Thoracic outlet syndrome—A myofascial variant: Part 3. Structural and postural considerations

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Thoracic outlet syndrome involves more than just local neurovascular compression. Myofascial release treatments and stretching exercises may be only partially or temporarily successful unless all related components of somatic dysfunction, including craniosacral mechanisms, are addressed. Structural and postural abnormalities in the frontal plane, as with a short leg, and in the sagittal plane, such as lumbopelvic imbalances, as well as neural involvement all contribute to thoracic outlet syndrome symptoms. Once segmental restrictions are treated and symptoms diminish, postural correction and strengthening exercises may be initiated. Osteopathic diagnosis and treatment of the local, regional, and remote structural problems is necessary for optimal treatment of thoracic outlet syndrome and the maintenance of a symptom-free status.

(Key words: Thoracic outlet syndrome, myofascial pain syndrome, somatic dysfunction, short leg, pelvic rotation)

Thoracic outlet syndrome (TOS) is often refractory to management. Many therapeutic approaches overlook or even omit factors necessary for successful treatment. Parts 1 and 2 of this paper made some common observations on initial clinical and diagnostic examination of patients with TOS. This part extends these observations to a more comprehensive overview of contributing factors, encompassing focal, regional, and remote involvement, as well as spinal pathology and neurophysiology.

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Structural and functional considerations

The previous emphasis on the myofascial aspects of TOS[1,2] confines the problem to mainly one paradigm. The myofascial pain theory of Travell and Simons[3] is based on the premise that "primary" dysfunction is diagnosed and treated in one muscle. The limitations of this focused approach are especially important in the parascapular region where overlapping thin muscles become dysfunctional and behave as though adhered together (*Figure 1*) within a unit. Osteopathic approaches that address this complexity more effectively are required.

Furthermore, the localized dysfunction at the thoracic outlet may be one aspect, perhaps just a regional component, of a widespread, global neuromusculoskeletal or even systemic viscerosomatic dysfunction. Most likely, either aspect of this diffuse process of dysfunction could be causative or perpetuating. Once established, all the following—the TOS (local somatic and myofascial dysfunction with neurovascular irritation at the thoracic outlet) and regional and remote somatic dysfunction interrelate and influence each other.

(continued on page 340)

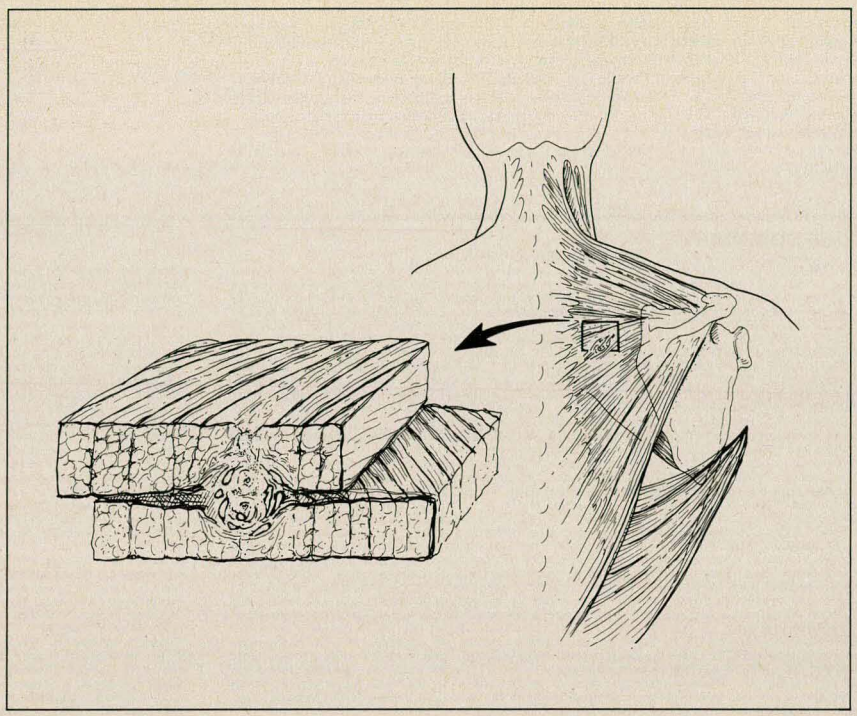

Figure 1. *Somatic/myofascial dysfunction: Dysfunction often occurs in more than one muscle, particularly about shoulder girdle. This dysfunctional unit affects surrounding or adjacent tissues, including bone (such as ribs and scapula), blood vessels, and nerves.*

