic activation of the contractile mechanism of muscle fibers [72].

Contraction

The classification *contraction* may be used synonymously with spasm and is defined as increased tension with or without shortening of a muscle caused by involuntary motor nerve activity that cannot be stopped by voluntary relaxation [73]. This hypertonicity is usually associated with an increased level of tissue reactivity to palpation and often correlates with articular dysfunction at the same level. The tissue end-feel encountered is described as reactive, firm, painful, and frequently accompanied by increased tissue temperature. In addition, there may be localized signs of inflammation including swelling or edema.

Cohesion-Congestion

Webster's New Collegiate Dictionary [74] defines cohesion as "the act or process of sticking together tightly" and congestion as "to bring together, to concentrate in a small or narrow space." "This category includes macro- and microcirculatory changes affecting fluidochemical transport and exchange (such as altered chemical exchange on a cellular level, impaired lymphatic flow, vascular stasis or ischemia, etc.), and considers various chemical substances which may influence myofascial tissue such as metabolites, electrolytes, hormones, neurotransmitters, neurogenic and nonneurogenic pain mediators, etc." [71]. This category includes those fluidochemical changes related to a diminution in tissue hydration that may go on to provide the foundation for biomechanical dysfunction as a sequela to ground substance dehydration and resulting changes within the fibrous and nonfibrous elements of connective tissue. It also, however, encompasses those situations in which there is an overabundance of tissue hydration with resulting biomechanical alteration.

Myofascial Anatomy of the Thorax

The myofascial tissues of the thorax, although multipurpose, maintain the distinction of providing one overriding function—respiration (Table 10.4). Together, the various tissues of the thoracic wall constitute a strong yet delicately regulated pump or bellows, providing the rigidity capable of resisting surrounding pressure, the mobility that allows active expansion and aspiration of air, and a resilience that imparts properties of elastic recoil [46]. In addition, these muscles act in an orchestrated fashion with the back musculature of the thoracolumbar and cervicothoracic regions to initiate and control functional movements of the thoracic spine and rib cage.

Understanding the anatomic topography and myokinematics of all of these muscles allows their normal and dysfunctional states to be more easily identified and managed. In addition, the pathomechanics of this region secondary to aberrant muscle function may be more commonly attributed to specific dysfunctional myofascial tissues as their anatomic presentation is understood. These muscles are listed topographically and in accordance with their function in Tables 10.4 through 10.10 (for further details, see Chapter 1).

Table 10.4. Muscles of the Thorax

Diaphragm
Intercostales externi
Intercostales interni
Intracostales
Triangularis sterni
Levatores costarum

Source: Adapted from H Gray. Anatomy, Descriptive and Surgical. Philadelphia: Running Press, 1974.

Table 10.5. Topographical/Layer Orientation of the Back Muscles of the Cervical, Thoracic, and Lumbar Spine

First Layer	Second Layer	Third Layer	Fourth Layer	Fifth Layer
Trapezius	Levator anguli scapulae	Serratus posticus superior	Sacral and lumbar regions:	Semispinalis dorsi
Latissimus dorsi	Rhomboideus minor	Serratus posticus inferior	Erector spinae	Semispinalis colli
	Rhomboideus major	Splenius capitis	Dorsal region:	Multifidus spinae
		Splenius colli	Ilio-costalis	Rotatores spinae
			Musculus accessorius ad iliocostalem	Supraspinales
			Longissimus dorsi	Interspinales
			Spinalis dorsi	Extensor coccygis
			Cervical region:	Intertransversales
			Cervicalis ascendens	Rectus capitis posticus major
			Transversalis cervicis	Rectus capitis posticus minor
			Trachelo-mastoid	Obliquus capitis inferior
			Complexus	Obliquus capitis superior
			Biventer cervicis	
			Spinalis colli	

Source: Adapted from H Gray. Anatomy, Descriptive and Surgical. Philadelphia: Running Press, 1974.

The musculature of the back (cervicothoracic, thoracolumbar) must receive equal consideration because it exerts a significant influence on the overall static and dynamic posturing of the thoracic spine and rib cage. Although the postvertebral muscles show little if any rhythmic activity in quiet respiration, their chief function is primarily postural, maintaining erect posture against gravity. Dysfunction of these tissues (i.e., increased muscle tone, decreased play, alteration in strength or neuromuscular responsiveness, or changes in functional excursion) may produce significant postural changes and resultant dysfunction of the rib cage [75]. These muscles may be identified topographically from a larger perspective (see Table 10.5).

Muscles of respiration may also play a key role in the structural, torsional, or respiratory rib dysfunctions (see Chapter 8). These muscles have been categorized as primary and accessory muscles of inspiration and primary and accessory muscles of expiration (see Tables 10.6, 10.7) [11, 76].

Finally, the muscles of the abdomen must be considered because they exert influence on and support for the thoracolumbar region and the rib cage. These muscles are topographically divided into superficial and deep groups (see Table 10.8).

Although not discussed in this text, additional consideration should also be given to the muscles of the upper extremities, particularly the rotator cuff musculature, because dysfunction of the upper extremity can directly affect the scapula and clavicle and indirectly the rib cage and thoracic spine. Each muscle group of the thorax, back, abdomen, and upper extremity and their related fascial attachments should be considered in somatic dysfunction of this region.

Table 10.6. Muscles of Inspiration

Primary
Diaphragm
Levator costarum
External intercostals
Internal intercostals (anterior)
Accessory
Scaleni
Sternocleidomastoid
Trapezius
Serratus anterior and posterior superior
Pectoralis major and minor
Latissimus dorsi
Thoracic spine extensors
Subclavius

Source: Adapted from HO Kendall, FP Kendall, DA Boynton. Posture and Pain. Huntington, NY: Robert E. Krieger, 1977.

Table 10.7. Muscles of Expiration

Primary
Abdominal muscles
Internal oblique, external oblique, rectus abdominis
Transversus abdominis
Internal intercostals, posterior
Transversus thoracis
Accessory
Latissimus dorsi
Serratus posterior inferior
Quadratus lumborum
Iliocostalis lumborum

Source: Adapted from HO Kendall, FP Kendall, DA Boynton. Posture and Pain. Huntington, NY: Robert E. Krieger, 1977.

Table 10.8. Abdominal Muscles

Superficial
Obliquus externus
Obliquus internus
Transversus abdominis
Rectus abdominis
Deep
Psoas major
Psoas minor
Iliacus
Quadratus lumborum

Source: Adapted from H Gray. Anatomy, Descriptive and Surgical. Philadelphia: Running Press, 1974.

Pathomechanics and Sequelae of Myofascial Dysfunction

Kinetic Chain Principles

A kinetic chain is a series of interconnecting segments that affects the position, movement, potential for movement and shock absorption, and attenuation of forces that are transmitted through those segments. Intimately linked and supportive of the skeletal structures, the myofascial tissues play a key role in the way this chain functions.

Myofascial Joints "Above and Below"

The axiom that thorough assessment of an individual articulation demands examination of the immediate articulations above and below the dysfunctional articulation [63] is equally germane to the soft-tissue system. Through a series of direct and indirect attachments or junctions, all myofascial tissues maintain an interrelationship that allows biomechanical influence on one another. Dysfunction in one myofascial structure may have a profound effect on the functional ability of a distant, but related, second myofascial structure.

Gratz's Functional Joint

Gratz defined the space that exists within and between these junctions as well as all structures of the human body as "functional joints" [77]. He went on to define a functional joint as "a space built for motion." From a functional and biomechanical basis, the mobility provided by the presence of this "space" [77] has been designated "muscle play" [29]. This concept will be critical in understanding the subtle yet profound impact that loss of accessory motion within the myofascial structures can have on the entire kinetic chain. The aberrance, or loss of normal "functional joint mobility" as defined by Gratz, should prompt one to consider the mechanical interface that exists between virtually all structures in the body. From a microscopic, cellular perspective to a macroscopic observation of structures such as muscle bellies, fascial sheaths, and underlying bony structures, the available spacing and mechanical interface relationship that exists must be considered, especially when attempting to restore normal mobility to the kinetic chain.

Myofascial Dysfunction in Primary and Compensatory Vertebral and Rib Dysfunction

Of primary importance in determining and sequencing treatment is whether presenting myofascial dysfunction represents the primary cause of a movement dysfunction or is a component of compensatory dysfunction. Primary sources of movement dysfunctions of the vertebral column, classified as type II non-neutral FRS or ERS dysfunctions (see Chapter 8), and of the rib cage, classified as structural, torsional, or respiratory, represent the causative agents of both aberrant position and motion of the involved motion segment. In the vertebral column, unisegmental muscles have been implicated as the possible source of

Table 10.9. Proposed Myofascial Sources of Thoracic Spine and Rib Cage Motion Dysfunction

Myofascial Structure	Potential Structural Dysfunction
Anterior scalenes	Superior subluxation of the first rib segment Exhalation restriction
Medial scalenes	Superior subluxation of the first rib segment Exhalation restriction
Posterior scalenes	Superior subluxation of the second rib Exhalation restriction
Pectoralis minor	Exhalation restriction External torsion
Transversospinalis muscles	Type II FRS/ERS dysfunction
Respiratory diaphragm	Inhalation restriction
Quadratus lumborum	Inhalation restriction 12th rib structural dysfuntion
Serratus posterior superior	Internal torsion ribs 2–4
Serratus posterior inferior	External torsion ribs 9–12
Latissimus dorsi	Exhalation restriction more frequent than inhalation restriction Lateral bucket-bail
Serratus anterior	Upper fibers: Inhalation restriction Lower fibers: Exhalation restriction, lateral bucket-bail
Internal intercostals (anterior)	Exhalation restriction
External intercostals	Exhalation restriction
Abdominals	Inhalation restriction
Iliocostalis lumborum	External torsion

myofascial dysfunction in primary movement dysfunction. These include the transversospinalis, multifidus, and rotatores muscles [1, 3, 7, 36, 78]. In the rib cage, the anterior and medial scalenes, the levatores costarum, the intercostals, the pectoralis minor, the serratus posterior superior and inferior, the serratus anterior, and the latissimus dorsi muscle groups are common sources of primary movement dysfunction. Specifically, these muscles may contribute to primary vertebral or rib dysfunction by compensatorily shortening (secondary to increased tone-contraction or connective tissue shortening contracture), thereby producing a significant impact on the possibility of structural correction through MET or mobilization efforts alone. Table 10.9 provides a list of muscles and their possible myokinematic influence on thoracic spine and rib cage dysfunction. It must be remembered, however, that the respective tone and length of a muscle do not constitute the only considerations for possible dysfunction; the entire surrounding fascial structures must be considered as well.

In response to type II non-neutral vertebral dysfunction of the thoracic spine and structural rib cage dysfunction, compensatory responses of the vertebral column, described as type I neutral dysfunctions, are expected (see Chapter 8). Spanning three or more vertebral levels with osteokinematic sidebending to one side and concurrent rotation to the opposite side, myofascial dysfunction with adaptive shortening often occurs concomitantly. This is particularly true in cases of chronicity (Fig. 10.3).

These myofascial adaptations frequently include aberrant muscle play and functional excursion/length on the concave side of the type I curve and alteration of neuromuscular responsiveness/strength on the convex side. These compensatory changes typically include multisegmental muscles such as the iliocostalis and longissimus groups. Changes in muscle length, tone, play, or accessory motion responsible for the causation or perpetuation of type I vertebral dysfunctions must be addressed because they often contribute to nonresponsive or recurrent lesions elsewhere in the kinetic chain.

Although type I dysfunction often correlates with concomitant myofascial dysfunction in this region, it may also be related to and demands differentiation from at least 13 other possible causes (see Table 10.10).

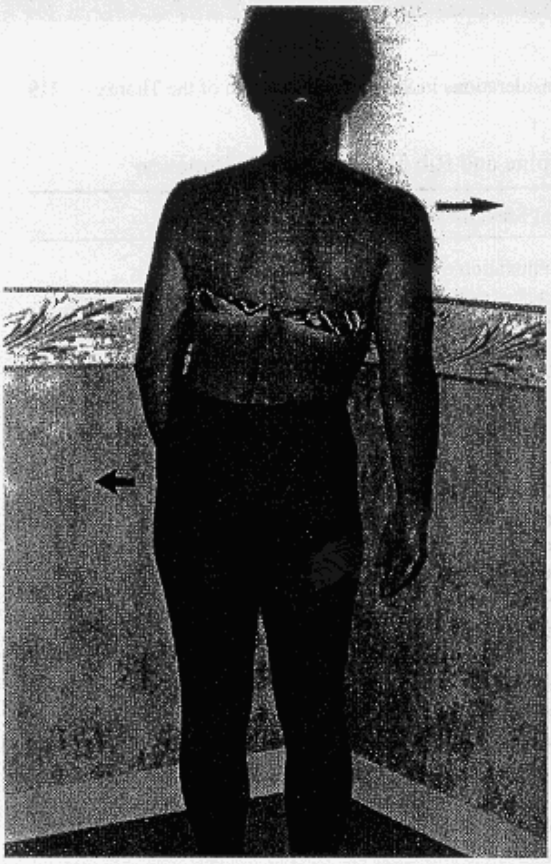


Figure 10.3. Type I neutral dysfunction of the thoracolumbar spine with thoracic sidebending to the left and concurrent rotation to the right.

Sequencing Treatment Strategies

Of paramount importance in determining treatment strategies are the identification and amelioration of myofascial sources of primary movement dysfunction. In the acute phase, these will typically be related to those sources of dysfunction classified as contraction, cohesion-congestion, or both (see Table 10.3) and will be most evident at or immediately adjacent to the dysfunctional vertebral or rib dysfunction. Correction of these dysfunctions should yield immediate changes in both position and motion characteristics of the involved motion segment. In addition, when these dysfunctions are present in the acute phase and are not associated with longstanding myofascial changes, compensatory myofascial dysfunctions (i.e., increased tone, muscle holding, superficial fascial dysfunction) will also normalize.

Equally important, however, is the identification of myofascial dysfunction associated with chronic, compensatory vertebral dysfunction (type I neutral dysfunction), which is often classified as "contracture" in nature (see Table 10.3). Because they are accompanied by adaptive myofascial changes, particularly alteration in muscle length/elongation and play/accessory mobility, failure to address these aberrancies may allow for poor correction of osteokinematic movement dysfunctions (i.e., type II non-neutral vertebral dysfunction or structural rib dysfunction). In addition, failure to normalize the myofascial structures associated with

Table 10.10. Thirteen Causes for the Presence of Type I, Neutral Dysfunction of the Vertebral Column, with Sidebending and Rotation Opposite

Type II nonneutral, vertebral dysfunction (FRS/ERS) below the type I dysfunction
Type II nonneutral, vertebral dysfunction (FRS/ERS) above the type I dysfunction
Subcranial dysfunction (particularly at the O/A articulation)
Rib cage dysfunction (structural or torsional)
MFD of the abdomen
Idiopathic scoliosis
Sacral base unleveling (sacroiliac joint dysfunction)
Innominate or iliosacral dysfunction
Structural leg length assymetries
Functional leg length assymetries (rearfoot pronation/supination deformities)
Adverse neural tension signs, upper extremity
Adverse neural tension signs, lower extremity
Visceral dysfunction

Source: JJ Ellis. LPI—Lumbo-Pelvic Integration, A Course Manual. Patchogue, NY: 1990.

compensatory vertebral dysfunction may provide the "environment" for primary movement dysfunctions to recur (i.e., recurrent tendencies).

Myofascial Assessment: CHARTS Methodology of Evaluation

Evaluation of somatic dysfunction of the thoracic spine and rib cage requires careful attention to detail. The use of an algorithmic approach will help the clinician avoid overlooking the smallest yet perhaps most significant of details. One such approach is the CHARTS methodology of evaluation [6] (Table 10.11). This system builds on the osteopathic evaluative acronym ART, which stands for *a*symmetry of bony landmarks, *r*ange of motion/mobility alteration, and *t*issue texture abnormalities [1, 36, 79]. In the CHARTS model, chief complaints, history (particularly recent biomechanical and systems review), and special tests (e.g., radiologic, blood analysis) are added for thoroughness and precision in arriving at a diagnosis. (See Chapters 3–7 for a thorough discussion of history and special tests.) In addition, tissue texture abnormalities, "T," have been embellished in keeping with this chapter's emphasis of the importance of this area.

Table 10.11. CHARTS Methodology of Evaluation

C: Chief complaint
H: Histories
Family history
Social/recreational history
Past medical history
Pharmacologic history
Current history/presenting dysfunction
A: Asymmetries of bony landmarks
Orthostatic postural assessment
Specific spinal/costal/extremity landmarks
R: Range of motion/mobility testing
Osteokinematic spinal/costal/extremity ROM
Arthrokinematic spinal/costal/extremity ROM
Special mobility testing
T: Tissue texture/tension/tonal abnormalities
Skin/fascial layer assessment
Muscle play/accessory mobility
Bony contour assessment
Functional excursion/length
Neuromuscular control/functional strength
S: Special tests
Neurologic screen
Vertebrobasilar clearance testing
Ligamentous integrity testing
Gait assessment
Radiologic screen
Laboratory profiles
Functional capacity/work hardening screen

Source: JJ Ellis. LPI—Lumbo-Pelvic Integration, A Course Manual. Patchogue, NY: 1990.

Evaluation and General Screening Procedures

Evaluation of myofascial tissues, "T" within the CHARTS methodology, requires consideration of several specific characteristics. These elements are assessed through static, dynamic, and physiologic movement patterns using a layer approach, which assesses tissues from the most superficial to the deepest—i.e., those inserting into bony contours (Table 10.12).

Static Postural Assessment

Static evaluation takes place with the patient standing, seated, prone, supine, and on all fours (quadruped). The evaluative process begins at the moment of initial visual contact with the patient. A keen sense of observation as the patient walks to the evaluative suite and then throughout the static postural screen will yield much information regarding static and dynamic postures, reactivity levels, muscle guarding/holding, gait deviations, tolerance to certain functional postures (e.g., sitting, standing), and perhaps appropriateness and psychological state. Ideally, the practitioner should provide an environment suitable for patient comfort and privacy, while allowing for unobstructed visual observation from a distance of 8–12 feet. Evaluation suites should be carpeted, adequately heated to allow disrobing, and have lighting arranged to avoid unnecessary shadowing or glare.

