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Literature Review

Correcting Postural Deviations: An Evidence-Supported Shift in Paradigm

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ABSTRACT

Somatic syndromes of the spinal region may develop secondary to postural dysfunction. Traditionally, emphasis has been placed on correcting postural asymmetries by attempting to change muscle-tendon unit length. The effectiveness of this approach in providing prolonged muscle-tendon unit length changes and optimal postural outcomes has met with controversy. The time has come for a redirection of goals in conservative care for postural dysfunction of the spinal regions. An emphasis needs to be placed on continuous, self-awareness training of postural correction that will facilitate lasting neuromuscular adaptations. The purpose of this review is to rationalize the need to veer from exercises attempting to adjust the muscle-tendon unit length and recognize the evidence-supported impact of simple self-correction strategies meant to facilitate neuromuscular adjustments and correct postural asymmetries.

Introduction

Poor posture, such as a forward head-rounded shoulder or slumped sitting type postural abnormality has been suggested as a risk factor for neck and low back pain in individuals in the workplace [1] or students in the classroom [2]. Janda introduced specific postural syndromes causing muscle tightness and weakness imbalances that present a risk factor for neck and low back pain that commonly occurs because of poor static postural positions [3]. For example, upper crossed syndrome results in the tight, overly active upper trapezius and pectoralis major muscles and weak, deep neck flexor, lower trapezius, and serratus anterior muscles. He also introduced a lower crossed syndrome that resulted from tight, overly active erector spinae and iliopsoas muscles and weak, abdominal and gluteus maximus muscles [3]. He suggested muscle imbalances occur with sustained positions making it necessary to address weak muscles with strengthening exercises and tight muscles with stretching exercises to restore normal muscle balance [3].

Research has suggested if these poor postural positions are held statically for long lengths of time, frequently, muscle physiological changes may take place [4]. For example, participants with a forward head posture demonstrated significantly lower sternocleidomastoid muscle activation compared to controls, suggesting a change in muscle activation secondary to the length-tension relationship [4]. Specific strengthening exercises to adaptively shorten the muscle-tendon units were proposed to correct postural malalignments [5] but muscle-tendon unit shortening has not occurred with resistance training protocols [6, 7]. Clinical approaches to address postural abnormalities that could result

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in neck or low back pain have traditionally been based on the theoretical correction of muscle-tendon unit length imbalances with stretching and strengthening interventions. An evidence-based approach to correct postural abnormalities in the clinical setting is needed. The purpose of this review is to analyze the mechanisms that are thought to result in muscle-tendon unit length changes. We also compare the effectiveness of traditional stretch-strengthening protocols to exercises that stimulate the sensorimotor control system on postural abnormality correction. This review will first examine theoretical muscle-tendon unit shortening and lengthening mechanisms and the effectiveness of strength and stretch training. We will then share evidence-supported training, resulting in the prolonged correction of postural abnormalities by spinal region.

Muscle-Tendon Unit Shortening Mechanism

When considering the muscle and its tendinous connective tissue as 2 separate components of a unit, shortening would need to take place by different physiological mechanisms.

Muscle Component

When isolating the muscle component of the muscle-tendon unit and providing an explanation of how it could potentially shorten, it appears there are 2 options. The passive length of muscle could potentially shorten by decreasing fascicle length via a decrease in the number of sarcomeres in series or an increase in the angle of pennation in pennate muscles [8]. A study using cat soleus muscle that was immobilized in a shortened position found there was a 40% decrease in the number of sarcomeres in series within the muscle fascicles compared to normal muscle [9]. This adjustment in sarcomere number in series appears to be the way muscle adapts to its new functional length. Immobilizing muscle in a shortened position also reduced muscle extensibility due to either the decrease in sarcomeres in series or a buildup of connective tissue within the muscle [9]. A study with human muscle inquired about changes in muscle physiology resulting from muscle disuse. Patients with unilateral diaphyseal fractures of either the femur or tibia requiring immobilization demonstrated a significant decrease in the pennation angle and muscle fiber length of the vastus lateralis found using ultrasound [10].