In the acute phase, after injury, and even in the subacute phase, the active trigger points, or acute somatic dysfunction, generates muscle spasm, which "pulls" (retracts) the scapula toward the midline or spine. In addition, use of the parascapular muscles, in particular, the rhomboids and middle trapezius (fibers), is relatively inhibited because of pain and dysfunction. A relative disuse or functional weakness ensues and progresses as long as the dysfunction or pain or both persist. Chronically, this disuse allows the shoulder girdle to protract (*Figure 2*), thus leading to TOS.

Whole body disturbances as related to the primary respiratory mechanism must also be considered. Thoracic outlet syndrome is commonly seen as a late sequela of traumatic hyperextension/hyperflexion injuries typical of automobile rear-end collisions (whiplash).[1] Craniosacral restrictions develop because the cranium is susceptible to "transverse bind," possibly with the thoracic inlet, and such tightness can affect the dynamics of the body as a whole.[4] Furthermore, spinal mechanics involving lumbar, sacral, pelvic, and lower extremity function can have major effects on the shoulder girdle and TOS.

Mechanical linkage

Frontal plane decompensation. When postural imbalance occurs, as with a short leg, the alignment in the frontal plane is disturbed. The result is asymmetric pull or tension on several myofascial units (*Figure 3*), which can progress to dysfunction and the development of trigger points or somatic dysfunction.[5-7] In fact, Beal has noted associated "thoracic distress"[7] as well as shoulder pain[8] with leg length discrepancy. This frontal plane decompensation may perpetuate shoulder girdle dysfunction leading to symptoms referred to as TOS.

Sagittal plane decompensation. In the sagittal plane, excessive anterior pelvic rotation (tilt) results in lumbar hyperlordosis (*Figure 4, center*). As a compensatory mechanism, there is a relative increase in thoracic kyphosis, which tends to "throw" the shoulders forward (protraction), thereby encouraging the "thoracic outlet posture."[1] A posterior pelvic rotation also creates a compensatory shoulder protraction, even though the lumbar spine becomes hypolordotic (*Figure 4, right*). Apparently any imbalance of the lumbopelvic mechanics could become an etiologic and perpetuating factor in TOS.

The dynamic relationship between the shoul-

der and pelvic girdles cannot be overstated.[9] The pelvis of many patients with TOS resistant to local treatment is literally "hung up." Until the sagittal pelvic mechanics are "neutralized" or stabilized the shoulder girdle will be "driven" forward, continuing to close the thoracic outlet, regardless of how effective the myofascial release treatments and stretching exercises are. Strengthening exercises also will have limited effectiveness, or may create other symptoms elsewhere, trying to overcome the disordered lumbopelvic mechanics.

Neural linkage

As the musculoskeletal and postural stressors contribute to TOS, neural influences also play a significant role. Denslow and coworkers[10] have demonstrated that reflex thresholds are lowered at segments of somatic dysfunction. These segments, which are hyperexcitable, have been designated "facilitated." By vertically organized communication, both motor and sympathetic outflow may show an exaggerated response to even innocuous stimuli from remote sites[11] (*Figure 5*). This heightened activity may cascade to neurovascular events and visceral functions as has been extensively discussed by Korr[11] and is beyond the scope of this article.

In the case of TOS, the associated upper thoracic somatic dysfunction may serve as a hub of aberrant neural communication. This could partially account for the development and perpetuation of TOS. W. E. Wyatt, DO, (personal communication, written July 1992) has identified a hypersensitive point in the deep fascia that, when palpated, will reproduce symptoms of TOS. Constant, deep, inhibitory pressure is