The static postural examination often yields valuable information regarding underlying osseous/articular structures as well as asymmetries and aberrancies within myofascial structures. This may directly correlate with altered motion and positional dysfunction within the thoracic vertebral column and

Table 10.12. Myofascial Assessment

- A. General screening procedures
  - 1. Static postural assessment
    - a. Skeletal observation
    - b. Soft tissue observation
  - 2. Dynamic postural assessment
    - a. Vertical compression testing (VCT)
  - 3. Physiologic movement patterns
    - a. Thoracic movement patterns
    - b. Upper extremity movements
    - c. Respiratory movements
    - d. Functional movement patterns (FMP)
- B. Skin and superficial fascial assessment
  - 1. Palpation
  - 2. Soft-tissue contours
  - 3. Skin condition
  - 4. Skin mobility
  - 5. Scar tissue
  - 6. Superficial/deep fascia
- C. Bony contours
  - 1. Thoracic spine
    - a. Vertebral segments
  - 2. Rib cage
    - a. Sternum
      - i. Manubrium
      - ii. Body
      - iii. Xiphoid
    - b. Clavicle
    - c. Rib segments 1–12
  - 3. Scapula
- D. Muscle assessment
  - 1. Muscle tone
  - 2. Muscle play/accessory
  - 3. Muscle length/functional
  - 4. Neuromuscular control

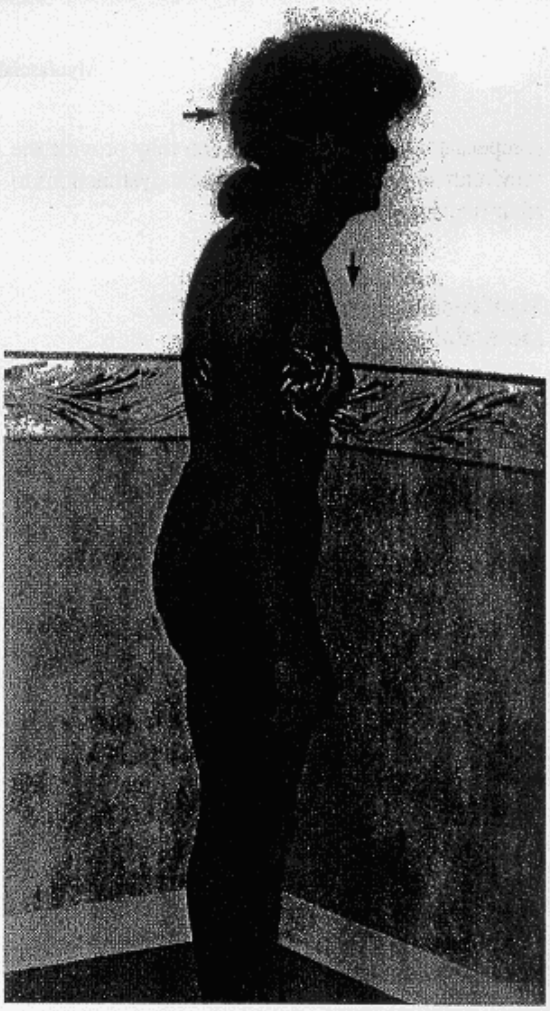


Figure 10.4. Forward head postural dysfunction with accompanying bilaterally protracted shoulders, depressed sternum, and thoracic kyphosis.

the rib cage. An example of this is a patient with forward head posture (FHP), bilaterally protracted shoulders with one greater than the other (the right shoulder in this example), a depressed sternum, and myofascial dysfunction of the pectoralis minor/major muscle complex (right greater than left) (Fig. 10.4).

This aberrant condition of the myofascial structures may be the primary cause of the resulting respiratory, exhalation dysfunction of the third, fourth, and fifth ribs on the right (see Chapter 8). In addition, asymmetric myofascial tightness may directly correlate with type I dysfunction in the thoracic spine.

Skeletal Observation

Beginning with posterior, anterior, and, finally, lateral views, static postural assessment should proceed in a caudal to cranial direction and include careful inspection of both skeletal and soft-tissue structures. Skeletal structures are evaluated with respect to symmetry from side to side and should include observation of aberrant position, spacing, size, and relative support. Anterior and posterior perspectives will provide information regarding skeletal and soft-tissue deviations of the craniovertebral structures, rib cage,

pelvis, and extremities in the coronal plane. Lateral shifts, type I dysfunctions, relative torsional and rotational patterns of the rib cage, pelvis and upper and lower extremities will be visualized in these postures. Lateral perspectives will yield information regarding sagittal deviations and include forward head posture (FHP), excessive cervicothoracic angulation ("dowager's deformity") or diminished, or absent cervical lordosis, sternal and sternomanubrial positions, shoulder posturing (protracted, retracted), rib cage resting position (exhalation, inhalation pattern), thoracolumbar and lumbopelvic angles, and genu recurvatum and/or flexion deformities.

The thoracic spine should have a smooth, uninterrupted curve reflecting a sagittal kyphosis with little or no coronal shift or lateral displacement. Sharp breaks or angulation in this contour often correlate with areas of focal hypermobility [80] and may present with increased muscle hypertonicity in the immediate surrounding tissues. In addition, these areas may reflect the presence of type II vertebral dysfunction or occur at transitional zones between two groups of type I vertebral dysfunction. Flattened areas within the thoracic sagittal kyphosis may relate to areas of hypomobility. Lateral deviations or alteration in the coronal plane may reflect the presence of type I multisegmental dysfunction.

In addition, inspection and side-to-side comparison of the individual relationships of rib segments (including spacing and superior, inferior, anterolateral and posterolateral contours), the scapula, and the clavicle should be considered. Asymmetric contours of the rib cage may yield valuable information regarding underlying somatic dysfunction (respiratory, torsional, or structural) in this region.

Soft-Tissues Observation

Soft tissues are also observed in the static postural screen from the posterior, anterior, and lateral perspectives. Beginning with a global view, the general patterns, types, and contours of soft tissues should be assessed. Initial impressions will often provide the direction of soft-tissue evaluation as central areas of dysfunction are discovered. Soft-tissue dysfunction of the thoracic spine and rib cage often has a proclivity for spiral and diagonal patterns, which course from a central, focal location of somatic dysfunction. These dysfunctions may zig-zag throughout the kinetic chain and provide a visual and palpatory link to primary sites of dysfunction. Observations should focus on several characteristics of the soft-tissue system including general contours, girth, muscle mass and development, symmetry from side to side, and three-dimensional relationships including depth, width, height, and length. Specifically, soft-tissue structures are evaluated for the presence of bands, restrictions, adhesions, and depressions within the superficial and deep fascial tissues. These characteristics may yield valuable information regarding underlying or adjacent osseous and articular somatic dysfunction as well as the functional characteristics and movement potential they possess. The presence of asymmetric contours or muscular development may provide valuable information regarding aberrant patterns of use, habitual postures, prior trauma, or improper training emphasis. Bands, contours, adhesions, and restrictions in the superficial tissues, often appearing as depressions or puckering of the dermis or epidermis, may direct attention to the possibility of aberrant superficial and deep fascial tissues, which often accompany or are responsible for underlying vertebral and rib cage dysfunction.

In addition, the examiner should look for and evaluate areas of focal neuromuscular activity or muscle holding because of their role as protective or pain avoidance mechanisms, or as part of aberrant postural or segmental mechanics. This is commonly seen in the scapulocostal region in the presence of forward head posture with abnormal muscle holding/neuromuscular activity of the levator scapulae muscle. In the chronic dysfunctional state, several aberrant static and dynamic dysfunctions may be seen in the sequelae that result from this abnormal muscle holding. These findings include cranial tilt to the ipsilateral side of increased muscle activity, elevated ipsilateral shoulder girdle, altered scapulocostal posturing with possible suprascapular and dorsal scapular nerve entrapment syndrome [43, 81], upper rib cage dysfunction, and upper thoracic spine dysfunction (Fig. 10.5).

Dynamic Postural Assessment

Vertical Compression Testing

Vertical compression testing (VCT) [29] is used to further assess the position, integrity, and force atten-

Figure 10.5. Myofascial dysfunction of the right levator scapulae muscle with increased muscle holding (contraction) secondary to postural dysfunction including forward head posture, protracted shoulders, cranial side tilting, and depressed sternum.

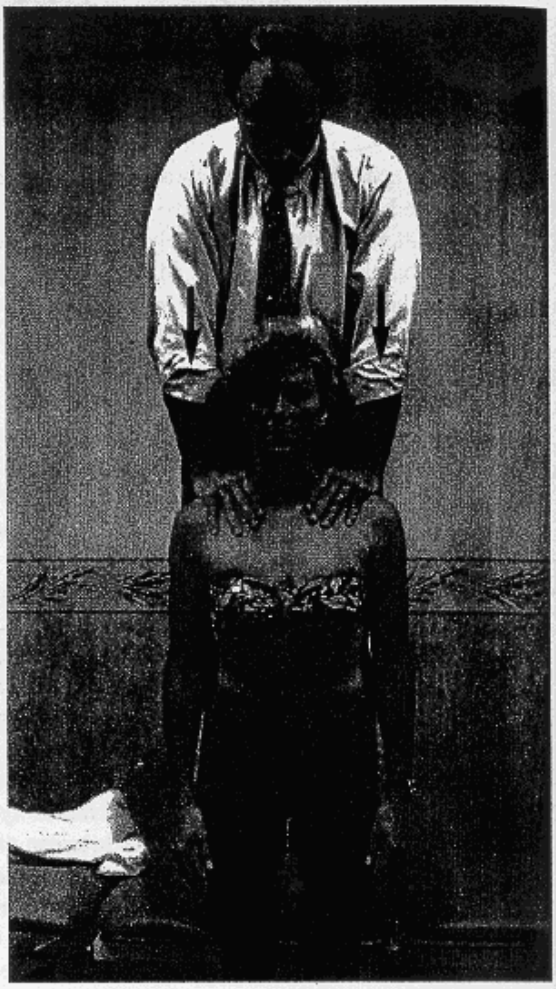


Figure 10.6. Vertical compression testing in the standing posture to assess postural dysfunction including the presence of type I and II vertebral dysfunctions.

uation characteristics of the vertebral column, pelvis, and lower extremities in various weight-bearing postures (i.e., standing and sitting). Aberrant positional dysfunction of the spine (i.e., type I and II) is both palpated and visually magnified through the vertical compression test. In addition, this testing procedure provides kinesthetic feedback to the patient regarding aberrant positional and motion dysfunction by emphasizing malalignment and then serving as positive feedback after correction and retesting. This often proves invaluable in enlisting the patient's support, understanding, and participation in a rehabilitation program.

The VCT is performed with the patient both standing and sitting and is accomplished by applying a gently increasing vertical compressive load through the shoulders and rib cage in a cranial-to-caudal direction (Figs. 10.6 and 10.7). This load may vary from ounces to several pounds as the clinician localizes to various levels of the vertebral column and lower extremity.

Vertical Compression Testing with Somatic Dysfunction

Observation of buckling, shearing, torsion, translation, or exaggeration of type I rotoscoliosis and increases in thoracolumbar kypholordosis should be noted (Fig. 10.8).

Sharp, acute apexes may indicate areas of focal hypermobility/instability and warrant further inves-

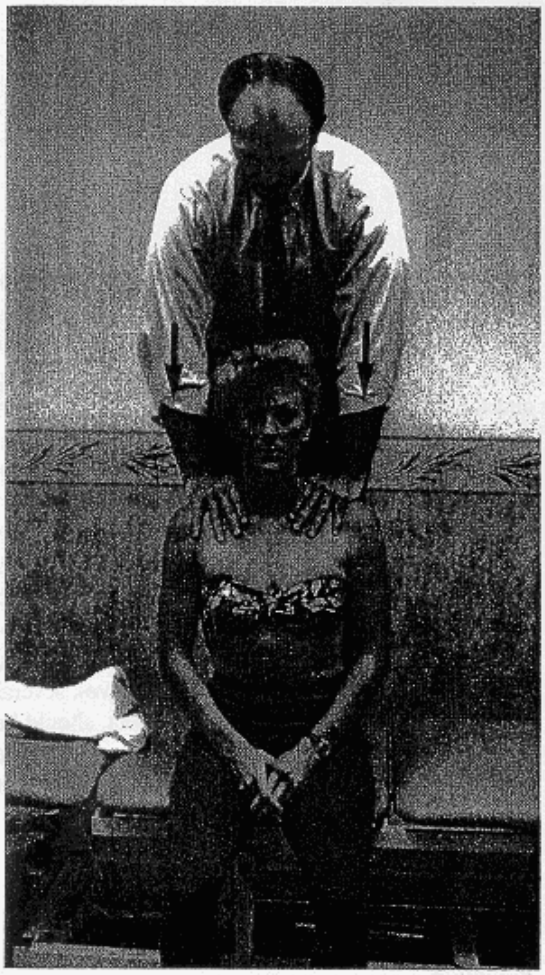


Figure 10.7. Vertical compression testing in the seated posture.

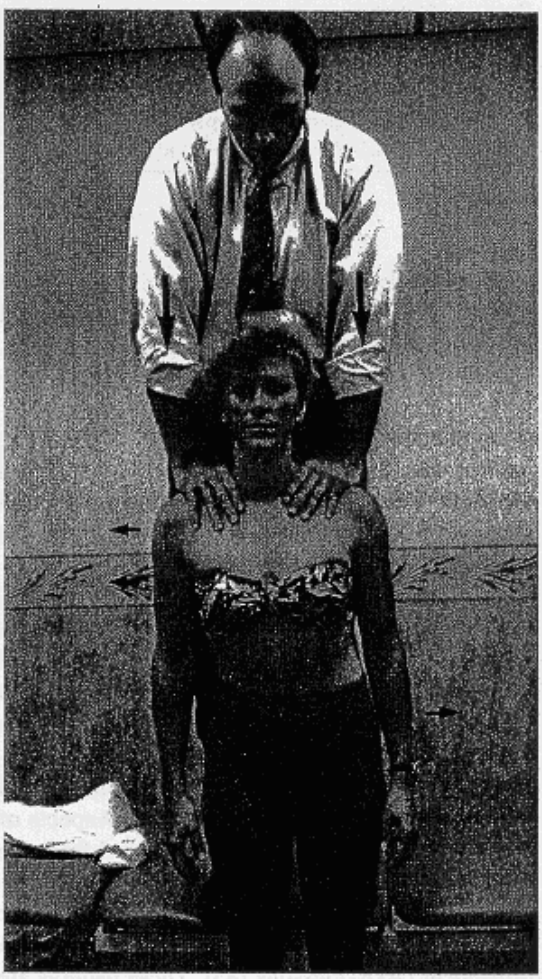


Figure 10.8. Vertical compression testing with shearing to the right secondary to the presence of a type I neutral dysfunction.

tigation (e.g., passive intevertebral motion testing [PIVMT], spring/translational testing and radiologic motion testing). These areas may also be related to type II non-neutral FRS/ERS vertebral dysfunctions and are frequently accompanied by tissue texture abnormalities (including increased muscle tone and decreased muscle play). Finally, these areas may occur at transitional zones in the vertebral column (i.e., cervicothoracic, thoracolumbar, and lumbosacral junctions) or at a rotoscoliotic curve. Left untreated, these areas may become sites of aberrant static and dynamic postural mechanics, resulting in neuromuscular imbalances with altered patterns of functional movement and, ultimately, focal areas of degenerative articular changes.

Although the emphasis of this diagnostic test is objective observation, the patient should also be encouraged to provide subjective feedback regarding symptoms during the procedure. Patients with a history of being "load sensitive" should be evaluated with caution, as this test may be provocational to them.

Physiologic Movement Testing

Dynamic postures used in assessing active range-of-motion (AROM)/combined movement testing include physiologic AROM of the thoracic spine in all planes (i.e., flexion, extension, side-bending, and rotation), AROM of the upper extremities, and respi-

Table 10.13. Observations During Active/Passive, Physiologic Motion Testing, and Functional Movement Patterns (FMP)

Excursion of movement
Quality of movement
Barriers to movement
Coordination of movement
Freedom of movement (both articular and soft tissue)
Substitutions to normal movement patterns
Subjective complaints before, during, and following

Table 10.14. Functional Movement Patterns (FMPs)

Pelvic clock
Unilateral hip rotation in various positions
Quadruped (arch/sag)
Unilateral lower extremity extension in prone
Lower trunk rotation in prone
Diagonal breathing
Sidelying arm circles

ratory patterns of breathing. Combined movement patterns are also performed and include lateral glide/translatory shear and quadrant patterns (i.e., combined flexion, side-bending, and concurrent rotation to the same side followed by extension, side-bending, and concurrent rotation to the same side). Assessment should include observation of both arthrokinematic and myokinematic function throughout each particular movement and should focus on several components (Table 10.13).

Functional movement patterns (FMPs) are defined as any functional motion that may be used to evaluate function, functional capacity, integration of movement segments, differentiation of individual segments, and sequencing of motion including both passive and active ranges of motion [4, 30]. In addition, motor recruitment and neuromuscular control can be assessed throughout the FMP. Aspects of these evaluative movements are derived from the proprioceptive neuromuscular facilitation (PNF) diagonal movement patterns [38, 82] and from the work of Feldenkrais and his Awareness Through Movement (ATM) lessons [83]. These patterns are applicable to both the spine and extremities and are listed in Table 10.14.

Once dysfunctions are identified, treatment is administered during the performance of a portion of or throughout the entire FMP. An example of an FMP, sidelying arm circle, is a pattern that is appropriate for both patients with a cervicothoracic upper quarter dysfunction, and patients with a lumbopelvic lower quarter dysfunction. This pattern is accomplished as follows:

With the patient in the sidelying position, the trunk is stabilized by the inferior hand holding the superior leg while movement occurs through the superior arm scribing the widest circle possible around the axis of the shoulder. In the efficient state, the hand maintains contact with the floor throughout the full arc of motion. This occurs through the orchestrated movement of the shoulder, shoulder girdle, rib cage, and thoracic and lumbar spine. In the presence of restrictions in any of the articulations or soft tissues of this region, hand contact to the floor will be altered, as will the synchronous motion throughout the arc. The practitioner should observe and palpate for specific motion and dysfunctional tissue barriers. Treatment is begun while the patient performs the FMP, moving in and out of the restricted range while soft-tissue mobilization is concurrently performed. During physiologic movement patterns (both passive and active), several variables, including response to treatment, should be observed while concurrently palpating the respective tissues for additional points of restriction and aberrant motion (Fig. 10.9).