Research using resistance training and its effect on muscle fascicle length was also reviewed. A study using healthy males found 6 weeks of free-weight resistance training at 80% of 1 repetition maximum with elbow extension movements increased muscle physiological cross-sectional area and strength but did not change fascicle length [6]. A cross-sectional observation study compared resistance trained to untrained individuals and found the fascicle lengths of their medial gastrocnemius muscle were not significantly different [7].

The evidence suggests a muscle that is shortened and immobilized, or experiences disuse will result in a decrease in muscle fiber length. In theory, shortening occurs with casted limbs as an adaptation to their new functional length.

The research does not support the theory of shortening muscle via decreasing the number of sarcomeres in series with a strengthening exercise protocol. Shortening may take place by increasing the pennation angle of a pennate muscle with resistance strength training [11]. This may, however, be irrelevant in most postural abnormality cases when the stabilizer musculature attached to the spine is predominately not of pennate architecture.

Tendon Component

To shorten tendons, analysis at the molecular level will be introduced for an understanding of potential options. Collagen molecules within tendons anatomically consist of tropocollagen and microfibrils, which are made of 5 tropocollagen [12]. A tropocollagen is composed of a triple helix of polypeptides bound by hydrogen bonds [12]. The potential for irreversible elongation through slipping is minimal, but the helix could uncoil through rupturing of the strong hydrogen bonds with high tensile overload, [13] but is unlikely. Microfibrils can undergo deformation through the slipping of the individual microfibrils on each other [12]. They are bound together by cross-links or covalent chemical bonds which appear to have the ability to detach with a low tensile load of long durations [12]. A study immobilizing

rabbit soleus muscle in a shortened state concluded many of the shortened length changes were caused by a decrease in tendon resting length [14]. The tendon component may shorten via detachment and reattachment of cross-links between collagen microfibrils as an adaptation to a static length with immobilization such as casting [14]. The physiological shortening of tendons with a form of exercise was not discovered in this review.

Muscle-Tendon Unit Lengthening Mechanism

As with shortening a muscle-tendon unit, the muscle and tendon can be examined as 2 separate components of a unit where lengthening would need to take place by different physiological means.

Muscle Component

Stretching exercise protocols have been administered to determine if this type of activity lengthens muscle-tendon units. Healthy individuals participated in a randomized controlled trial using a stretching protocol for the hamstring muscle that consisted of static stretching for 30 minutes, 5 times per week for 6 weeks to the experimental group and no stretching with the controls [15]. The findings suggested no difference occurred between groups when measuring for a difference from controls in extensibility (hip flexion with 18Nm of torque) [15]. An additional study using a static stretching protocol of the hamstrings for extensive durations of time (30 minutes each day for 4 weeks) was used on a population with paraplegia or quadriplegia to find if any changes would occur in extensibility at 30Nm of torque [16]. The results demonstrated no changes in extensibility occurred in patients with spinal cord injuries using an aggressive static stretching protocol [16].

Research inquiring a stretching program consisting of 2, 1-minute static stretches, daily, for 4 weeks was performed on human gastrocnemius muscles [17]. The findings of this protocol were an increase in range of motion but no changes in fascicle length. Conflicting findings occurred when static stretching of the soleus muscle of rats was conducted using a protocol of 1 minute, 10 times per session, 3 times per week for 3 weeks. The results from this animal study found an increase in sarcomeres in series [18].

Specific resistance training protocols were also used to inquire if muscle fiber lengthening occurs. Studies assessing a shift in torque-joint angle have been used to determine muscle length changes based on the optimal length-tension relationship [19]. For example, a shift of the torque-joint angle curve to the right would indicate an increase in muscle length to achieve optimal torque. Conversely, a shift of the torque-joint angle curve to the left would indicate a decrease in muscle length to achieve optimal torque. The adjusted length would occur secondary to an increase or decrease in the number of sarcomeres in series respectively. Several studies have suggested an increase in sarcomeres seems to occur with resistance training [19-21]. One such study asked participants to perform knee flexion resistance exercise using both concentric and eccentric muscle actions from 0^{o} - 30^{o} [19]. Training produced a right shift of the torque – joint angle curve suggesting resistance training at the end-range of joint motion may increase the length of the antagonist muscle by adding sarcomeres in series [19]. Another study used a Nordic curl eccentric strength training protocol for the hamstrings and found a similar shift of the torque – joint angle curve to the right immediately post exercise, again, indicating muscle fiber lengthening [20]. A third study inquired eccentrically training the quadriceps using a closed kinetic chain mode and discovered following 10 minutes of single-leg step downs there was a right shift of the torque – joint angle curve in the quadriceps muscle [21]. A study using ultrasound measures found the Nordic curl exercise created an increase in sarcomere length in the biceps femoris long head but questioned it being the result of additional sarcomeres in series. Instead, the findings suggested length changes occurred specific to the distal region of the biceps femoris long head, possibly due to changes in connective tissue or titin protein stiffness [22].