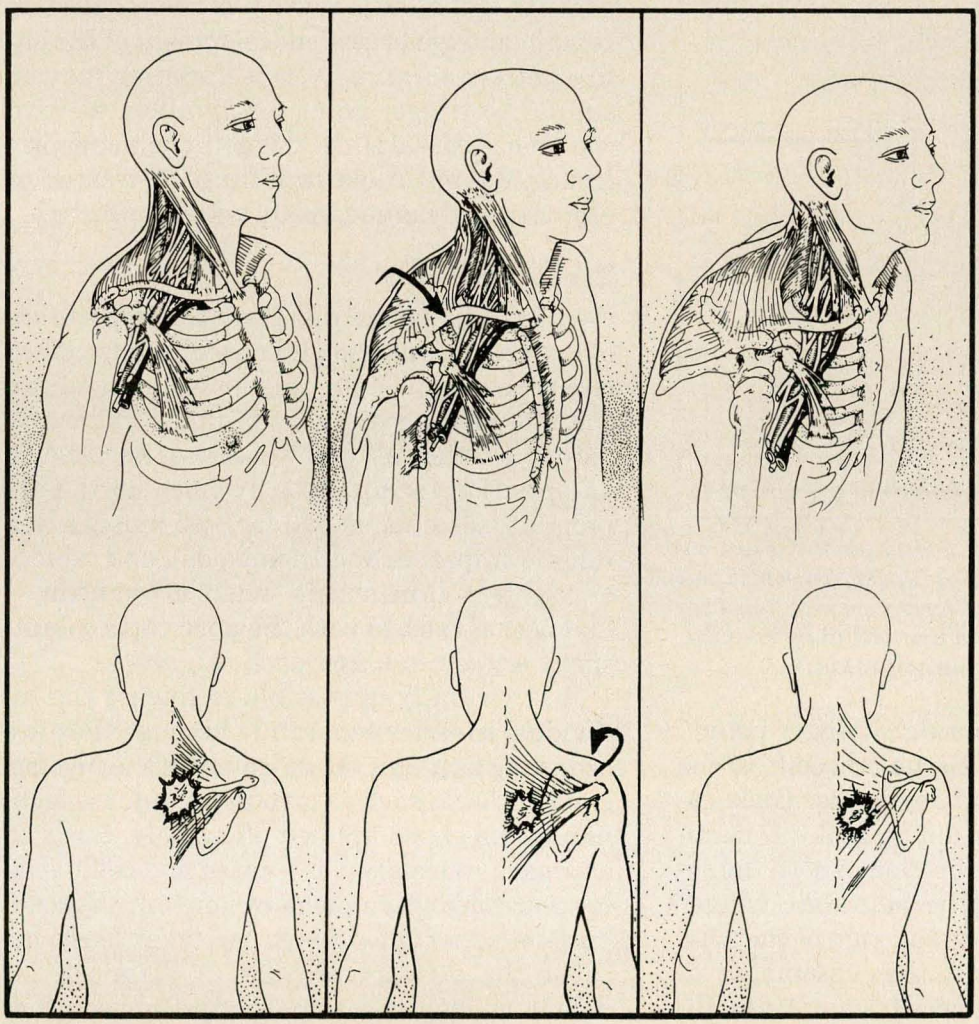

Figure 2. *Progressive scapular protraction: Functional weakness gradually develops as a result of disuse secondary to painful inhibition of muscular activity from active trigger point or somatic dysfunction. Anterior view with secondary effects on thoracic outlet is illustrated in top frame and posterior view, in bottom frames.*