Skin Assessment

Palpation

Whereas the observational evaluation is used to identify myofascial structures that may be dysfunctional and contributing to symptomatology, manual palpation is the primary modality used to assess the condition of the myofascial tissues. Developing and refining one's skill in palpation is paramount in both the successful evaluation and the treatment of somatic dysfunction. Specific distinctions of myofascial dysfunction are identified most commonly via digital compression, shear, and specifically appropriated tension on soft tissues. Firm yet gentle pressure should be directed with a specific tissue depth in mind and with a three-dimensional perspective.

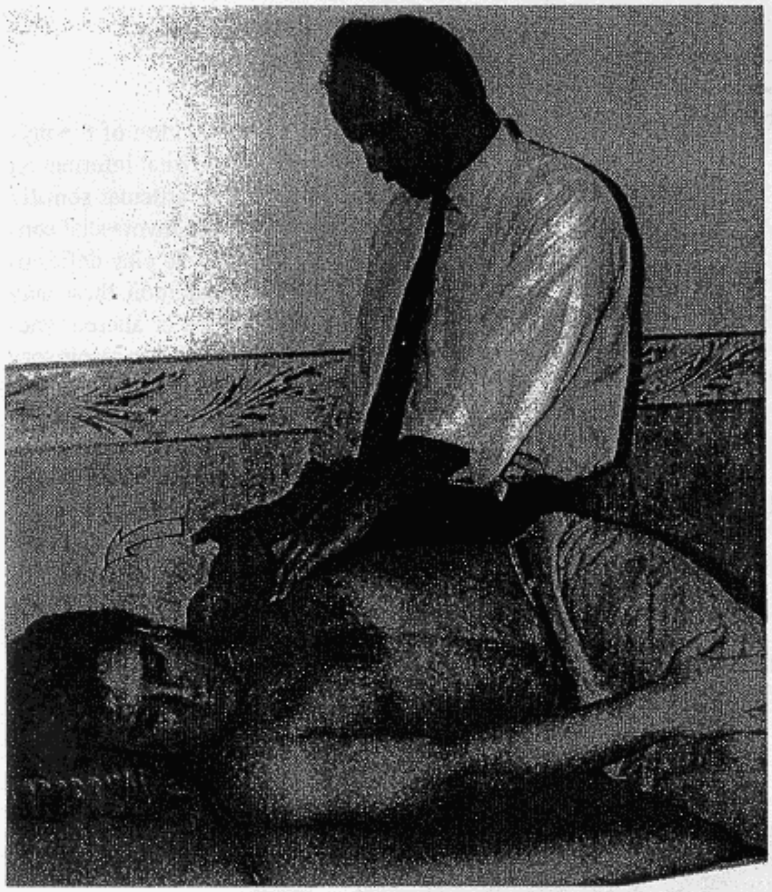


Figure 10.9. Functional movement pattern—arm circles. Performed in a right sidelying position with the inferior arm supporting the superior knee while the superior arm and shoulder are evaluated in a 360-degree circular motion. Myofascial structures are palpated during the entire pattern.

Subtle alterations in tone, excursion, end-feel, fibrous content, contours, reactivity, elasticity, recoil, and response to active contraction and passive lengthening should be assessed. Although emphasis should be placed on objective variables, the patient's subjective response to palpation may also provide insight into biomechanical dysfunction and the relative level of reactivity. Numerous contacts may be used during palpation and include the finger tips, finger pads, thumb, open palm, knuckles, heel of hand, elbow, and forearm. Specifically, the evaluation of myofascial somatic dysfunction of the thoracic spine and rib cage should include careful attention to the components listed in Table 10.15. These elements must be evaluated in both static and dynamic postures (including physiologic, combined movement, and functional movement patterns) and correlated with associated articular dysfunction.

Table 10.15. Elements of Emphasis During Myofascial Examination

Skin mobility
Superficial/deep fascia
Bony contours
Muscle play (accessory mobility)
Muscle tone (hypertonicity/trigger points)
Muscle length/functional excursion
Functional strength/neuromuscular responsiveness

Evaluation of the myofascial tissues should logically begin with the most superficial tissues and progress inward toward deeper layers. The most superficial tissues include the outer epidermis of ectodermal origin and the deeper dermis of mesodermal origin [8]. The superficial epidermis, the dermis, and the superficial fascia are continuous with the deeper fascia and underlying structures via direct attachment to the basement membrane [47]. Arbitrary compartmentalization of the fascia has been pro-

Table 10.16. Fascial Layers

Dermis/epidermis
Superficial fascial layer
Potential space (between superficial and deep fascial layers)
Deep fascial layer
Subserrous fascia (over body cavities)

Source: Adapted from RF Becker. The Meaning of Fascia and Fascial Continuity. New York: Insight Publishing, 1975.

posed based on anatomic disposition and function [84] and is provided in Table 10.16. The recognition of these layers may assist the practitioner in appreciating the various and varying depths of restrictions during both the evaluation and treatment of MFD.

The skin and superficial tissues of the thorax are assessed with a combination of open palmar and digital contacts (Figs. 10.10 and 10.11) for:

1. Soft-tissue contours, symmetry, bulk, draping, and proportions.
2. Skin condition.
3. Skin mobility, excursion, and recoil.
4. Superficial and deep fascia.
5. Scar tissue mobility, extensibility, and adherence to underlying and surrounding structures.

Contact

Soft-Tissue Contours, Symmetry, Bulk, Draping, Proportions

Visual as well as palpatory evaluation of the myofascial structures will often yield vital information regarding the underlying bony or articular somatic dysfunction. Puckered or adherent myofascial contours commonly accompany muscle play deficiencies and tonal abnormalities. In addition, these may correlate with areas of diminished or altered function such as in the case of long-standing respiratory rib cage dysfunction. This is particularly true of the "key rib" (see Chapter 8). Changes in symmetry, bulk, and proportions frequently occur concurrently with tonal or strength deficiencies and may also reveal chronic, compensatory patterns of movement or function. Observation of how myofascial tissue drapes over its underlying osseous and articular structures may also yield information regarding its relative viability and movement potential in addition to the functional capacity of the immediate associated articular structures.

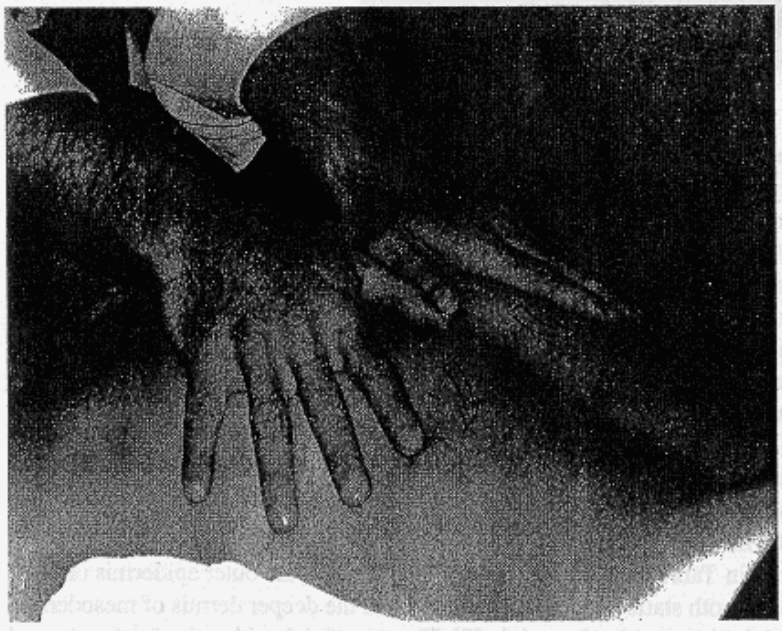


Figure 10.10. Open palmar contact for evaluation of superficial tissues.

Skin Condition

Light palpation of the skin and superficial fascia will reveal much regarding the health and vitality of the tissues being assessed. Tissues should be palpated for their relative dryness, moistness, warmth, coolness, or flaking. Textural abnormalities such as dry, shiny, smooth, or flaking skin will often accompany chronic conditions such as psoriasis, seborrhea, and scleroderma. Elevated skin temperatures with commensurate increases in skin moisture may indicate an active inflammatory process, whereas cool skin temperatures may accompany chronic tissue or articular dysfunction.

Skin blemishes, rashes, moles, and nodules should also be identified. Particular scrutiny should ensue if these alterations are of recent onset, are progressive in nature, or demonstrate adhesions to underlying structures. Subcutaneous nodules or fatty lipomas are common; however, they should be monitored for their role in creating aberrant myofascial kinematics.

Skin Mobility, Excursion, and Recoil

With the patient positioned in the prone and supine positions on an imaginary clock face (i.e., 12 o'clock cranially, 6 o'clock caudally), objective criteria for defining directions of fascial dysfunction are provided. Beginning with a light, open palmar contact, with the hands placed on either side of the vertebral column, tissues are evaluated in a multidirectional fashion with side-to-side comparison. This process is continued in a cranial-to-caudal direction from the cervicothoracic junction to the thoracolumbar junction. Note that complete assessment of the thorax should always include inspection of the craniovertebral and upper/lower quarter regions as well; however, this will not be elaborated on in this chapter.

Tissue excursion, end-feel, and recoil of myofascial tissues after digital deformation should be evaluated. Firm, hard, arresting end-feels with diminished recoil often accompany collagenous restrictions with associated alteration in nonfibrous elements (i.e., glycosaminoglycan depletion, dehydration, and resulting thixotropy). These tissue characteristics are consistent with those categorized as contracture and/or cohesion-congestion (see Table 10.3).

A variety of techniques are used to assess changes in the dermis, epidermis, and superficial tissues, including general skin sliding/shearing, finger gliding, and specific-point skin sliding [29] (Figs. 10.12, 10.13, and 10.14). These three techniques allow the examiner to:

1. Identify a "general region" of fascial dysfunction (i.e., left upper posterior quadrant versus right) with general skin sliding/shearing.

Figure 10.11. Digital contact for evaluation of superficial tissues.

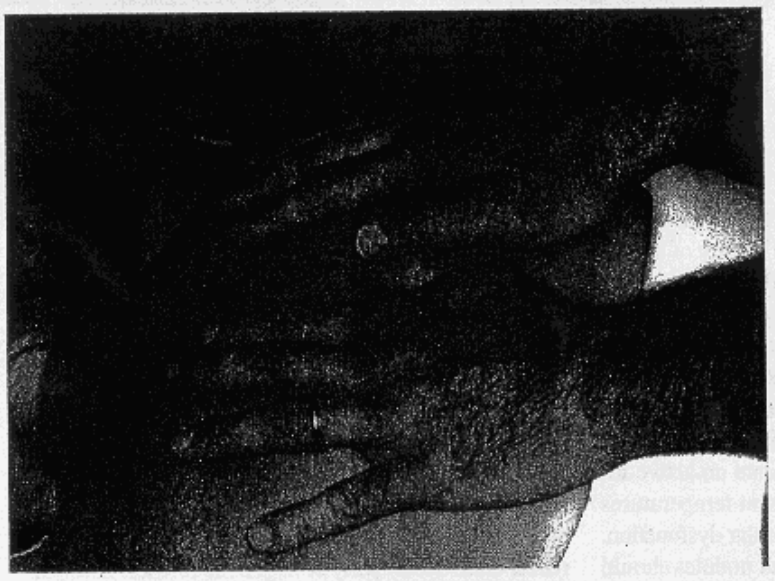


Figure 10.12. General skin slide. Used to localize a "general region," or quadrant, of myofascial dysfunction.

Figure 10.13. Finger glide. Used to localize a "specific site," or epicenter, of myofascial dysfunction.

2. Identify a specific spot or "epicenter" of the restriction within that region with finger gliding.
3. Identify a specific direction of fascial restriction within the restrictive barrier with specific skin sliding.

Superficial and Deep Fascia

Distinguishing between superficial and deep fascial restrictions is vital in establishing and directing appropriate treatment. This is accomplished through a combination of palpatory finesse, angle of inclination of the palpating contact, and varying pressures. Palpatory experience and the skill and tacit information derived through repetition cannot be replaced with even the most eloquent of technical explanations. Determining the existence of myofascial barriers, their direction of restriction, and their exact location in the fascial planes requires much practice. The identification of "depth" is achieved via the angle of inclination the palpating digit or contact as-

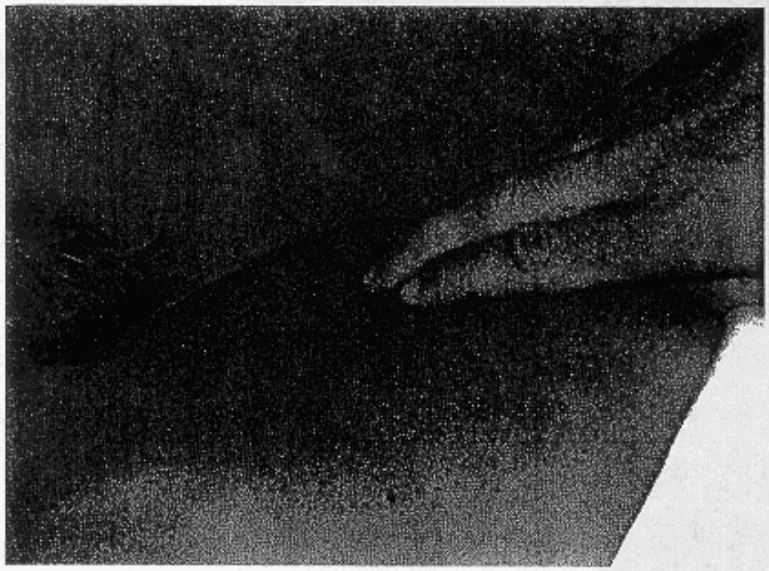


Figure 10.14. Specific skin slide. Used to localize and determine the specific depth and direction of a myofascial dysfunction.

sumes. The more horizontal the contact, the more superficial the tissue being palpated. Shearing tissues in this fashion most often identifies restrictions in the dermis and epidermis. Changing the angle of inclination to a more vertical orientation will allow for greater depth (Figs. 10.15 and 10.16).

In addition, gradually increasing the force used in palpation will provide greater depth. This option, however, should be used last, especially by a novice or inexperienced practitioner. Perceiving that additional force will provide greater proprioceptive feedback is one of the most frequent mistakes in manual medicine. Firm yet gentle compression/palpation will in fact yield the most information and give the least extraneous feedback. Force is, however, an option for depth, and is used to reach deeper fascial structures such as those existing between the septae of the muscle bellies.

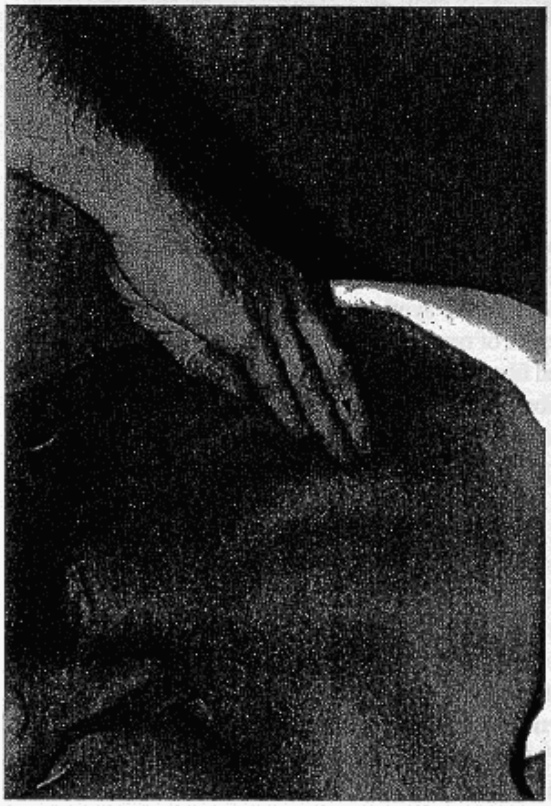


Figure 10.15. Vertical angle of inclination of the mobilizing hand to achieve greater tissue depth and treat deeper structures.

Scar Tissue

Scar tissue formation results from major or minor trauma to tissues with similar histologic consequences, albeit differing in severity. Macrotrauma to myofascial tissues may include surgical incisions, traumatic lacerations, and punctures as well as intrinsic muscle and fascial tears. Microtrauma includes repetitive myofascial strain patterns and habituated postures with aberrant function. In both cases an inflammatory process or

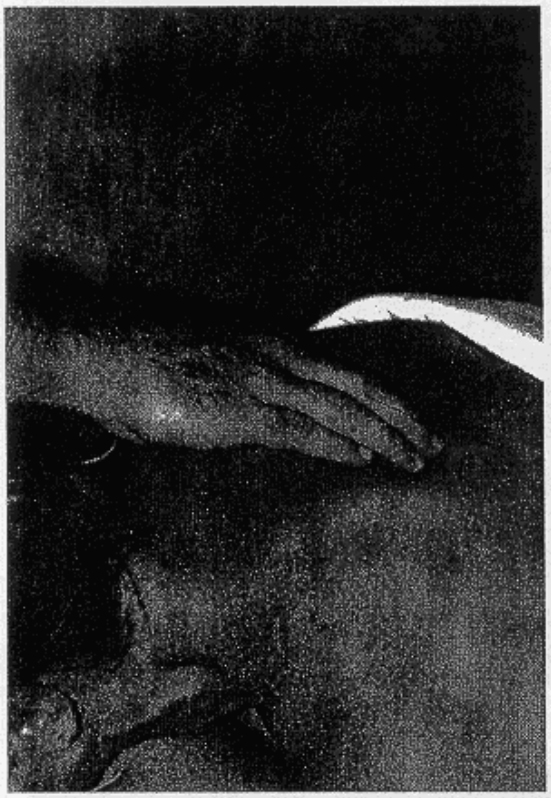


Figure 10.16. Horizontal angle of inclination of the mobilizing hand to achieve access to more superficial tissues.

phase, usually lasting 1–6 days, is followed by a postinflammatory, fibroblastic phase lasting 6–21 days [5, 63, 85]. It is during this fibroblastic phase that a proliferation of newly synthesized collagen fibers occurs with a degree of randomness. "Crosslinking" of normal collagen fibers may occur, dramatically reducing the normal "fiber glide" and therefore the mobility of the implicated tissues. Hollingshead [50] remarked that aberrance of myofascial mobility secondary to scar tissue formation ". . . may be a major factor in altering the biomechanics of the whole kinetic chain, placing strain on all related structures." This strain or altered biomechanics can have profound effects on the subtle arthrokinematics of the rib cage and thoracic vertebra and may be implicated in the primary motion restriction present in respiratory/structural lesions and type II non-neutral and type I neutral vertebral lesions. In addition, abnormal patterns of stress caused by adherent and inextensible scar tissue may contribute to chronic inflammatory disorders and perpetuate symptomatology [62–64]. Remodeling of these newly synthesized fibers in an organized, mobile framework is critical to regaining extensibility and dynamic function at the associated motion segments as well as the entire kinetic chain.