Increases in muscle-tendon unit length have been found with resistance exercise at joint end-range [19] and eccentric strengthening exercise [20-22]. It appears the muscle is adapting to provide a more optimal length-tension relationship which requires greater length that may be explained by an

addition of sarcomeres in series. Stretching exercise studies have not supported muscle–tendon unit length changes even when incorporating interventions that provide tension loads for durations far beyond traditional protocols [15, 16]. Hamstring muscle extensibility was not impacted with spinal cord patients who had no sensation in their legs, suggesting changes in range of motion secondary to stretching are not physiologically induced [16]. It has been suggested after extensive study that initially, stretching causes viscoelastic stress relaxation in muscle due to viscosity changes, primarily with the perimysium connective tissue, but this returns in approximately 1 hour [23]. After 3 weeks of stretching, an increase in range of motion is due to a stretch tolerance or adaptation to the muscle tension perception, while viscoelastic characteristics of the muscle remain unchanged [23]. There is evidence of stretching protocols using stretches of long durations resulting in additional sarcomeres in series in an animal study, but similar results were not found with human participants.

Tendon Component

The tendon component has been subjected to tensile loads in experiments to analyze its response. Human Achilles tendon under constant, cyclic loading resulted in significant tendinous damage [24]. Rabbit Achilles tendon, when exposed to low cyclic loads, adapted by going through a stem cell differentiation into tenocyte and increased strength while if high cyclic loads were administered the stem cells were directed into non-tenocytes or a breaking down of tendinous tissue [25]. During human growth, tendon collagen fibrils grow in length by tip-to-tip fusion until they become continuous [26]. A collagen fiber forced to lengthen beyond its critical length may undergo cracking, weakening its resistance to tensile load [26]. This evidence of tendon collagen maturation during growth suggests high tensile loads with mature tendon may be a risk factor for tendinopathy.

Lengthening of tendon may be an event that if attempted beyond maturation, where the collagen fibrils are fused at their tips, could inflict cracking of the collagen, and weaken the tendon suggesting they are structurally incapable of lengthening and remaining healthy [26].

Postural Correction Intervention

Exercises attempting to correct postural deviations are reviewed. We will present postural abnormality correction interventions for all 3 regions of the spine.

Cervical

There have been several attempts to correct a forward head-rounded shoulders postural abnormality. Patients with chronic neck pain were either assigned to a group that performed a craniocervical flexor exercise, emphasizing the deep neck flexors, or a head lift cervical muscle strengthening exercise that addresses more of the global neck flexor muscles [27]. The results demonstrated a significant increase in the craniovertebral angles with the craniocervical flexor exercise group suggesting a greater correction of the head forward posture [27]. A randomized controlled trial provided an experimental group with an exercise protocol suggesting stretching the "shortened" muscle (cervical extensors and pectoral muscles) and strengthening the "lengthened" muscles (deep cervical flexors and shoulder retractors) compared to a control group who were asked to continue normal activities [28]. The findings of the study were that both groups increased in craniovertebral angle [28].

Participants of another study had ≤ 49° craniovertebral angle for inclusion criteria and found that with an intervention of sustained natural apophyseal glide (SNAG) mobilizations, the craniovertebral angle increased significantly [29].

The forward head postural abnormality was influenced positively with exercises that were activating the deep cervical flexor muscles [27]. Interestingly, when a strengthening and stretching protocol addressing lengthened and shortened muscles respectively was compared to regular activity, they both improved the forward head posture equally [28]. This may be explained by the control group being cognizant of their posture because of the study's purpose and possibly self-correcting. The mobilization techniques also provided a positive impact on postural correction [29]. This type of manual technique may provide sensory stimulus facilitating neuromuscular adjustments resulting in mild postural changes.