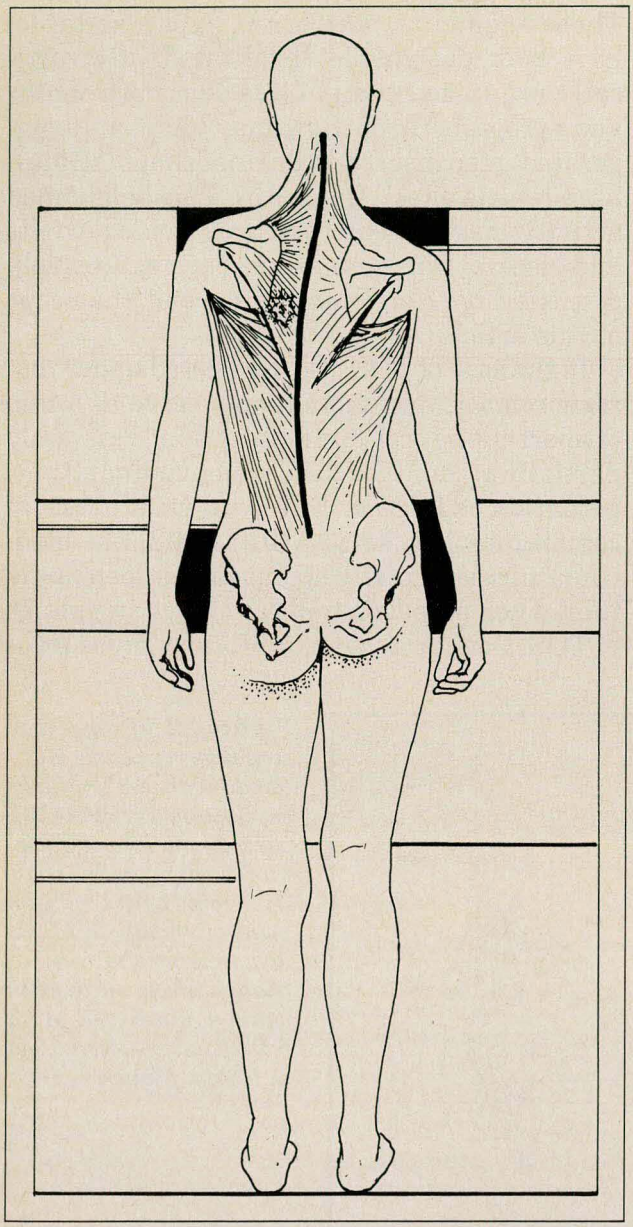

Figure 3. *Frontal plane decompensation: A short leg will create obvious mechanical strain pattern, which will be transmitted superiorly, leading to or perpetuating trigger point activation and somatic dysfunction as far cephalad as parascapular region or shoulder girdle.*

used to release this "neuralgic trigger point." It is located in the soft tissue adjacent to the upper thoracic vertebral segments. Once established, it is self-perpetuating and will maintain TOS symptoms. Resolution of both the somatic dysfunction and the neuralgic trigger point are necessary for dissolution of the TOS symptoms. Larson[12] noted that vasomotor responses in an upper extremity may be facilitated in association with somatic dysfunction producing a clinical syndrome resembling the reflex sympathetic dystrophy. The strategic location of sympathetic nerves makes this a vulnerable site that may cause havoc in all related visceral structures. In addition, peripheral nociceptor branches from the upper thoracic region are believed to project to the brachial plexus[13] and may further enhance the neural involvement.

The enduring nature of the associated upper extremity symptoms may be explained on the basis of a form of "memory," at least at the spinal cord level. This view is similar to the "engram" theory of myofascial pain[2] and the "spinal fixation" phenomenon discussed by Patterson and Steinmetz.[14] Memory is a critical factor. It is clinically observed that treating only one or two focal areas of a somatic dysfunction pattern with three or more components will allow a portion of the memory to remain and generate redevelopment of the entire pattern (*Figure 5*). This situation may be similar to trigger point reactivation, as with the so-called satellite trigger phenomenon.[4] Hence, the entire pattern must be treated or eradicated if management is to be effective.

Treatment

Part 2 of this series[2] discussed the importance of myofascial release and stretching for effective treatment of TOS. It is essential to relieve the immediate symptoms of upper extremity pain, paresthesias, and weakness. Because no one position simultaneously lines up the appropriate direction of pull for both muscles (the middle trapezius and rhomboids), one rapidly encounters limitations when attempting a myofascial release with the pure vapocoolant-spray and stretch approach (*Figure 1*).

It is virtually impossible to release the individual muscles separately because they are operating as a unit. Modification of the myofascial approach, such as myofascial release technique,[2] has been applied effectively. Such an approach in isolation is limited and could lead to recurrence of symptoms or long-term dependence on active treatment, partly, at least, because the primary problem or diagnosis involves widespread somatic dysfunction, not