Scar tissue is assessed by appreciating and observing:

1. Stage of healing/reactivity.
2. Intrinsic mobility of the scar (in all planes).
3. Dissociation from adjacent and underlying structures.
4. Influence of scar tissue on osteokinematic motion patterns of related articular segments.

As with skin mobility, assessment is accomplished by shearing tissues in multiplanar directions to determine barriers to movement, directions of those barriers, and quality of end-feel.

Assessment of Bony Contours

The assessment of myofascial tissues as they insert and anchor into the periosteum of the spine and extremities provides information regarding the deepest myofascial structures. The significance of these structures has been noted, since a "great deal of spinal pain may well be felt where muscle, tendon, ligament and capsule are attached to sensitive periosteum of the spine" [86].

Bony contours are evaluated via digital palpation, which proceeds along the osseous structure in a parallel or longitudinal manner. Restrictive barriers, increased tone, and adhesions between adjoining structures are noted as attention is given to the depth and direction of the tissue barrier evaluated. As with superficial tissues, increased depth is accomplished by increasing the angle of vertical inclination of the palpating digit or by increasing force. Table 10.17 identifies the key bony contours that should be evaluated in the thoracic spine and rib cage.

The assessment of bony contours also provides vital information regarding the positional dysfunction of those osseous structures. Aberrance of myofascial tissue and the related position of associated osseous structures should be correlated with motion testing (see CHARTS Method of Evaluation). This is particularly applicable in the rib cage, where structural rib dysfunction will most commonly appear with positional alterations as well as accompa-

Table 10.17. Assessment of Bony Contours

I. Supine
  A. Sternum
    1. Manubrium
    2. Body
    3. Xiphoid
    4. Sternoclavicular joints
    5. Sternorib joints
    6. Costochondral joints
    7. Anterior ribs 1–12
  B. Clavicle
    1. Superior, inferior, anterior
II. Sidelying
  A. Scapula/lateral aspect
  B. Humerus
  C. Lateral ribs
  D. Iliac crest
III. Prone
  A. Vertebral column
    1. Spinous process
    2. Posterior arch
    3. Transverse process
  B. Posterior ribs
  C. Scapula/posteromedial aspect

nying myofascial dysfunction or tissue texture abnormalities. Bony contour, myofascial abnormalities in this region commonly include the intercostal muscles circumferentially, the thoracic fibers of the iliocostalis lumborum muscle posteriorly (especially at its insertion into the rib angle) [53], and the soft-tissue attachments at the posterior costotransverse and the anterior sternocostal articulations of a dysfunctional rib or ribs (Fig. 10.17).

Laterally, the attachment of the latissimus dorsi and serratus anterior muscles become common sites of bony contour abnormalities, especially in the presence of respiratory exhalation dysfunctions. This is particularly common on the superior surface of the key rib (see Chapter 8) or with chronic laterally elevated lesions. Anteriorly, the sternum, sternal manubrial, and costochondral junctions should be carefully assessed (Fig. 10.18).

Other common areas of bony contour abnormalities include the inferior border of the clavicle (with structural or respiratory dysfunction of the first rib), the inferior border of the costochondral arch (with

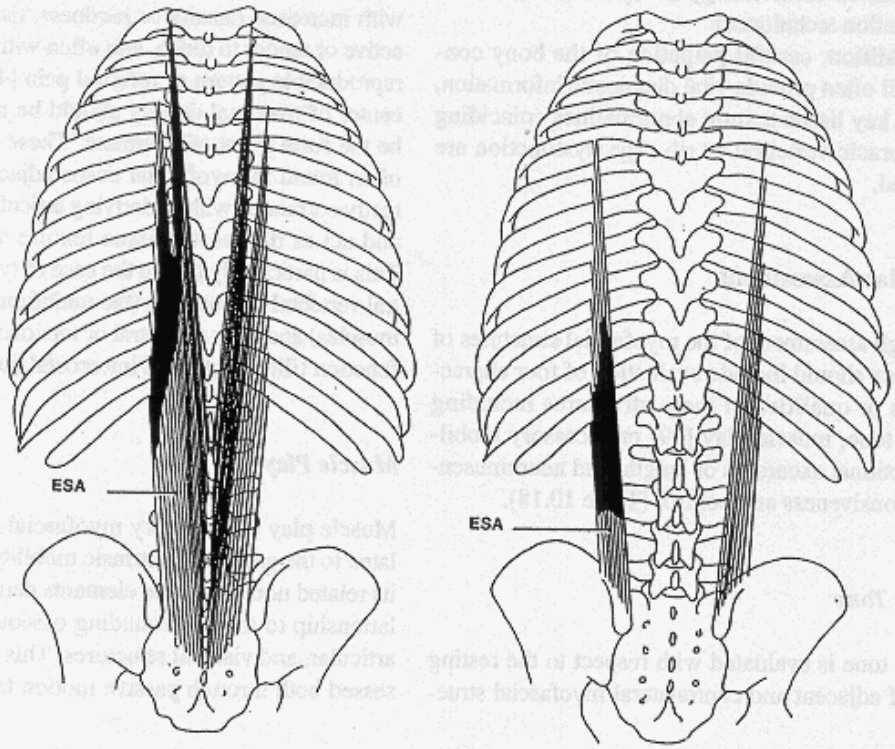


Figure 10.17. Attachment of the iliocostalis lumborum muscles to the rib angle. (ESA = erector spinae aponeurosis.) (Reprinted with permission from N Bogduk, LT Twomey. Clinical Anatomy of the Lumbar Spine. New York: Churchill Livingstone, 1987.)

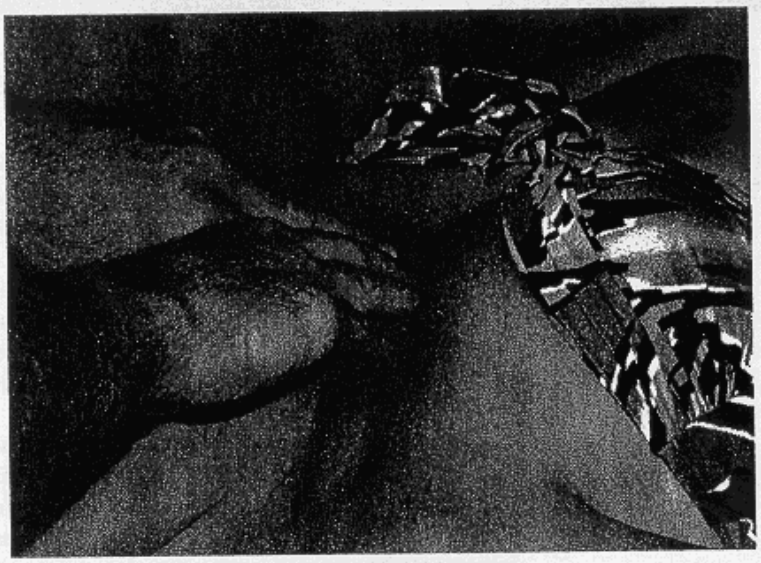


Figure 10.18. Bony contour assessment of the sternomanubrial junction.

respiratory inhalation dysfunction), and the existing groove formed between the spinous and transverse processes (i.e., the posterior arch/lamina of the thoracic spine) in the presence of type II, non-neutral vertebral dysfunction. Left untreated, this myofascial dysfunction may contribute to nonresponsive rib cage and/or vertebral dysfunction, especially when treated with an approach relying predominantly on an articulation-driven methodology (i.e., joint mobilization, manipulation techniques).

In addition, careful palpation of the bony contours will often provide vital diagnostic information, because key tissue texture abnormalities coinciding with thoracic vertebral or rib cage dysfunction are identified.

Muscular Assessment

Thorough assessment of the myofascial structures of the thorax should include evaluation of four characteristics or qualities of these structures including muscle tone, muscle play [29] or accessory mobility, functional excursion or length, and neuromuscular responsiveness and control (Table 10.18).

Table 10.18. Muscle Assessment

Muscle tone
Muscle play/accessory mobility
Muscle length/functional excursion
Neuromuscular responsiveness and control

Muscle Tone

Muscle tone is evaluated with respect to the resting tonus of adjacent and contralateral myofascial structures. Aberrance of muscle tone typically presents with increased density or hardness, tissue that is reactive or tender to touch, and often with a typical and reproducible pattern of referred pain [4, 14]. An epicenter of maximal density should be noted and will be the focal point of treatment. These epicenters are often found in myofascial tissue adjacent to and directly correlated with underlying articular dysfunction and act as diagnostic tissue texture abnormalities. This is particularly true in the case of type II non-neutral vertebral dysfunction (the multifidus and rotatores muscles) and with structural or torsional rib cage dysfunction (iliocostalis and intercostal muscles).

Muscle Play

Muscle play or accessory myofascial mobility is related to the amount of intrinsic mobility a muscle and its related noncontractile elements demonstrate in relationship to their surrounding osseous, myofascial, articular, and visceral structures. This mobility is assessed both through passive motion testing and dur-

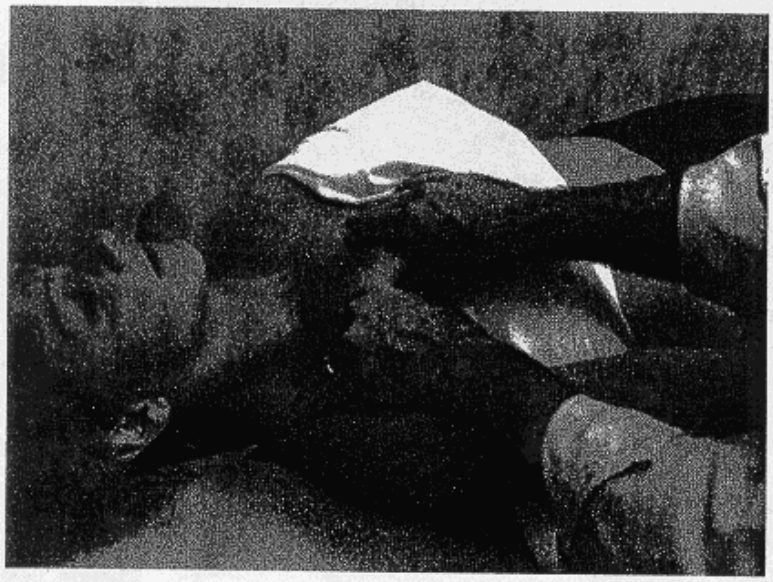


Figure 10.19. Perpendicular deformation of the pectoralis minor muscle with bilateral thumb contact to evaluate medial-lateral, lateral-medial muscle play.

ing functional movement patterns of excursion or lengthening, as well as during the shortening and broadening of fibers associated with muscle contraction. Passive motion testing is accomplished through perpendicularly directed forces (thumbs or tips of digits) described as a perpendicular/transverse deformation, and through "strumming techniques," which assess mobility in two planes [29] (Fig. 10.19).

The ability to shear muscles freely from their adjacent and underlying structures, as well as the ability to move in an uninterrupted fashion through the septae of muscle groups, is evaluated with these techniques. Specific sites, depths, directions, and degrees of restrictions should also be noted. In addition, related thoracic and costal segments and their mobility should be considered, because muscle play restrictions frequently accompany and precipitate somatic dysfunction in these regions. This is commonly seen in the rib cage between the pectoralis major, the pectoralis minor, and underlying costal segments 3, 4, and 5. Aberrant muscle play between the pectoralis major and minor muscles, or between the deeper pectoralis minor muscle and the rib cage, creates abnormal tension in the costal segments and commonly provides an environment for respiratory rib cage dysfunction. Other specific key muscle groups of the thorax that require careful assessment for muscle play abnormalities are provided in Table 10.19.

Table 10.19. Muscle of Emphasis in Muscle Play Assessment of the Thorax: Muscle Play/Accessory Mobility

Spinalis/longissimus/iliocostalis
Pectoralis major/minor complex
Rotator cuff muscles
Serratus anterior/posterior (superior/inferior)
Latissimus dorsi
Trapezius/levator scapulae
Intercostals
Respiratory diaphragm
Quadratus lumborum
Abdominals
Psoas major/minor

Functional Excursion

Functional excursion is the ability of an individual muscle to lengthen as it concomitantly narrows, as well as its ability to broaden as it simultaneously shortens. The importance of balance and symmetry with respect to excursion and length of agonist and antagonist muscle groups has been clearly identified in patients suffering from low back pain [87–89]. Tightness or diminished functional excursion in paired muscle groups (i.e., bilateral hip flexors, hamstrings, paraspinal muscle groups) are viewed as potentially contributory to aberrant static posture and

Table 10.20. Functional Excursion Axiom

With *symmetrical* muscle tightness *be aware...*
With *asymmetrical* muscle tightness *beware.*

warrant investigation. These deficiencies may result in an accentuation of sagittal plane, primary and secondary curves (i.e., cervical/lumbar lordosis, thoracic kyphosis), and the development of postural dysfunctions such as forward head posture (FHP), cervical and thoracic dowager's deformity, and thoraco-lumbar kypholordosis. Asymmetric tightness or loss of functional excursion in paired muscle groups, however, demands the utmost of evaluative scrutiny. This is chiefly because asymmetric muscle length in paired groups may create alteration in the coronal plane with rotational or torsional forces imposed on the pelvis, vertebral column, and rib cage. These forces may result in the presence of non-neutral mechanics in the vertebral column and the increased risk of myofascial and articular dysfunction. An axiom to follow when evaluating functional excursion is presented in Table 10.20.

Muscles are evaluated by taking their associated sites of origin and insertion through patterns of movement that functionally lengthen the appropriate tissues. As all osteokinematic movements in the extremities occur in curvilinear or elliptical arcs, movements should be directed with care and consideration for these pathways. Osteokinematic motions should include straight planar (i.e., sagittal, coronal) patterns of movement as well as combined patterns to create functional, diagonal patterns. In addition, movements should be performed with careful observation given to barriers of increased resistance within an overall range of excursion. Total range may belie minute yet significant restrictions in the myofascial unit and only be identified through visual and palpatory means during actual functional excursion.

Functional excursion should also be assessed through active and resisted movements where possible. Palpation of relative muscle bellies and musculotendinous and tenoperiosteal junctions should be performed throughout movements of functional excursion to identify specific points or sites of maximal restriction. These sites will frequently be the starting place for treatment (Fig. 10.20).

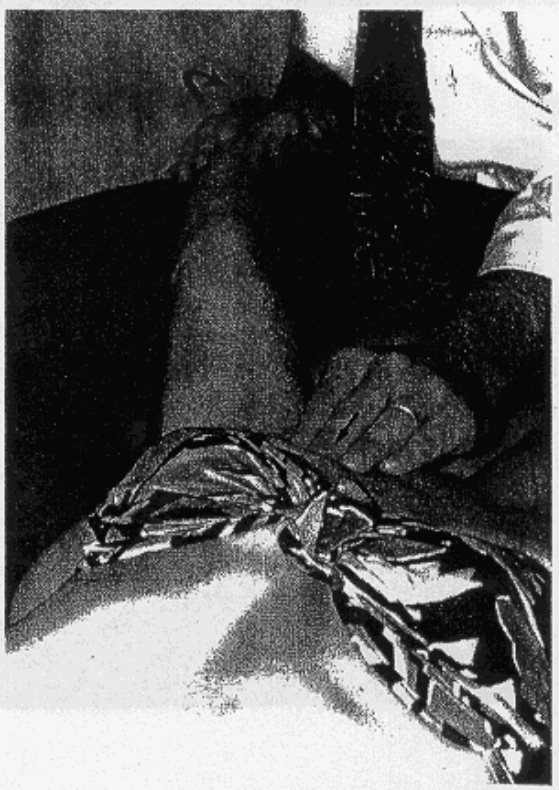


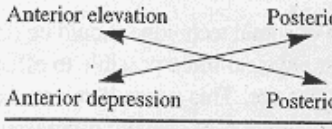
Figure 10.20. Evaluation of functional excursion with concurrent palpation of the pectoralis major muscle to identify specific dysfunctional tissue barriers.

Neuromuscular Responsiveness and Control

Neuromuscular responsiveness consists of the initiation, control, recruitment, strength, timing, and endurance, among other qualities, of a myofascial tissue [82]. These components are critical in achieving proper function of the related articular structures and kinetic chain as a whole. This is particularly true in the case where the chronicity of somatic dysfunction has significantly affected the neuromuscular attributes of the myofascial tissues associated with articular dysfunction.

Assessment of the neuromuscular responsiveness and control of a myofascial tissue can be accomplished through the use of proprioceptive neuromuscular facilitation (PNF) patterns [38, 39, 82]. In the thoracic spine and rib cage, shoulder and pelvic girdle patterns (Table 10.21) are a valuable tool for both evaluation and treatment strategies. Guidelines for effective monitoring and administration of these patterns are also provided (Table 10.22).

Table 10.21. PNF Patterns of Assessment for Neuromuscular Responsiveness and Control



Source: Adapted from G Johnson, V Saliva. PNF-I: The Functional Approach to Movement Reeducation. San Anselmo, CA: The Institute of Physical Art, 1987.

Table 10.22. Guidelines for Effective Administration and Monitoring of PNF Patterns

Provide complete tension at lengthened ROM without strain to cervical or thoracic components.
Allow minimal trunk motion during pattern.
Motion should remain in a straight diagonal with an arc component.
Motion proceeds from a superior to inferior, anterior to posterior direction, or vice versa.
The head of the humerus should cross the midline.