Thoracic

Exercise interventions have also been used to correct hyper-kyphosis of the thoracic region. A study placed participants into 2 groups, 1 was asked to perform stretching exercises meant to emphasize the pectoral muscles and strengthening exercise for the back extensor muscles of the thoracic spine [30]. The other group was educated in self-correction training in addition to the stretch and strengthening exercises [30]. The findings suggested the addition of postural self-correction strategies were significant in increasing both the craniovertebral and kyphosis angles [30]. A randomized controlled trial placed postmenopausal women in either a group educated in postural self-correction with taping and joint mobilization strategies or a group on a wait list [31]. A significant increase in the angle of kyphosis was found in the experimental group, suggesting 1 or more of the interventions were effective [31]. Another study used participants who were placed either in a group receiving a specific therapeutic taping technique of the thoracic spine or a group receiving a sham taping and one with no taping [32]. The results demonstrated a significant increase in the angle of kyphosis with the therapeutic taping and not with the other 2 groups. A surface electromyography (EMG) was also performed in this study showing no changes in surface muscle activation in any of the 3 groups.

Self-correction strategies activate most muscles simultaneously which may improve sensory input and facilitate proprioception [30]. When attempting taping strategies [32], changes in posture are not likely the result of a rigid response from the tape. The improved postural results with therapeutic taping application is more likely secondary to changes in deep muscle activity secondary to sensory input. In much of the exercise interventions for scoliosis, a reduction in vertebral rotation occurred [33-36]. The neuromuscular training of local trunk musculature may re-educate the spinal muscles and improve stability against rotation encountered with a scoliotic abnormality. Research suggesting supervised training is optimal to unsupervised [35] suggests the need for feedback on self-correction, which could advocate for referring to a mirror for corrections when initiating independent strategies.

Lumbar

An attempt to correct scoliotic postural abnormalities has also been pursued with exercise. A study placing participants into 2 groups, 1 requiring them to perform stretching and strengthening exercises along with bracing and the other also performing the exercises but instead of bracing they included specific stabilization exercises emphasizing local muscle training of the trunk [33]. There were no significant changes in cobb angles but there was a reduction in rotation of the lumbar vertebrae in the trunk stabilization group [33]. The scientific exercise approach to scoliosis (SEAS) plan consists of active, self-correction exercises in all 3 planes to activate the intrinsic muscles of the spine and facilitate the central nervous system's ability to coordinate muscle actions of the spine [37]. A randomized control trial using adolescent idiopathic scoliosis participants compared a self-correction type protocol similar to the SEAS plan with a stretching-strengthening program and found a significant decrease in cobb angle and vertebral rotation with the SEAS type training group [34]. Another protocol developed specifically for scoliotic patients is the Schroth exercise protocol that consists of self-correction type exercises such as de-rotation and rotational breathing along with basic stretching and strengthening exercises [35]. A study including a group using Schroth exercises while being supervised was compared to a group attempting the Schroth exercises as a home program along with an inactive control group [35]. The findings suggested the group involved in supervised Schroth exercises experienced a significant decrease in cobb angle and vertebral rotation [35]. Another study compared a group required to perform Schroth and core stability type exercises with a group attempting general stretching and strengthening exercises with the same core stability exercises [36]. Although both groups experienced a decrease in cobb angle and vertebral rotation, the measured changes were greater with the Schroth group [36]. The ability to influence changes in lumbar lordosis have been investigated using primarily stretching exercise protocols. Research inquiring hamstring length and its possible impact on lumbar lordosis has been frequently investigated. A randomized controlled trial required 1 group to stretch their hamstrings in sitting and standing positions