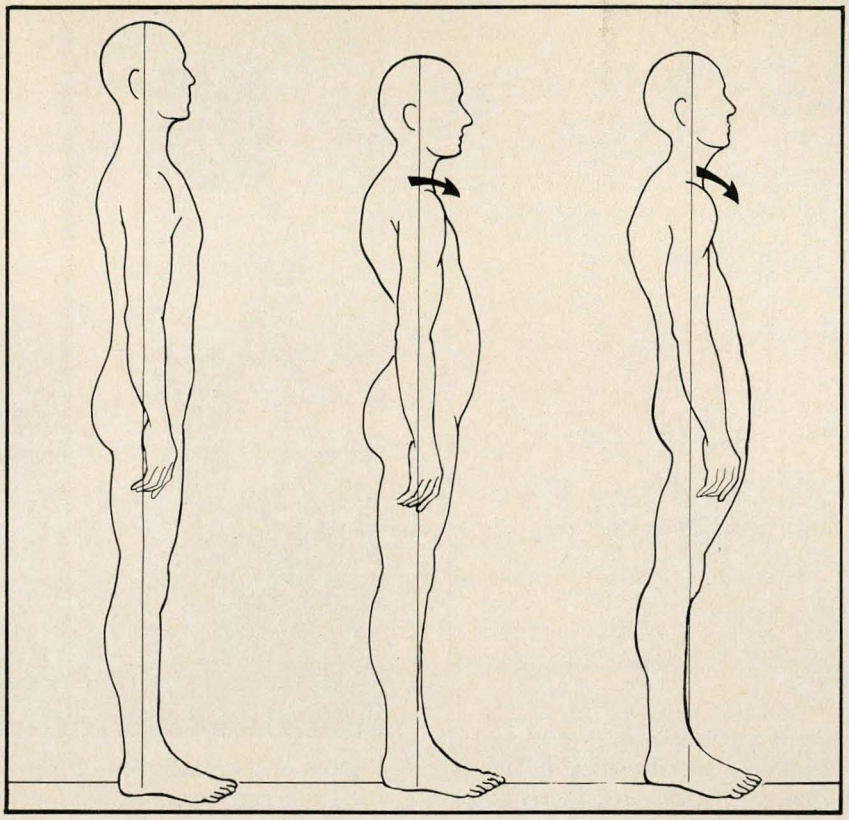

Figure 4. *Sagittal plane decompensation: Two abnormal (center and right) postures, in contrast to normal posture (left), can lead to shoulder protraction and thoracic outlet syndrome. With both hyperlordosis (center) and hypolordosis (right), the upper thoracic and shoulder girdle region destabilizes to compensate for lumbopelvic shift.*

just local neurovascular compression at the thoracic outlet.

Larson[12] discussed the need to address both myofascial and segmental components with TOS, emphasizing that after the deep musculature has been released, treatment of restrictions of isolated spinal segments can be accomplished relatively easily. Thus, it is essential to evaluate and treat all related components of somatic dysfunction, including the craniosacral mechanism.

Postural changes in the sagittal plane, especially in the lumbopelvic region, must be addressed (*Figure 6*). Osteopathic structural treatment, stretching exercise, and posture retraining with strengthening usually are required. The use of a pelvic orthosis such as the Levitor[9] may be considered. By stabilizing or controlling the pelvic region in the sagittal plane, such a dynamic orthosis will indirectly improve mechanics and alignment of the shoulder girdle. Similarly, postural factors in the frontal plane must be controlled, as with appropriate use of a heel lift.

Strength and especially endurance of the parascapular muscles must be regained to maintain or even achieve adequate postural and structural integrity. An exercise program beyond simple stretching is required. Weights or resistance exercise must be used. Initially, emphasis is placed on high repetition and low resistance. Otherwise, excess shortening of the muscles will lead to recurrence or reactivation of trigger points and pain.

The use of elastic bands (Thera-Band, the Hygenic Corp, Akron, Ohio) to generate resistance and build strength is ideal for the shoulder girdle region and therefore TOS. One key to effective technique is to minimize upper extremity abduction (transverse plane) against resistance. Abduction beyond 45 degrees will allow excessive shortening of the parascapu-