Source: Adapted from G Johnson, V Saliva. PNF-I: The Functional Approach to Movement Reeducation. San Anselmo, CA: The Institute of Physical Art, 1987.

Principles of the Treatment Approach

Management of somatic dysfunction of the thoracic spine and rib cage requires a multifaceted, eclectic approach with careful attention paid to the objective findings enumerated in the evaluative process. In addition, several guidelines for treatment may be helpful in directing both the novice and experienced practitioner. These guidelines are provided in Table 10.23.

Be Guided by Objective Findings

Treatment must always be guided predominantly by objective findings rather than subjective presentations. Pain in the thorax is most ubiquitous and confusing, because virtually all somatic structures possess at least three spinal levels of innervation [53, 90]. In light of this, applying treatment with a palliative methodology based on subjective direction by the patient is most futile. In addition, differentiation of the various pain presentations (i.e., superficial spondylogenic, deep spondylogenic, neurogenic, radicular, viscerogenic, psychogenic), although possible and important, will provide little in the way of treatment direction. Instead,

Table 10.23. Treatment Guidelines

Be guided by objective findings rather than subjective complaints.
Treat motion dysfunction versus positional dysfunction.
Sequence treatment in order of maximal motion loss according to dysfunctional tissue end-feel or barrier.
Never barge a barrier; however, once "one has a foot in the door, never lose it."
Use the least force possible to effect the desired change.
Clear compensatory somatic dysfunction following primary dysfunctions to avoid recidivism.
Reevaluate following each procedure ("onion skin concept").

Source: JJ Ellis. LPI—Lumbo-Pelvic Integration. A Course Manual. Patchogue, NY, 1990.

the manual practitioner should trust in the observation and identification of specific and reproducible objective findings (i.e., positional and motion).

Treat Motion Dysfunction Versus Positional Dysfunction

Because function follows form and form is determined by function, it is imperative that treatment be guided toward the restoration of appropriate motion and not be guided by apparent positional findings in and of themselves. Positional findings frequently present as the end-result of aberrant myofascial function. In addition, the presentation of aberrant positional findings in the absence of motion loss may belie bony anomalies with relatively normal arthrokinematics.

Sequence Treatment in Order of Maximal Motion Loss

Although numerous axioms and strategies for treatment progression exist in the osteopathic literature [1, 35, 36], perhaps the most significant driving principle is the administration of treatment through a sequence that identifies the areas of maximal motion loss in accordance with dysfunctional tissue end-feels. This may include myofascial, articular, and, at times, visceral structures. This is particularly true in the thorax, where the rib cage provides attachment for numerous myofascial and visceral structures that often provide the primary barrier in motion restriction. Departure from a traditional progression is often warranted in this area

because the costal segments and their associated myofascial structures, particularly where chronicity prevails, provide an environment for aberrant function of the thoracic vertebral segments. An example of this is the case where both a type II non-neutral ERS dysfunction and an external rib torsional dysfunction exist concomitantly. In accordance with the traditional biomechanical model, the thoracic vertebral dysfunction would be addressed first because this is seen as primary and the cause of the associated external torsion dysfunction [1, 35, 36]. This, however, may not be the most judicious approach, especially where chronicity prevails. In this example, the costal segment's chronic aberrant position is frequently associated with significant myofascial dysfunction of the related intercostal muscle groups and related connective tissue. Attempting to address the thoracic vertebral segment first may not only prove unsuccessful, but may with repeated attempts create iatrogenically induced instability in the costovertebral and costotransverse articulations. The costal segment, acting like an anchor (secondary to MFD), provides an environment where correction of the thoracic vertebral dysfunction is unattainable until an environment (of the myofascial tissues) is provided in which correction can occur and be maintained. Departure from previously learned sequences that emphasize articular technique is not only prudent in this case, but necessary if success is desired. However, a combined articular technique that addresses the costovertebral and costotransverse articulations is often effective (see Chapter 9).

Never Barge a Barrier

The application of manual therapy procedures, particularly in the myofascial tissues, demands the utmost of sensitivity and respect for patient tolerance. Aside from recognition of a tissue's reactivity and its relative stage of repair (i.e., inflammatory, fibroblastic, or remodeling stage), the patient's tolerance of treatment depth and progression must be considered. This is particularly true in the presence of increased muscle holding or tonicity. Progression of treatment must be accomplished with acute awareness of the patient's response to treatment. As the attainment of increased depth is attempted through subtle yet progressive tissue deformation, the patient's response should be carefully monitored, both verbally and somatically. Progression or depth should never substitute for patient tolerance and comfort. Once additional depth or "ground" is achieved, however, all attempts should be made to maintain that ground, keeping in mind the adage, "never pay for the same real estate twice."

Least Force Possible

The implementation of actual technique should be delivered with the least physical force possible to effect the desired somatic change. This necessitates an exacting diagnosis, enumeration of specific myofascial and articular findings, the identification of specific barriers (i.e., depth, degree, direction), and the precise localization of those barriers. Treatment should be directed at these specific barriers, with increased force being reserved as a last option. The angulation of hand contacts, use of an assisting hand, position of the body part being treated (i.e., shortened or lengthened position), and the use of associated oscillatory motions are alternative strategies that should be considered before increased force is used. Communication between the patient and practitioner will prove valuable as the patient is encouraged to assist in both the identification of the barriers and in the process of mobilizing soft tissues. In addition, patients are encouraged to report subjective changes experienced during the treatment process. This will aid them in localizing and treating these same restrictions in their own home program.

Clear Compensatory Somatic Dysfunction

Effective management of somatic dysfunction of the thoracic spine and rib cage requires thorough assessment of and amelioration of compensatory dysfunctions that exist concomitantly with the primary dysfunction. According to the biomechanical model methodology, this compensatory dysfunction may respond and correct itself concurrently with the normalization of the primary dysfunction. However, where chronicity prevails, such compensatory dysfunctions (particularly myofascial) may require specific attention. In addition, compensatory dysfunction, especially where it is associated with myofascial dysfunction, may create an environment where normalization of primary dysfunction will not occur, or where there exists a predilection for a recurrence of that dysfunction. This is seen in a patient with a chronic type II non-neutral FRS right dys-

function of T5 with a compensatory neutral type I rotoscoliosis above at T1–T4 (side-bent to the left, rotated to the right), cervical spine side-bending to the right, and subcranial sidebending back to the left. With attention focused on correction of the type II FRS dysfunction and then the thoracic type I dysfunction, the cervical and subcranial dysfunctions often are left untreated. Myofascial dysfunction of this region may provide an environment of altered posturing in the coronal plane and for non-neutral mechanics to dictate in the upper thoracic region. Although type I neutral dysfunctions are not commonly recognized as the primary dysfunction, in this case, they may provide the biomechanical prerequisites for recurrence of the type II dysfunction.

Re-Evaluate After Each Procedure

An axiom for all manual therapy procedures includes re-evaluating after each corrective procedure. Because this therapeutic intervention is "corrective versus palliative" in definition and intent, objective variables defined in the evaluative process must be continually reassessed. The normalization of myofascial dysfunction associated with vertebral and rib dysfunction frequently has profound effects on the overall presentation of that somatic dysfunction. Evaluation should focus on the positional and motion characteristics of the region being treated and include palpation of related bony landmarks and myofascial structures and the administration of one or two relative motion tests. Although re-evaluation should include the monitoring of subjective complaints, an emphasis should not be placed on this in the re-evaluation.

Principles of Myofascial Techniques

The successful administration of soft-tissue mobilization (STM) requires a keen sense of both patient and tissue response. This response is monitored through a variety of senses, including sight, palpation, and auditory feedback. Constant reassessment and adjustment of contacts, including localization, duration, and force, as well as patient positioning and assistance with the treatment technique are required for a successful outcome. In addition, a variety of soft-tissue mobilization techniques and strategies by which to implement them contribute to the success in this endeavor.

Contacts

Numerous contacts may be used during STM and commonly include the tips of the digits, the thumb, the pisiform, the open palm, the forearm, and the elbow. Determination of which contact is used depends largely on practitioner comfort, the body part being worked on, and the dexterity and skill of the practitioner administering treatment.

Depth

Localization of the treatment contact to the dysfunctional tissue barrier is imperative for success in myofascial mobilization. Barriers must be engaged with respect to tissue depth and direction of the restriction, with localization maintained throughout the technique. Depth is obtained through the judicious use of force, contact angulation (vertical versus horizontal), and the positioning of associated myofascial tissues in a shortened position. Angulation of the contact will determine the depth of a technique and should always be used before increasing force. As with the evaluative process, the more vertical the orientation of a contact, the greater the depth achieved. Positioning adjacent soft tissues in a shortened position decreases tissue turgor and tension and will allow greater penetration. Identification and maintenance of appropriate tissue depth often proves most challenging for practitioners new to STM; however, they are critical for success. Relocalization to the new barrier must occur throughout each specific technique.

Duration

In accordance with the viscoelastic properties of connective tissue, there is a specific time dependency for the elongation of collagen and elastin fibers [48, 52]. This time may vary depending on the viability and conditions of the soft tissues being treated. Various clinicians have attempted to outline specific time frames for treating a specific myofascial restriction. These range from 10 seconds to 90

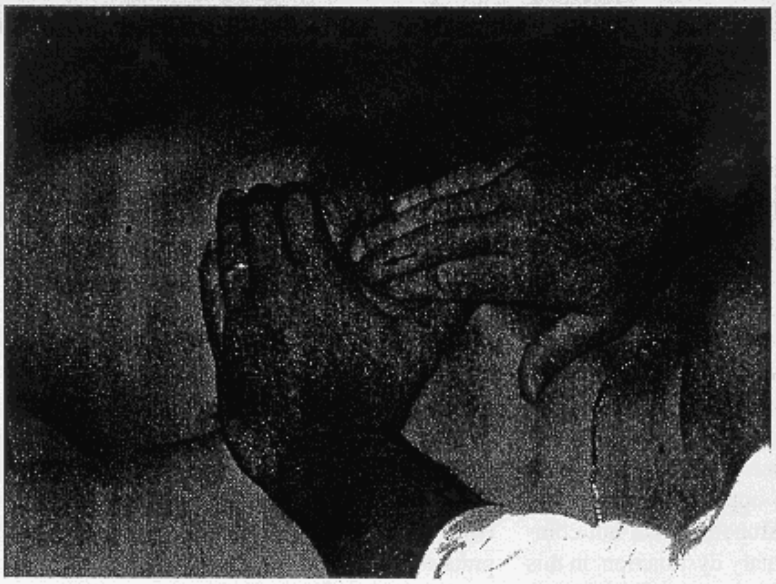


Figure 10.21. Soft-tissue mobilization of the superficial clavipectoral fascia in a shortened position.

seconds [29, 30, 79, 91]. Perhaps a more functional approach, and the one we endorse, is to apply treatment while concurrently evaluating and assessing the response of the treated and surrounding soft tissues. Instead of dogmatically assigning a time constraint on treatment, duration is based more dynamically on ongoing tissue response. Changes in tissue density (i.e., softening, elongation), turgor, tone, and elasticity (particularly with respect to end-feel) are noted as significant and warrant continued efforts. If after two to three attempts with a particular technique no noticeable change is perceived, consideration should be given to other sources as being primary or causative.

Force of Technique

As with most manual therapy procedures, force is never a substitute for accuracy or specificity of localization or for an appreciation of ductile (time dependant) properties of myofascial tissue. In STM, forces used should be only those that allow for dysfunctional tissue barriers to be engaged and corrected in accordance with their time-dependent nature of motion. Force should also be sufficient enough to maintain engagement of a barrier throughout the technique. As elongation or softening of a dysfunctional tissue barrier is perceived, the path of release should be followed.

Patient Positioning

Patient positioning should incorporate positioning the involved tissues and respective body parts in one of three static positions—resting neutral, shortened range, or lengthened range—and through various dynamic postures.

Static Postures

In the resting, prone-neutral position, the lumbopelvic girdle is in a soft lordotic position with a pillow or towel under the abdomen. The shoulders are supported to eliminate protraction, while the forehead (i.e., the table face cutout is used) and the ankles are supported. The emphasis is on attaining an environment of comfort, while reducing the cervicothoracic and thoracolumbar curves.

In the shortened position, respective tissues are placed in a slackened range by altering the surrounding tissue (at the same level) or through the positioning of a related bony or articular segment. This strategy provides an environment that dampens the feedback from surrounding myofascial structures and often allows the most efficacious palpation of the dysfunctional tissue barrier (Fig. 10.21).

Treatment with tissues in the shortened range is followed by progressing to the resting neutral position and finally into the lengthened range. In the

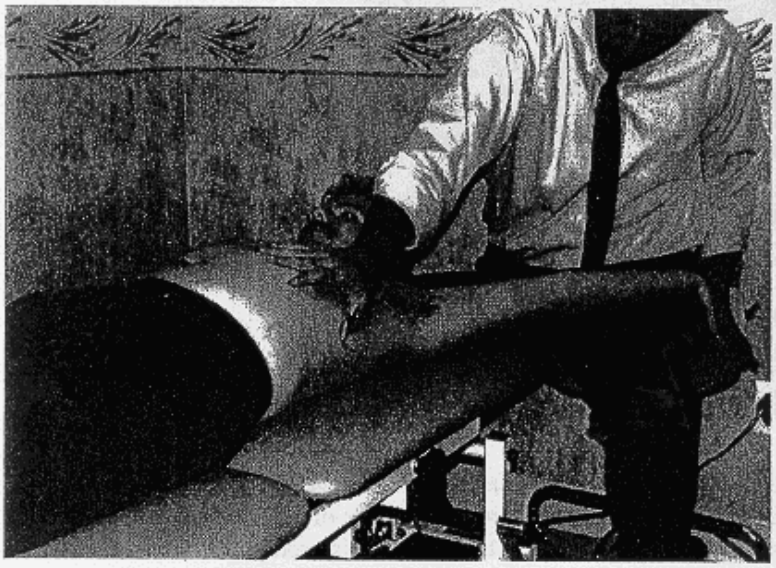


Figure 10.22. Parallel soft-tissue mobilization technique to the latissimus dorsi muscle group in a lengthened range.

lengthened range, respective tissues are placed in a position of increased tension or length by altering the surrounding tissues (at the same level) or a related bony or articular segment (Fig. 10.22).

This strategy attempts to maximize and concentrate surrounding tissue tension forces to the dysfunctional tissue barrier. Treatment progression most often concludes with the lengthened range and should be used to ascertain complete resolution of the myofascial dysfunction. These postures may be assumed through the use of such props as pillows, wedges, physio or Swiss balls, and adjustable, sectioned tables.

Dynamic Postures

Treatment of any myofascial tissue should include the use of both static and dynamic postures. As with assessment, treatment may be accomplished during dynamic movement patterns of the trunk or related extremity and should include engagement of the dysfunction tissue barrier, maintenance of localization and mobilization force throughout the movement, and a progressive increase in the tolerable amplitude of movement. These movements may include classic osteokinematic motions, or they may incorporate passive, associated oscillations of the trunk or body part. These oscillations, produced through a rhythmic rocking, most commonly at the pelvis, shoulder, or rib cage, may be inhibitive, if slow and rhythmic, or facilitory, if fast and erratic.

Patient Assistance

When appropriate, patients should be actively involved in the treatment process. This is accomplished in several ways. The patient may be asked to contract or relax an area, provide appropriate resistance (isometric, concentric, eccentric), or to provide assistance through alternative methods such as inhalation/exhalation and coughing efforts. Each of these options provides an opportunity for greater localization of forces and more effective mobilization of dysfunctional tissue barriers. In addition, when effective, patients are encouraged to use similar strategies in their home programs.

Selected Myofascial Mobilization Techniques

During the application of STM, both hands of the practitioner participate, with one designated as the mobilizing or treating hand and the other as the assist hand. This allows for specificity of localization and the option of dynamically lengthening or shortening surrounding tissues, as well as incorporating associative oscillatory forces. A variety of specific mobilization forces are available for use with the

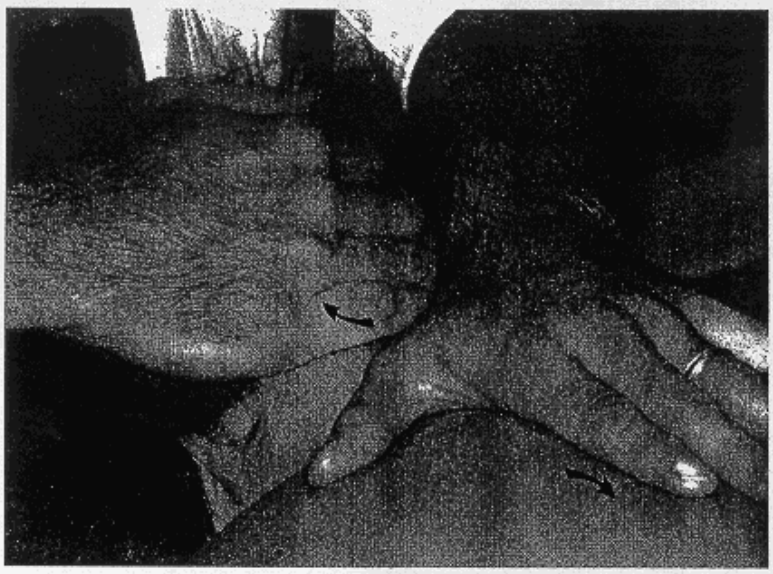


Figure 10.23. General soft-tissue mobilization technique to the superficial fascia of the posterior cervicothoracic junction.

mobilizing or treating hand, while the assisting hand controls the immediate tissue environment. The mobilizing hand is used to engage the dysfunctional tissue barrier through a pushing force (push) or a pulling/hooking force (pull). These mobilization forces are directed at the site of maximal restriction and may be delivered as sustained pressure, a perpendicular mobilization effort, end-range oscillation, a strum, or parallel mobilization technique [29].