using a protocol of 2 times 30 seconds, 4 times per week for 4 weeks and a control group absent from stretching [38]. The results suggested the group performing the stretches acquired a significant change in hamstring length determined by increased hip range of motion but neither group demonstrated a change in lumbar lordosis using an electronic goniometer [38]. Another study had the participants using 4 different stretching exercises with a protocol consisting of 3 sets of 20 seconds for each exercise and a 30 second rest between with measurements taken immediately post-stretching [39]. The results demonstrated an increase in posterior pelvic tilt with maximum trunk flexion with the knees extended determined by a spinal mouse system [39]. An additional study separated elite paddlers into 3 groups of varying hamstring extensibility determined by measuring hip flexion during a passive straight leg raise [40]. The results demonstrated the paddlers with low extensibility had an increase in posterior pelvic tilt in maximum trunk flexion with the knees extended but there was no association between hamstring extensibility and lumbar angle in any postural position [40]. The impact of the abdominal muscles on lumbar curvature was explored when measuring the abdominal strength of patients with chronic low back pain using a leg lowering test [41]. There was no association found between strength and lumbar lordosis or pelvic inclination in standing [41]. Psoas muscle studies and its impact on lumbar lordosis are limited. Research investigating the cross-sectional area (CSA) of the psoas muscle in sprinters using magnetic resonance imaging (MRI) found no correlation with the CSA and lumbar lordosis [42]. When considering the psoas muscle and its role with the lumbar spine, fine wire EMG results showed the psoas major attachments to the transverse processes provide greater activation with active trunk extension movement [43] and isometric effort in extension [44] while more vertebral attachment activation with hip flexion movement [43] and isometric trunk flexion [44]. It has also been suggested that when considering force vectors, the psoas attachments at L1-2 and L3-4 provide a posterior force, L3-4 and L4-5 attachments a more in-line with the vertebrae force, and L5 – S1 provide an anterior force suggesting a role in lumbar segmental stability [45]. A study with adolescent idiopathic scoliosis participants were placed in one of 2 groups [46]. One group participated in a general stretching and strengthening program and the other also did the general exercise program but added craniocervical flexor exercises [46]. The group that added the craniocervical flexor exercise resulted in acquiring an increased craniovertebral and kyphosis angles, and a decrease in lumbar curvature [46]. This evidence suggests deep cervical flexor muscle activation may have an impact on the central nervous system resulting in postural neuromuscular activity and ultimately repositioning throughout the spine.

Conservative interventions to adjust for lordodic abnormalities come with little success. Several hamstring stretching protocols did not have an impact on lumbar lordosis of any significance, other than when the trunk is fully flexed and the knees fully extended [38, 40]. Abdominal strength was also not found to influence lumbar curvature [41]. A study investigating different CSAs of psoas muscles in sprinters did not find differences in lordosis of the lumbar spine [42]. This would suggest that possible differences in static tension and external forces facilitating increased lordosis does not have an impact on postural changes. When analyzing fine wire EMG results and force vector studies, it appears the primary role of the psoas muscle is to provide lumbar segmental control and lumbar stability [43-45]. An interesting finding with deep cervical flexor training is its impact globally on spinal posture, supporting this type of training for postural abnormalities throughout the spine [46].

Conclusion

In summary, postural abnormalities should not be ignored as they may lead to somatic dysfunction and pain. The traditional clinical approach consisting of an attempt to shorten or lengthen a muscle-tendon unit to correct postural abnormalities may be futile, suggesting a re-direction of evidence-supported goals. An alternative suggestion is an emphasis on local spine muscle activation to inhibit involuntary vertebral rotation in addition to providing vertebral stability and protection from shear forces. Also, postural awareness using self-correction and feedback strategies to maintain an optimal or neutral posture with

activities could facilitate an adaptation of the sensorimotor control system through a coordination of inhibition and facilitation of stabilizing musculature. Further, there is evidence suggesting activation of deep flexor cervical muscles may not only positively impact a forward head posture but also facilitate the correction of postural deviations throughout the spine.

Conflicts of Interest

Declare conflicts of interest or state "The authors declare no conflict of interest." Authors must identify and declare any personal circumstances or interest that may be perceived as inappropriately influencing the representation or interpretation of reported research results. Any role of the funding sponsors in the design of the study; in the collection, analyses or interpretation of data; in the writing of the manuscript, or in the decision to publish the results must be declared in this section. If there is no role, please state "The founding sponsors had no role in the design of the study; in the collection, analyses, or interpretation of data; in the writing of the manuscript, and in the decision to publish the results".

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