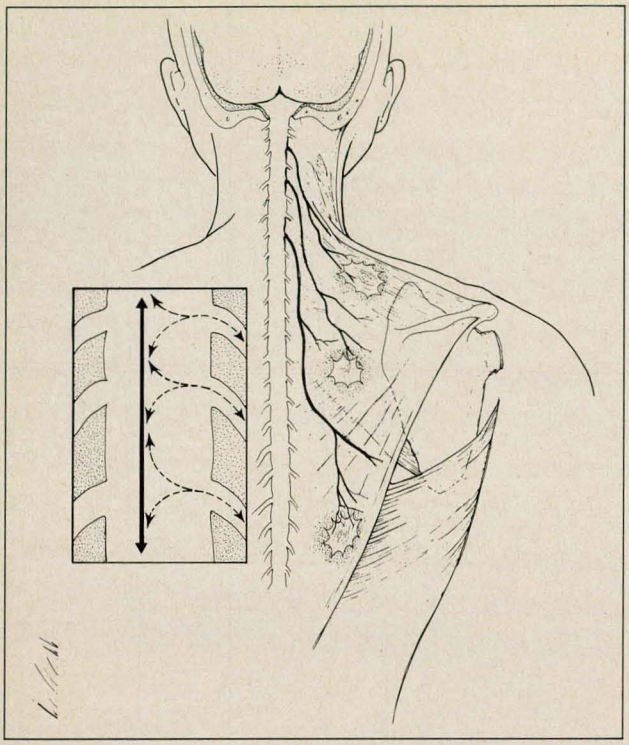

Figure 5. *Vertically organized intersegmental influences can profoundly affect other segments, particularly at hyperirritable areas of somatic dysfunction. To eradicate or alleviate pattern of dysfunction, all focal, segmental areas must be treated. If even one segment is left, entire pattern may be reactivated, because of "memory" phenomenon.*

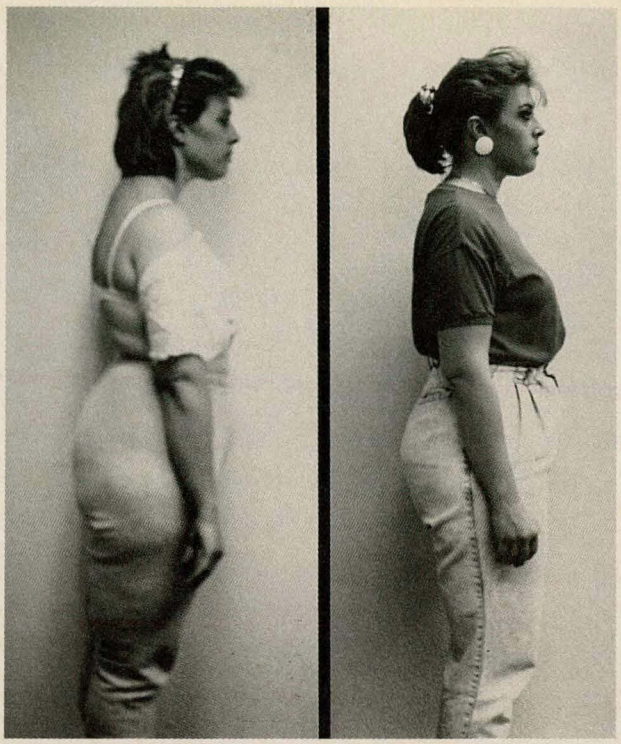

Figure 6. *Sagittal plane posture of patient with thoracic outlet syndrome before (left) and after (right) treatment. Notice "release" of pelvis that occurred, with reduction in hyperlordosis, which allowed shoulder girdle to "set back" and open thoracic outlet.*

lar muscles (the rhomboids and middle trapezius) and possible reactivation of trigger points. Additionally, arm position should be varied to include all components of the parascapular system. This variation requires horizontal (90 degrees humeral flexion-sagittal plane) positioning as well as angling upward and downward 45 degrees.

Comment

The upper extremity pain, paresthesias, and weakness usually referred to as TOS are considered sequelae of a local shoulder girdle phenomenon, but in most cases they have distant connections or associations, both cephalad and caudad. These other associations may be either perpetuating or causative. Such complexities could account for the controversy often associated with TOS. Effective treatment, to be more curative than palliative, must be holistic and address the entire body structure. Simple "release" of all areas of restriction is suboptimal without including exercise to correct posture. Exercises to improve strength and endurance are necessary to relieve strain on the thoracic outlet and allow the patient to maintain the new posture and remain symptom-free.

The upper extremity symptoms associated with TOS represent the effects of a more diffuse and generalized pathologic process of somatic dysfunction and trigger points. Vigorous and localized treatment directed to the outlet itself is usually required to break up the vicious cycle that commonly results in the predominantly upper extremity symptoms. Attention to the global neuromusculoskeletal considerations then is required to achieve the most successful outcome.

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