General Junctional Release Techniques

Type II non-neutral vertebral dysfunctions and accompanying rib dysfunctions demonstrate a predilection for occurring at junctional areas of the vertebral column (i.e., occipitoatlantal, cervicothoracic, thoracolumbar, and lumbopelvic junctions). Myofascial dysfunction is also frequently encountered at these levels. General junctional release techniques provide a quick screen and clearance approach to the skin and the superficial and deep fascia of these regions before MET or mobilization efforts are used. Treatment consists of contacting the region with a bilateral, open palmar contact paravertebrally or in a cranial-to-caudal orientation, and then engaging the superficial or deep dysfunctional tissue barriers. This is accomplished through gentle compression, shearing tissues into the greatest restrictive barrier, and finally adding clockwise or counterclockwise rotation of the hands. Maximal tissue tension is maintained as both hands continue to shear in the direction of the dysfunctional tissue barrier. This approach is used posteriorly at the cervicothoracic and thoracolumbar junctions, and anteriorly at the clavipectoral tissues and the cervicothoracic/sternomanubrial junction (Figs. 10.23, 10.24, and 10.25).

Sustained Pressure

Sustained pressure involves engaging the dysfunctional tissue barrier (most commonly through the use of the distal tips of the digits) while carefully monitoring depth, direction, and degree of restriction, and maintaining a static force against the restriction until a change in density or length is perceived. This technique is primarily used for muscle tone problems; however, it is effective for myofascial play dysfunction as well. Patient participation is encouraged (especially when treating muscle tone problems) through the use of biofeedback-like techniques including controlled breathing, visual imagery, and active contraction/relaxation techniques. With the epicenter of the dysfunction tissue barrier engaged, the patient is encouraged to gradually inhale, further engaging the barrier, while pressure is maintained. During exhalation, the new barrier is engaged without provoking a localized response of pain or an increase in tone (Fig. 10.26). This is repeated until muscle tone is normalized.

Figure 10.24. General soft-tissue mobilization technique to the superficial fascia of the anterior cervical thoracic/sternomanubrial junctions.

Figure 10.25. General soft-tissue mobilization technique to the superficial fascia of the posterior thoracolumbar junction.

Perpendicular Mobilization

Perpendicular mobilization efforts involve engaging the dysfunction tissue barrier and then, through a series of graded mobilization efforts of varying amplitudes, deforming the respective tissues at right angles. These amplitudes may be graded similarly to those used in the articular system (i.e., grades I–V) [92, 93]. This technique is most frequently used with muscle tissue (midbelly, tenoperiosteal) and at bony contours, and is most effective with diminution of myofascial play, although it can be used in the presence of increased muscle tone (Fig. 10.27).

End-Range Oscillating Mobilization

The last phase of a perpendicular mobilization effort, end-range oscillating mobilization, is administered to myofascial tissue at the end-range of its available accessory motion. Applied in a perpendicular or transverse orientation, mobilization forces in

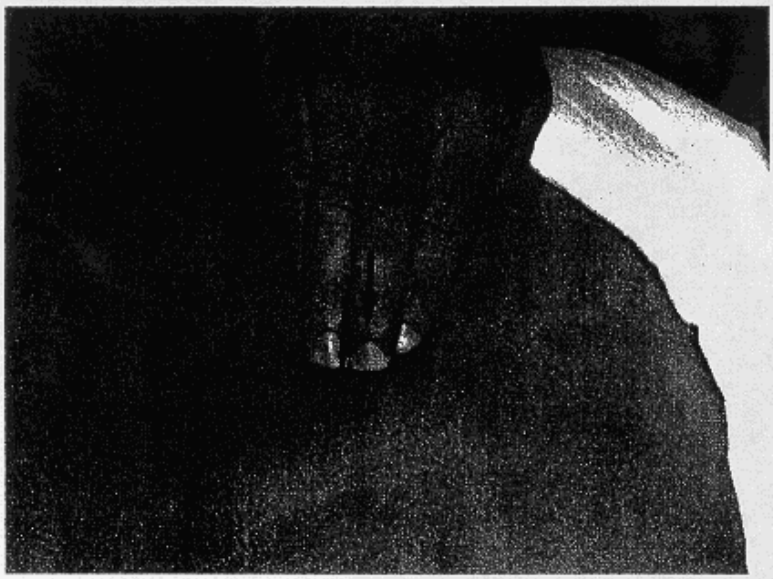


Figure 10.26. Sustained-pressure soft-tissue mobilization technique to the rhomboid muscle group.

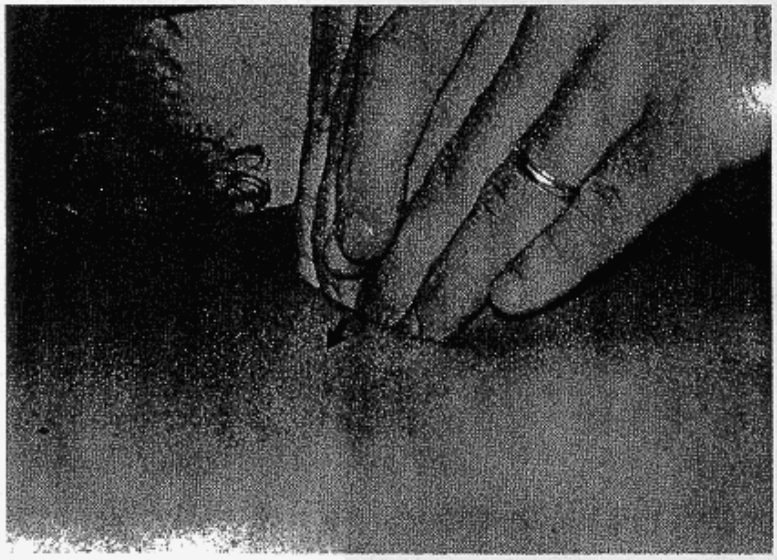


Figure 10.27. Perpendicular soft-tissue mobilization technique.

this technique are of very small amplitudes and create a transverse, end-range deformation to the site and direction of maximal tissue dysfunction. This technique is effective for both myofascial play and tonal abnormalities.

Strumming

Strumming technique incorporates a transverse mobilization effort that begins at the medial or lateral seam of a muscle belly, deforms the belly through a push or pull effort, and then strums across the belly to the opposite side without sliding over the skin. This rhythmic, synchronous movement can be used to treat both myofascial play and muscle tonal aberrations (Fig. 10.28).

Parallel Mobilization

Parallel mobilization techniques are applied between muscle belly septae, at the lateral or medial borders of a muscle, or along a bony contour. As the mobi-

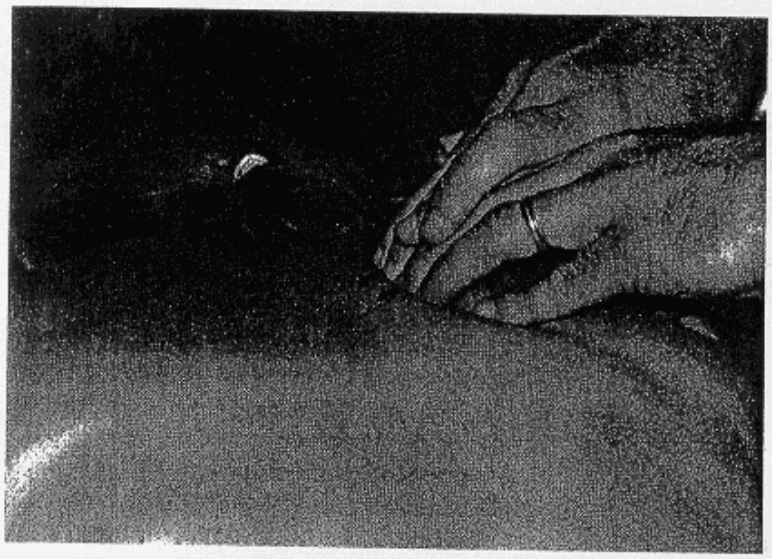


Figure 10.28. Strumming soft-tissue mobilization technique.

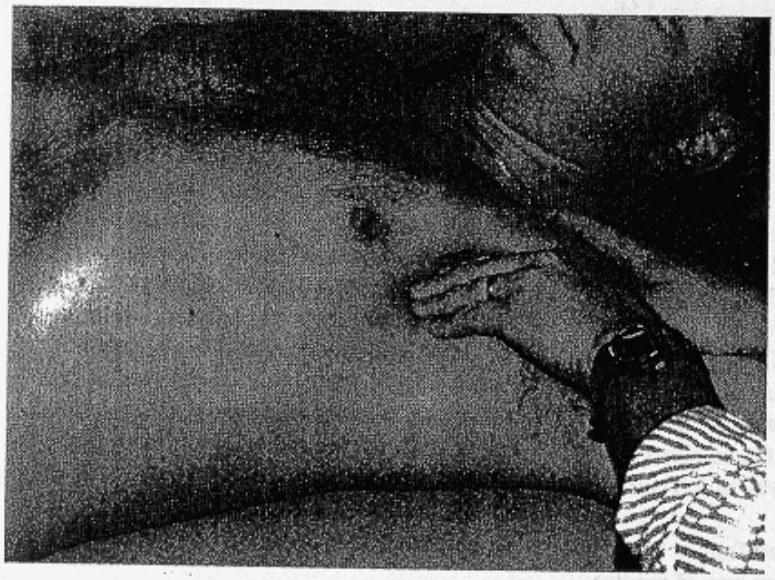


Figure 10.29. Parallel soft-tissue mobilization technique.

lizing hand or digit glides along the contour or between the septum, various angles of inclination are used to attain appropriate depth to localize and ameliorate specific dysfunctional tissue barriers. This technique is effective for both myofascial play and tonal dysfunctions (Fig. 10.29).

Functional Excursion and Muscle Length

Restoring functional excursion or length to the myofascial unit demands careful attention and cognition of that particular structure's fiber type or preponderance, orientation, relative stage of reactivity, and the osseous or articular structures to which it attaches. Myofascial structures may initially have diminished excursion in one direction. However, they may maintain a multiplanar fiber direction that, when taken through full excursion, presents multidirectional limitations. Slow, judicious, and incrementally small ranges of excursion will allow the examiner to identify and treat tissue limitations.

Specific treatment techniques for increasing excursion include static stretching [94, 95], hold-relax,

contract-relax stretching [38, 82], and a combination of isotonic contractions to that particular myofascial structure (concentric, eccentric, isometric, slow reversal hold) [4, 82].

Case Studies

The following case studies illustrate the importance of identifying objective clinical findings of somatic dysfunction of the thorax. Particular attention is placed on myofascial tissues in their causal relationship of biomechanical dysfunction, as well as their potential role in promoting recalcitrant dysfunction.

Case 1

C: Chief Complaint

A 29-year-old woman reported a 6-week history of right upper quadrant and cervical pain; right greater than left episodic paresthesia in the right hand (digits 1 and 2); discomfort with all active cervical motions, most notably left side-bending; and increased symptoms on full inhalation efforts.

H: History

The patient described being injured while playing volleyball. On spiking the ball and following through, she experienced immediate cervical and right shoulder pain, which progressed to the right hand approximately 2 days later. Over a period of 1 week, the pain continued to increase in intensity and was present during examination with all inhalation efforts, with numbness in the right hand reproduced and increased with inhalation as well as left cervical side-bending efforts.

The patient denied any related family history, past mechanical or medical history, use of medications, or prior treatment for this condition. Her occupational history appeared noncontributory in the development of this dysfunction.

A: Asymmetries of Bony Landmarks

The key asymmetric findings for this patient included:

1. Elevated first rib on the right.
2. A type II non-neutral FRS left dysfunction at T1.
3. A type II non-neutral FRS right dysfunction at T2.

R: ROM/Mobility Testing

Positive findings on key motion tests for this patient included:

1. Confirmation of the type II non-neutral FRS left and FRS right dysfunctions at T1 and T2 (with diminished extension, side-bending, and rotation to the right at T1 and diminished extension, side-bending, and rotation to the left at T2).
2. Diminished caudal spring testing to the first rib on the right with firm, arresting, reactive end-feel.
3. Aberrant respiratory motion of the right upper rib cage on full exhalation.
4. Diminished active cervical range of motion, with side-bending left 75%, rotation right 50%.

T: Tissue Texture/Tension/Tonal Abnormalities

The key tissue abnormalities for this patient included:

1. Significant increase in tone of the right anterior and medial scalenes, right sternocleidomastoid, and right levator scapulae musculature.
2. Diminished mediolateral muscle play of both of the right anterior and medial scalenes at their respective musculotendinous and tenoperiosteal junctions.
3. Diminished mediolateral muscle play of the right sternocleidomastoid muscle at the mid-belly, with poor disassociation from the underlying anterior and medial scalene muscles.
4. Decreased functional excursion of the right anterior and medial scalene and right levator scapulae muscles.

S: Special Tests

Special tests for this patient revealed:

1. Positive radiologic evidence of a mildly cranially displaced first rib on the right.
2. Positive adverse neural tension signs in the right upper extremity with combined right upper ex-

tremity abduction, external rotation, and wrist and finger extension.
3. Increased subjective complaints of pain and paresthesia with concomitant cervical side-bending left and rotation right, or with full inhalation efforts.

Musculoskeletal Diagnosis

1. Elevated first rib on the right.
2. Type II non-neutral FRS left dysfunction at T1.
3. Type II non-neutral FRS right dysfunction at T2.
4. Myofascial dysfunction of the right anterior and medial scalenes (increased tone, decreased play, and functional excursion), right sternocleidomastoid (increased tone, decreased functional excursion), and levator scapulae musculature (decreased functional excursion).

Treatment

Initial treatment efforts were focused on mobilization of the type II non-neutral dysfunctions at T1 and T2 with muscle energy and high-velocity technique (see Chapter 9 for details). With vertebral correction noted with respect to positional and motion attributes, treatment efforts were directed at the first rib. Using MET, significant improvement in rib position and correlative cervical range of motion was noted (side-bending left, rotation right 90%). In addition, the patient reported a significant (75%) decrease in pain and hyperesthesia of the cervical musculature, as well as elimination of right upper extremity paresthesia (Fig. 10.30).

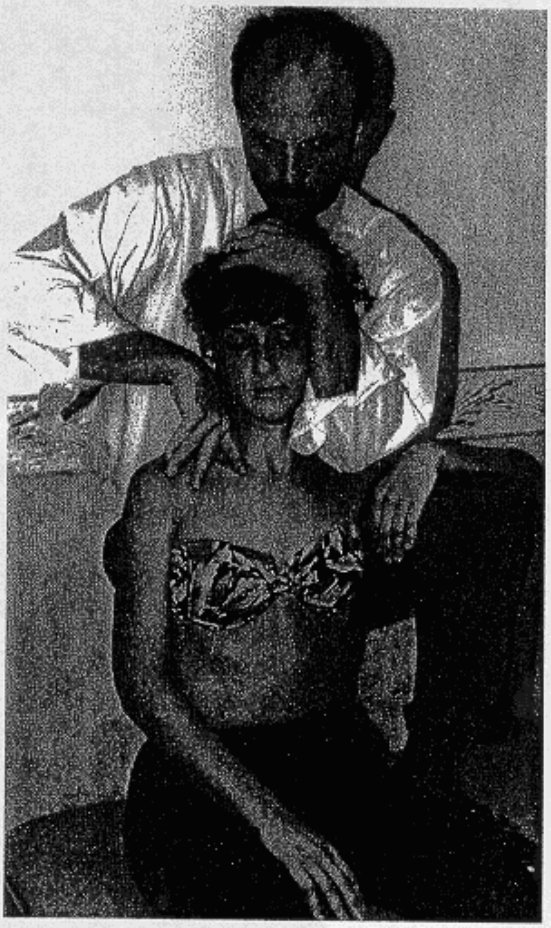


Figure 10.30. Muscle energy technique (MET) to the first rib in the seated posture to correct a superior subluxation of that rib.

The patient was subsequently instructed in postural re-education, as well as self-stretching of the right anterior and medial scalene, sternocleidomastoid, and levator scapulae muscles.

On returning to the clinic for her second visit, the patient complained of a re-exacerbation of the right cervical and upper extremity symptoms (pain and paraesthesia) after self-stretching. Re-examination revealed maintained correction of the type II non-neutral dysfunctions at T1 and T2. However, the right, first rib was once again displaced cranially with associated myofascial dysfunction as initially noted.

Treatment in this session focused on normalization of myofascial dysfunction of the anterior and medial scalene, sternocleidomastoid, and levator scapulae muscles with respect to increased tone, diminished play, and functional excursion. Techniques used included end-range perpendicular strumming of the scalene muscles (in a shortened range), parallel technique to the sternocleidomastoid, and strumming technique to the levator scapulae (seated/lengthened position) (Figs. 10.31, 10.32, and 10.33). Muscle energy technique was then used once again with the patient in a seated position to normalize the first rib.

In addition, after the position of the first rib was restored, functional excursion of the anterior and medial scalenes was addressed with the patient in a seated position, while providing counter support and stabilization to the right first rib. This was accomplished via digital contact and through a series of hold-relax elongation techniques. The patient was then instructed in home self-stretching technique of these muscles with counter support provided

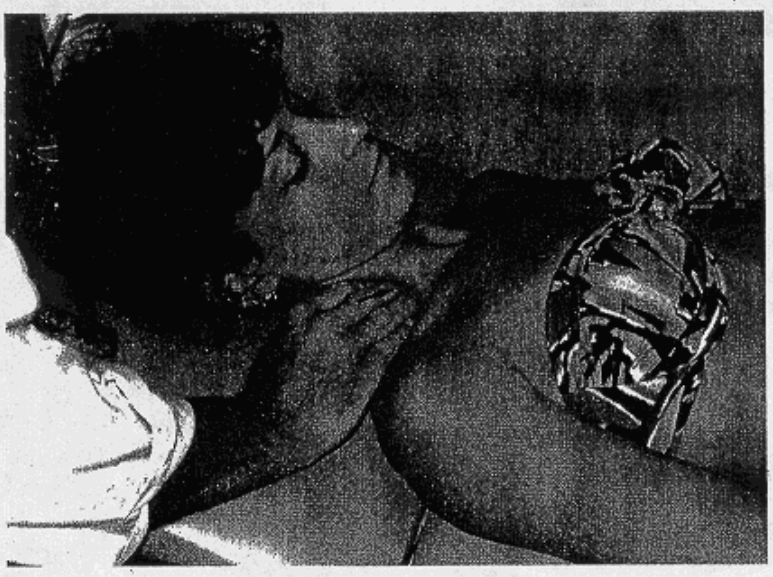


Figure 10.31. End-range perpendicular soft-tissue mobilization technique to the scalenes in a shortened position.

Figure 10.32. Parallel soft-tissue mobilization technique to the sternocleidomastoid.

through the use of a sheet, with a caudally directed force over the first rib (Figs. 10.34 and 10.35).

Treatment Progression and Analysis

Initial treatment efforts were directed at the correction of the type II non-neutral dysfunctions at the T1 and T2 vertebral levels. This strategy was used because position/motion dysfunction of these vertebral segments is commonly associated with and frequently causes dysfunction of the first rib, with resultant upper extremity brachialgic complaints. These efforts were followed by successful treatment of the first rib as noted. The patient was instructed in prescriptive stretching of the involved soft tissues (anterior and medial scalenes, sternocleidomastoid, levator scapulae) for a home program. However, on returning to the clinic the patient demonstrated recurrence of the elevated first rib on the right. This reportedly occurred when the patient was performing home stretching of the anterior and medial scalenes. Treatment was redirected toward the myofascial dysfunction of the involved soft tissues with particular emphasis on normalizing the play and

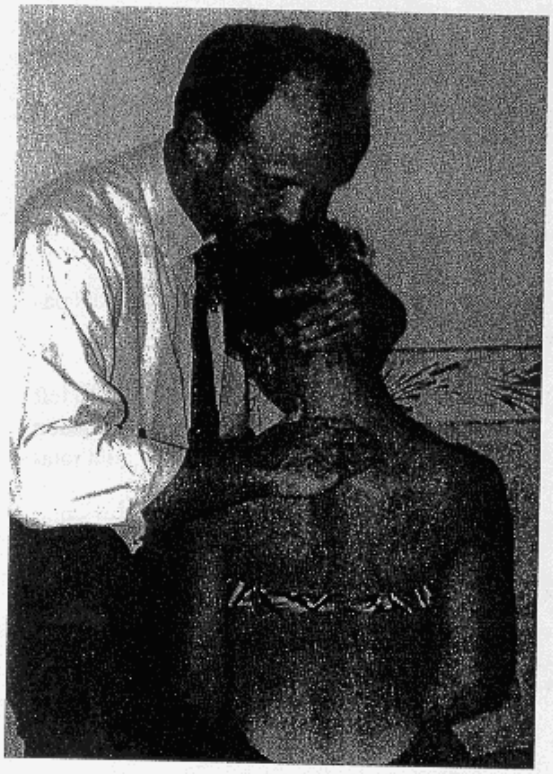


Figure 10.33. Strumming soft-tissue mobilization technique to the levator scapulae muscle.

length of the anterior and medial scalene muscles and the play of the sternocleidomastoid. In addition, the patient was instructed in self-stretching with appropriate counter support to the first rib.

This case emphasizes the importance of clearing soft-tissue dysfunction that may be implicated in the pathogenesis of vertebral and rib cage dysfunction. It also serves to illustrate how these tissues may perpetuate or cause recurrence of these dysfunctions if left untreated.

Case 2

C: Chief Complaint

A 32-year-old right-handed man was seen with complaints of localized left anterior chest wall pain, especially on full exhalation, over a 5-week period of time. He denied any neurologic symptoms including tingling, numbness, weakness, or radiating pain into either upper extremity.

H: History

This patient described a mechanical history of injuring himself while weight training. He described performing bilateral overhead flies with a 50-pound dumbbell while in a supine position and experiencing a strain of the left pectoral muscle group, which progressed over a 3-day period to include the anterior chest wall. After approximately a 3-week rest period, with treatment including ice packs and gen-

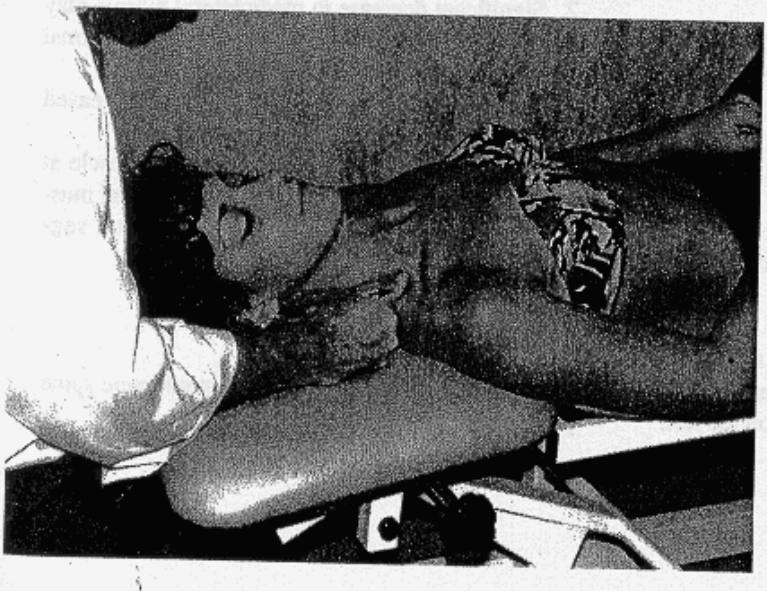


Figure 10.34. Functional excursion/elongation of the anterior and medial scalenes with counter support provided to the first rib through the thumb and second digit.

Figure 10.35. Self-stretch home program for the anterior and medial scalenes.

tle self-stretching, the patient attempted weight training once again. However, the chest wall pain increased and on examination was present with all exhalation efforts as well as overhead movements of the right upper extremity.

Other details of the history for this patient, including familial, past medical, pharmacologic, social, and occupational aspects, proved to be noncontributory.

A: Asymmetries of Bony Landmarks

The key asymmetric findings for this patient included:

1. Diminished anterior intercostal space between ribs 4 and 5 on the left with increased spacing posteriorly between ribs 4 and 5 on the left.
2. Anterior, posterior, and lateral rib contours that were unremarkable with respect to asymmetric prominence.
3. The superior margin of the fifth rib was palpable posteriorly, with a sharp border.
4. A type II non-neutral ERS left dysfunction was noted at the T4 vertebral dysfunction.
5. All other vertebral and rib segments appeared symmetric with the patient in the static seated posture.

R: ROM/Mobility Testing

Positive results of the key motion tests for this patient included:

1. Confirmation of the type II non-neutral ERS left dysfunction noted at the T4 vertebral dysfunction with diminished flexion, side-bending, and rotation to the right.
2. Aberrant respiratory motion testing on full exhalation on the left.
3. Diminished spring testing of the superior border of the left fifth anterior rib (cranial to caudal direction) with firm end-feel.
4. Diminished spring testing of the inferior border of the left fifth posterior rib (caudal to cranial direction) with firm end-feel.

T: Tissue Texture/Tension/Tonal Abnormalities

The key tissue abnormalities for this patient included:

1. Diminished caudal shear of the skin and superficial fascia in the left anterior clavipectoral region.
2. Significant decrease in mediolateral muscle play of the left pectoralis minor muscle at its proximal insertion of ribs 4 and 5.
3. Decreased functional excursion and increased tone of the left pectoralis minor muscle.
4. Increased tone of the left iliocostalis muscle at the fifth costal segment and the intercostal muscles between the fourth and fifth costal segments.

S: Special Tests

Radiologic assessment of the cervicothoracic spine produced negative findings.

Musculoskeletal Diagnosis

1. Type II non-neutral ERS left dysfunction at T4.
2. External torsion dysfunction of the left, fifth rib.
3. Exhalation respiratory dysfunction of ribs 3, 4, and 5 on the left, with rib 5 designated as the "key rib."
4. Myofascial dysfunction of the left pectoralis minor muscle (decreased muscle play, increased tone), the intercostal muscles (between costal segments 4 and 5), and the iliocostalis muscle at the fifth costal segment.

Treatment

Initial treatment efforts included MET directed at normalizing the type II non-neutral T4 ERS left dysfunction with correction noted after the first treatment session (Fig. 10.36). In addition, efforts were directed at normalizing the external torsion dysfunction of the left fifth rib with MET with the patient in both the supine and seated positions. This, however, proved resistant to treatment

During the second treatment session efforts to normalize the external torsion dysfunction with MET once again proved to be unsuccessful. Treatment efforts were redirected at the myofascial dysfunction of the superficial and deep clavipectoral fascia and the pectoralis minor and intercostal muscle groups.

The superficial fascial restrictions were ameliorated by identifying and directing treatment at the epicenter of the restrictive barrier in the fascia overlying the fifth costal segment. This was accomplished with a push technique (treating hand) and through placing the immediate surrounding tissues in a shortened range (assist hand) (Fig. 10.37).

The pectoralis minor demonstrated significant diminution in muscle play at the fourth and fifth costal segments on the left. In addition, the left pectoralis minor muscle demonstrated poor dissociation from the overlying pectoralis major muscle. These dysfunctions were treated by placing the muscle group in a shortened position and directing a perpendicular mobilization force to the respective barrier (keeping the tissue barrier level and the direction of the restriction in mind). In addition, a strum technique was used to restore muscle play while decreasing muscle tone, as well as to improve dissociation of the two muscles. A parallel technique

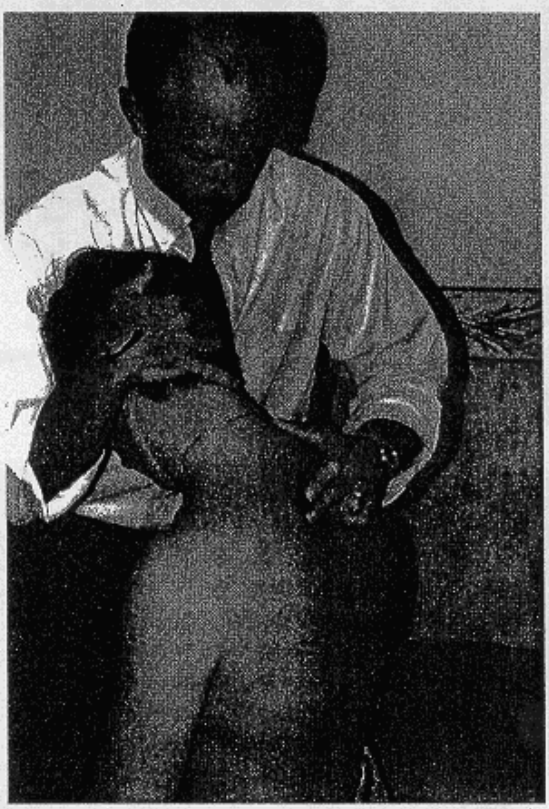


Figure 10.36. Muscle energy technique (MET) to the T4 vertebral segment in the seated position to correct a type II nonneutral ERS left dysfunction with diminished flexion, sidebending, and rotation to the right.

was also used along the lateral seam of this muscle with a significant reduction in tone and moderate gains in muscle play noted. These techniques were used while progressing from a shortened to fully lengthened position (Figs. 10.38 and 10.39).

The intercostal muscle groups between the left fourth and fifth ribs were treated with bony contour technique with the patient in a sidelying position to lengthen or increase the intercostal spacing. This technique was performed circumferentially (Fig. 10.40).

After treatment, reassessment of the external rib torsion dysfunction demonstrated improvement of approximately 75% (both objective and subjective). MET was next used to completely normalize position and motion characteristics to the structural rib dysfunction. After this correction, the respiratory movements also proved to be normalized, with absence of the previous group exhalation dysfunction noted at ribs 3, 4, and 5.

Figure 10.37. Superficial fascial soft-tissue mobilization push technique to the clavipectoral fascia over the fifth costal segment.

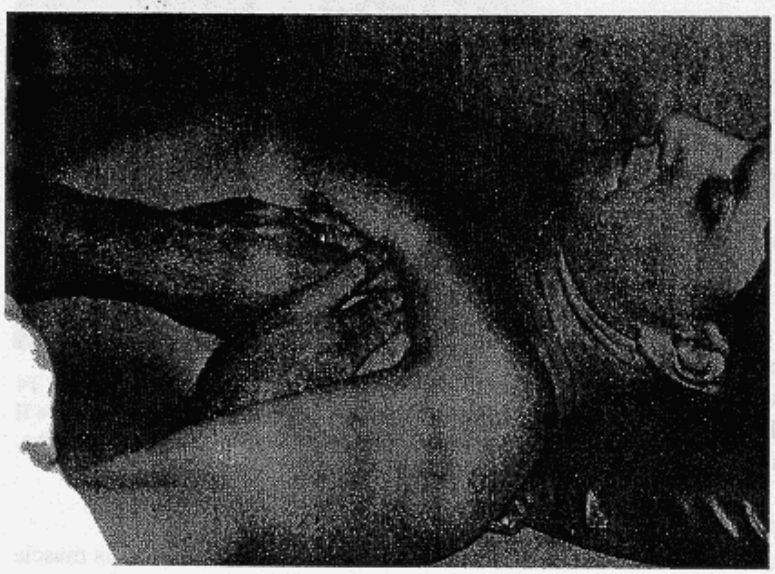


Figure 10.38. Strumming soft-tissue mobilization technique to the pectoralis major/minor complex with digital contact.

The treatment session was completed with the patient engaged in a posterior-elevation/anterior-depression proprioceptive neuromuscular facilitation (PNF) pattern with a combination of isotonics (placing, isometrics, concentrics, eccentrics, slow reversal hold) to provide neuromuscular re-education, improved functional excursion, and challenge the pectoralis minor muscle and its corresponding rib segments (Fig. 10.41 and 10.42).

The patient was instructed in a home program that included self-strumming and doorway stretching to the left pectoralis minor muscles, followed by diaphragmatic, diagonal breathing patterns to maintain respiratory correction.

Treatment Progression and Analysis

Initial treatment attempts were directed at correction of the ERS T4 dysfunction because this commonly causes external torsion dysfunction of the rib cage. Correction of the vertebral dysfunction will often yield concomitant correction of the rib dysfunction.

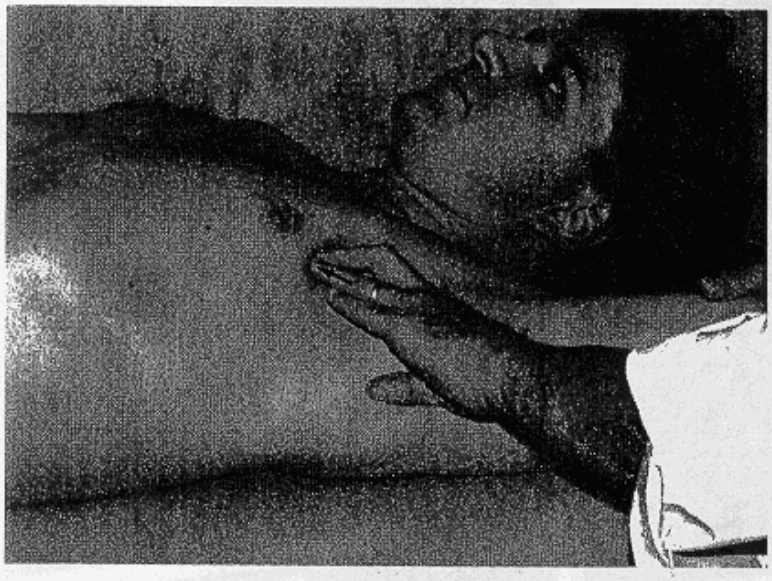


Figure 10.39. Parallel soft-tissue mobilization technique to the pectoralis major/minor complex in a lengthened position.

Figure 10.40. Bony contour soft-tissue mobilization technique to ribs 4 and 5. This is performed circumferentially about the entire rib cage.

This, however, is not true where chronicity and myofascial dysfunction coexist. Attempts to use MET in correcting the external rib torsion dysfunction (which was also acting as the "key rib" in the exhalation dysfunction) were unsuccessful secondary to the significant myofascial dysfunction of the pectoralis minor muscle (most notably, diminished play and increased tone). After normalization of the myofascial tissues (particularly muscle play and tone at the pectoralis minor muscle), both muscle energy and PNF techniques were carried out with success, with the external torsion dysfunction normalized and the associated exhalation dysfunction corrected simultaneously.

This case serves to demonstrate the importance of myofascial work in combination with traditional MET as both preparative and corrective. In addition, it illustrates the importance of considering all of the attributes of soft tissues (i.e., muscle play, tone, functional excursion, and neuromuscular control) in contrast to length or functional excursion only.

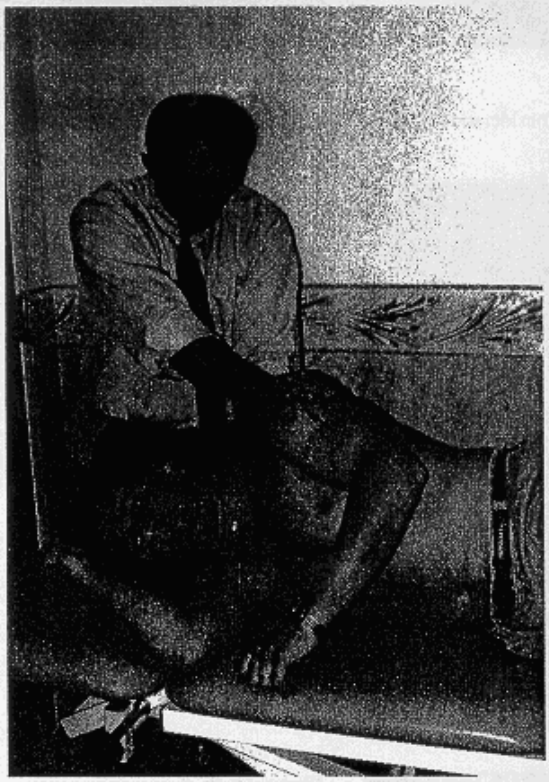


Figure 10.41. Proprioceptive neuromuscular facilitation, posterior elevation pattern to the shoulder girdle to challenge the corrected rib segment as well as improve neuromuscular control.

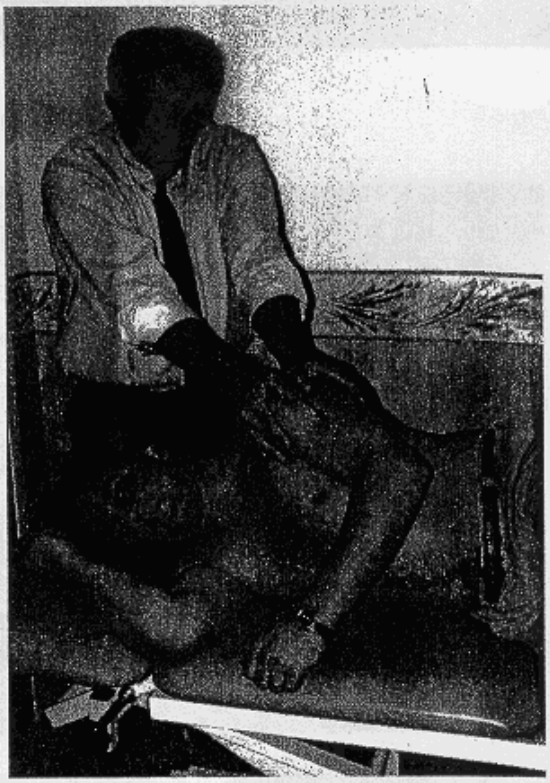


Figure 10.42. Proprioceptive neuromuscular facilitation, anterior depression pattern to the shoulder girdle.

Case 3

C: Chief Complaint

A 25-year-old female body-builder was seen with a 3-month history of thoracolumbar and right lower rib cage pain, especially with rotation to the left and on inhalation efforts.

H: History

This patient described injuring herself while attempting to lift a heavy object from a flexed position. She described a "catching sensation" in the lower thoracic spine and immediate discomfort in the right anterior lower rib cage. She also reported a similar injury in the same region approximately 2 years earlier.

Other details of the history for this patient, including familial, pharmacologic, social, and occupational aspects, were noncontributory.

A: Asymmetries of Bony Landmarks

The key asymmetric findings for this patient included:

1. A type II non-neutral FRS right dysfunction at T10.
2. A type I neutral, side-bent right, rotated left dysfunction at T11, T12, and L1.
3. Leg length discrepancy (short left approximately 6 mm) with an elevated right iliac crest, elevated right anterior superior iliac spine, elevated right posterior superior iliac spine, and elevated right greater trochanter and fibular head.

R: ROM/Mobility Testing

Positive findings on the key motion tests for this patient included:

1. Confirmation of a type II non-neutral FRS right dysfunction at T10 with diminished extension, side-bending, and rotation to the left.
2. Confirmation of a type I neutral, side-bent right, rotated left dysfunction at T11, T12, and L1 with diminished side-bending left and rotation right at T11, T12, and L1.
3. Aberrant respiratory motion testing on full inhalation of the right lower rib cage.

T: Tissue Texture/Tension/Tonal Abnormalities

The key tissue abnormalities for this patient included:

1. A significant increase in tone of the right iliopsoas muscle, the right respiratory diaphragm (along the inferior border of the anterior rib cage), and the right multifidus musculature (at the T10, T11, and T12 levels).
2. Diminished mediolateral muscle play of the right iliopsoas muscle at the L1, L2, and L3 levels.
3. Diminished mediolateral muscle play and functional excursion of the right quadratus lumborum musculature.

S: Special Tests

The results of special tests were all noncontributory or negative for this patient.

Musculoskeletal Diagnosis

1. Type II non-neutral FRS right dysfunction at T10.
2. Type I neutral, side-bent right, rotated left dysfunction at T11, T12, and L1.
3. Respiratory inhalation dysfunction of ribs 10, 11, and 12 on the right.
4. Leg length discrepancy, short on the left (approximately 6 mm).
5. Myofascial dysfunction of the right iliopsoas muscle (decreased muscle play, increased tone), right respiratory diaphragm (increased tone), right multifidus (increased tone), and right quadratus lumborum (decreased play and functional excursion).

Treatment

Because of the significance of this patient's myofascial dysfunction, initial treatment efforts were directed at the increased muscle tone identified along the inferior border of the rib cage (respiratory diaphragm) and the tone/muscle play problems of the right iliopsoas muscle complex. The respiratory diaphragm was initially treated with the patient seated (shortened position), followed by the supine position (lengthened position). Soft-tissue mobilization efforts included direct, sustained pressure administered to the respiratory diaphragm while the patient was encouraged to provide gentle inhalation and exhalation efforts. In addition, the bony contours of the costochondral arch were addressed with direct, end-range oscillatory mobilization technique directed at the specific restrictions encountered (Figs. 10.43 and 10.44).

The right iliopsoas muscle dysfunction was treated with a combination of perpendicular mobilization and strumming techniques. This was performed with the patient in a supine 90/90 posture (shortened position), which progressed to a lengthened position. This was followed by soft-tissue mobilization of the multifidus muscle through strumming and bony contour techniques directed along the groove between the spinous and transverse processes of the thoracolumbar spine with the patient in a quadruped, sitback position (lengthened position) (Figs. 10.45, 10.46, and 10.47).

After the correction of this MFD, MET was used to correct both the type II FRS right dysfunction at T10 (which was already approximately 50% improved) and the respiratory (inhalation) dysfunction. These efforts resulted in approximately 75% improvement with respect to vertebral and rib position/motion dysfunction, as well as specifically enumerated myofascial dysfunction. Three additional treatment sessions were required (with similar progression) to completely normalize vertebral and rib dysfunction.

On returning to the clinic for the fourth session, the patient demonstrated recurrence of the FRS right dysfunction at T10 and had complaints of pain at the thoracolumbar junction posteriorly. Treatment was redirected at the quadratus lumborum with a functional excursion/lengthening technique (treating hand), in a lengthened position over a bolster, while the assisting hand provided end-range associated oscillations (Fig. 10.48).

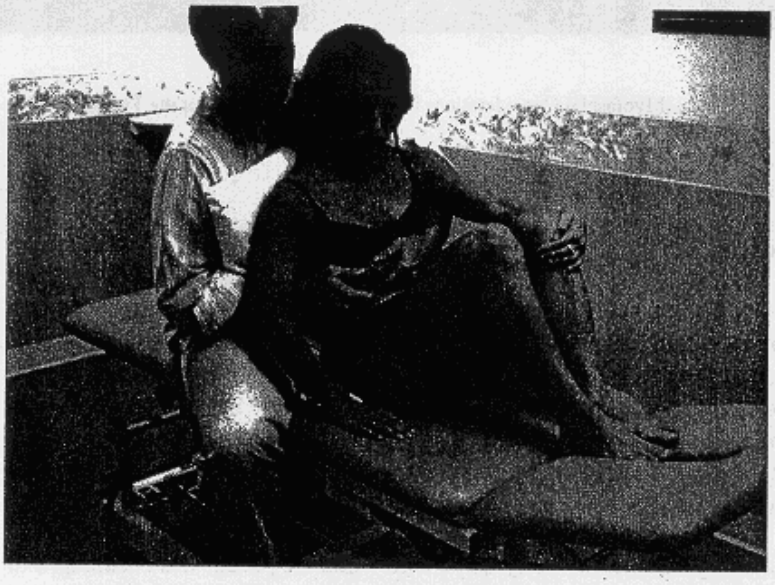


Figure 10.43. Respiratory diaphragm, bony contour, soft-tissue mobilization technique performed in a seated, shortened position.

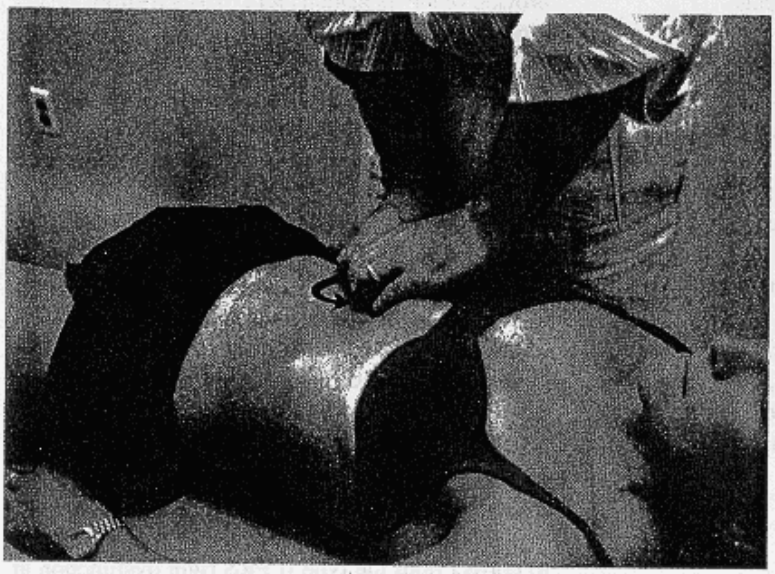


Figure 10.44. Respiratory diaphragm, bony contour, soft-tissue mobilization technique performed in a supine, lengthened position.

Once again, MET was used to correct the type II FRS right dysfunction at T10, followed by proprioceptive neuromuscular facilitation (PNF) for re-education with anterior elevation/posterior depression patterns through the pelvis (Figs. 10.49 and 10.50).

This treatment was followed by the introduction of a 6-mm heel lift to the short lower extremity to level the sacral base. No further complaints were offered and the patient was discharged.

Treatment Progression and Analysis

Treatment efforts initially directed at the myofascial dysfunction of the iliopsoas muscle and respiratory diaphragm allowed substantial progress to be realized in the normalization of the type II FRS and respiratory rib cage dysfunctions. This, however, was only temporary, and recurrence of these movement dysfunctions appeared related to the remaining my-

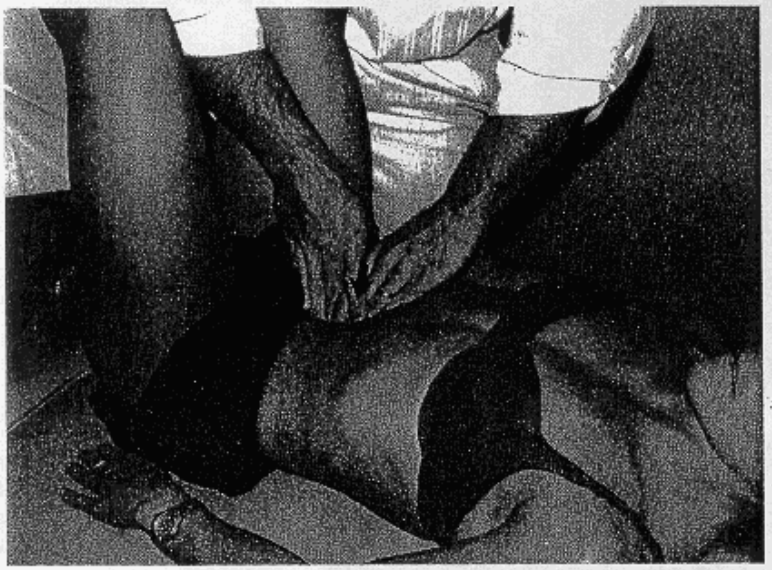


Figure 10.45. Muscle play/tone, soft-tissue mobilization technique to the iliopsoas muscle complex in a supine, shortened position.

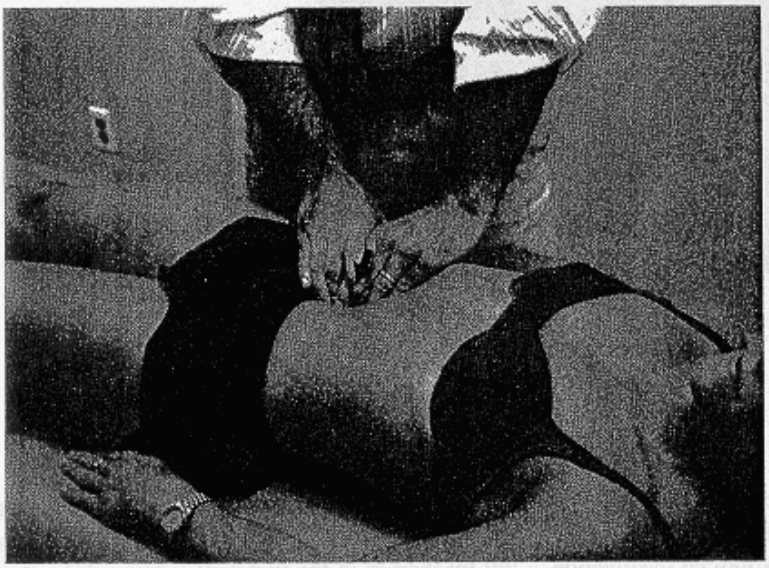


Figure 10.46. Muscle play/tone, soft-tissue mobilization technique to the iliopsoas muscle complex in a supine, lengthened position.

ofascial dysfunction and leg length discrepancy. Asymmetric muscle length and aberrant tone of the quadratus lumborum (secondary to the long right leg) provided an environment for non-neutral mechanics to exist in the thoracolumbar spine and apparently contributed to the recurrence of the type II dysfunction.

Treatment was redirected toward the quadratus lumborum and the correction of the leg length discrepancy. This provided an environment for the correction of the type II FRS dysfunction, accomplished through MET, to be maintained. Although type II nonneutral FRS and ERS dysfunctions are not typically recognized as being compensatory to a type I neutral dysfunction (in this case, secondary to a leg length), they are commonly seen as nonresponsive or recurrent in the presence of this static postural alteration. In addition, where chronicity prevails, related myofascial

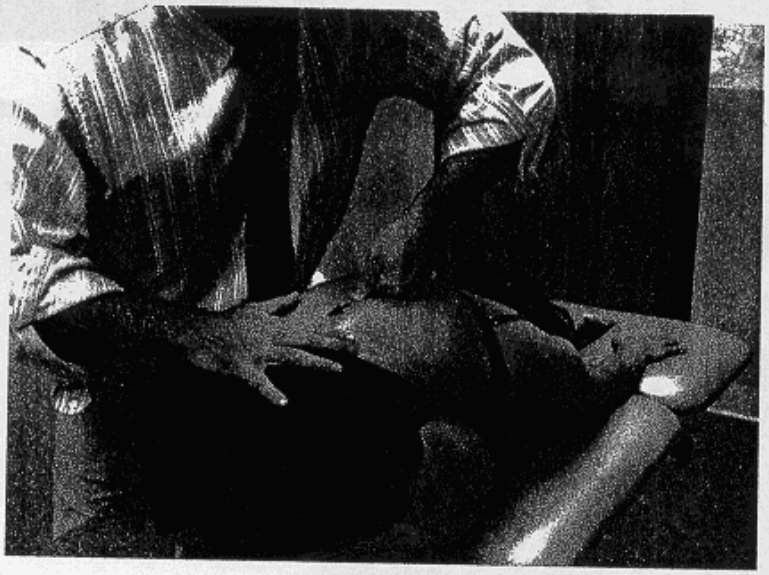


Figure 10.47. Bony contour, soft-tissue mobilization technique to the thoracic spine, paravertebral muscles and myofascial tissues in a quadruped (all fours), sit-back position.

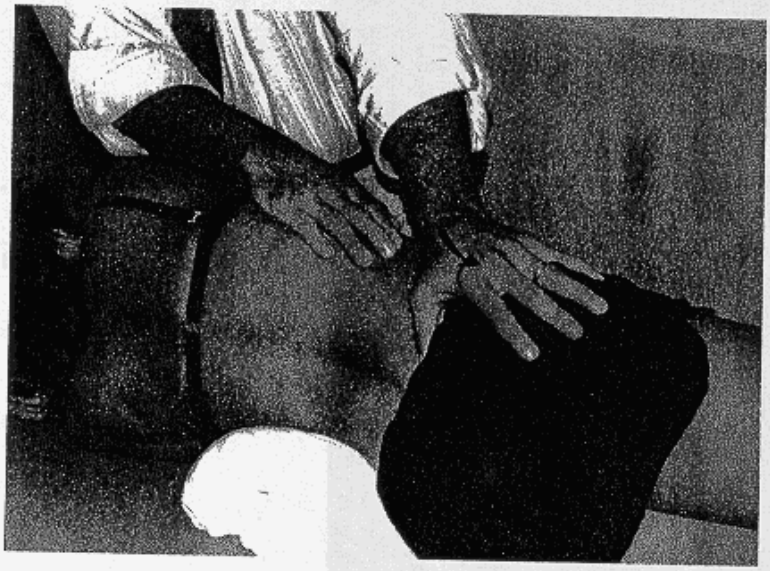


Figure 10.48. Functional excursion/lengthening technique to the quadratus lumborum with the assisting hand providing associated oscillations.

dysfunction makes correction of these articular dysfunctions more recalcitrant to treatment efforts.

This case illustrates how myofascial dysfunction accompanying static postural dysfunction (in this case, leg length dysfunction) may provide biomechanical alterations that make correction of the primary somatic dysfunction difficult, if not impossible. It also emphasizes the importance of achieving neutral mechanics in the vertebral column through the normalization of both myofascial and skeletal structures in efforts to avoid recurrence of thoracic and rib cage dysfunction.

Figure 10.49. Proprioceptive neuromuscular facilitation, anterior elevation pattern to the pelvic girdle.

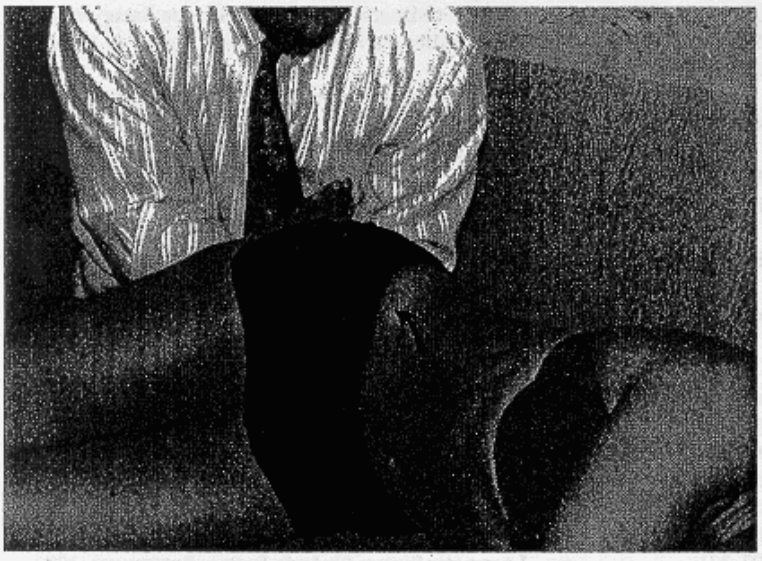


Figure 10.50. Proprioceptive neuromuscular facilitation, posterior depression pattern to the pelvic girdle.

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