Critical and Theoretical Perspective on Scapular Stabilization: What Does It Really Mean, and Are We on the Right Track?

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Stabilization exercises have been a focus and mainstay of many therapeutic and performance training programs in the past decade. Whether the focus is core stabilization for the spine or scapular stabilization, clinicians and trainers alike have endorsed these programs, largely on the basis of conceptual theory and anecdotal experience. The notion that an unstable scapula is related to shoulder dysfunction and pathology is well accepted, but is it accurate? The aim of this perspective article is to challenge the concept of scapular stabilization through the application of biomechanical and motor control constructs. The objectives are to critically examine current beliefs about scapular stabilization, to discuss definitions of stabilization and stability in the context of the scapulothoracic region, and to evaluate key evidence regarding scapular stabilization and scapular dyskinesia. Several new approaches that may affect the understanding of normal and atypical scapula motion are explored. Finally, a historical analogy is presented and future research and clinical directions are suggested. The aims are to lead readers to the essential concepts implied on scapular stabilization, to increase the critical thought process in rehabilitation practice, and to suggest some open topics to be explored in future research.

The notion that an unstable scapula is related to shoulder dysfunction and pathology is well accepted.[1,2] The prevailing theory asserts that for proper function of the glenohumeral joint, the scapula must provide a stable base upon which upper extremity tasks are accomplished.[2–5] Without scapular stability, in theory, there is increased risk for pathologies such as impingement or cuff tears.[1] Several implications are strongly linked to the idea of scapular stability, including the belief that dyskinesia[2] is a sign of instability and is a result of weak or unbalanced scapulothoracic (ST) muscles.[4–11] Laboratory and clinical approaches based on these implications include the classification of dyskinesia and the use of stabilization exercises, braces, or taping to increase scapular stability.[4,7,9,12]

The importance of scapular stabilization has drawn considerable attention from research scientists, educators, and clinicians. However, to our knowledge, there has been no critical evaluation of the available literature with regard to whether scapular stabilization is a solid paradigm on which to build. In this perspective article, we explore the mechanistic theory underlying the approach to scapular stabilization and the evidence of training efforts to improve scapular stabilization.

Describing Scapular Position and Movement

In the resting posture, the scapula sits on the thorax at a 30- to 40-degree angle anterior to the coronal plane. The anterior/posterior tilt of the scapula is variable and has been reported to be 10 to 13 degrees.[1,13] The upward rotation of the scapula at rest has been reported to be 4 to 10.5 degrees.[1,13] The position at rest can be influenced by the functional use of the upper limbs or by habitual postures. For example, in pitchers, the scapula on the side of the dominant upper limb has been reported to be more upward and internally rotated and to have greater anterior tilt than that on the nondominant side.[14]

Studies of scapula motion in groups of people who are healthy have defined a pattern of progressive upward rotation, posterior tilt, and highly variable internal/external rotation[1,15,16] during arm elevation in the coronal, sagittal, or scapular plane. A comparison of the motion of dominant and nondominant arms showed that upward rotation and internal rotation of the scapula were equivalent during raising of the arm and lowering of the arm in the sagittal, frontal, and scapular planes of arm motion.[13] However, posterior tilt was significantly decreased in the nondominant shoulder during coronal-plane abduction.[13] Importantly, scapula orientation recorded during these constrained planar motions was noted to be different from scapula orientation recorded during functional tasks. Amasay and Karduna[17] compared scapula orientations achieved at the same planes and elevation angles during both constrained and functional movements and reported angular differences ranging from 3.2 to 9.7 degrees between movement conditions. Therefore, any association between shoulder pathology or dyskinesia and scapula orientation in studies with constrained movement patterns may not translate directly to functional tasks.

Similarly, there is considerable variability in the description of scapulohumeral rhythm. In the 1940s, Inman et al proposed the 2:1 ratio idea—in which, for each 2 degrees of humeral elevation, there is a corresponding 1 degree of scapula upward rotation.[18] More recent studies have shown that scapulohumeral rhythm ranges from 1:1 to 6:1, depending on factors such as how kinematics are measured, the plane of elevation of the arm,[1,19] and whether there is an external load.[20,21] Other factors that influence scapulohumeral rhythm include speed of motion,[22] pain,[23] shoulder tightness,[24] and fatigue.[13,15,25,26] In addition, accurate measurement of scapular kinematics is challenged by dependence on the precise notation of movement conditions, coordinate systems, and other technical 3-dimensional kinematic methodological issues.[27] In clinical settings, the observation of "abnormal" motion should be described but not assumed to represent an unstable scapula because, as we discuss below, the observed abnormal motion may simply represent normal kinematic variability.

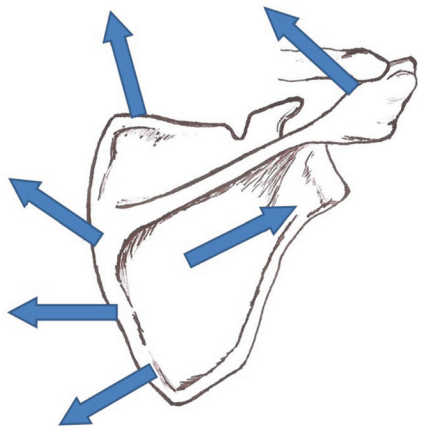

Figure.
Illustration of a system with multiple forces of different magnitudes and directions.

What Does "Scapular Stabilization" Mean?

The fundamental definition of stability is the degree to which a system can return to an orientation or movement trajectory after a perturbation.[28] An important concept embedded in this definition is that a system is either stable or unstable; a system cannot be partly stable. Joint instability is characterized by mobility that exceeds physiological limits without adequate control. When applied to the ST joint, this characterization suggests that the orientation or trajectory of the articulation would not be recoverable after a perturbation. Because this foundational definition is difficult to apply directly to the scapula, the term "scapular stability" has come to imply "normal" scapula movement on the thorax during upper extremity motions. What becomes obvious is that this clinical definition is not objective or quantifiable, presenting a major challenge for the discussion, evaluation, and study of scapular stability.

Another misleading belief is that muscle force balance plays a role in creating dynamic scapular stability or equilibrium. For dynamic stability to be achieved, synergistic muscles are not required to be balanced in terms of their activation or force generated. The idea of multiple competing forces in a system is illustrated in the Figure. The upper tra-

pezius (UT) muscle force is not equalized or balanced with the lower trapezius (LT) and serratus anterior (SA) muscle forces in producing an upward rotation moment on the scapula. Rather, all that is required is that the net moment satisfies the Newtonian condition for dynamic equilibrium (from Newton's second law of motion). This condition represents a state of dynamic rotational equilibrium or stability. Therefore, equal muscle forces are not mandatory—and could be clinically undesirable—because the muscles have different moment arms and thus different mechanical advantages for causing angular rotation in the joint (Figure).

The erroneous notion about expected muscle force balance often underlies the misinterpretation of scapular muscle electromyographic (EMG) studies—namely, that specific exercises must be initiated in selected muscles because of lower EMG responses.[6] Among other reasons for this misinterpretation, if a muscle at a particular degree of motion has a mechanical advantage because of its moment arm, then its motor unit activation requirements will be low even though it is capable of generating significant torque on the scapula. Confounding the issue is the fact that because the scapula both translates and rotates in 3 dimensions, its instantaneous axis of rotation is constantly changing, affecting the moment that each scapular muscle can generate to rotate the scapula.

The key point is that stability is context specific, depending on the system and the task being performed. The same argument was made by Cholewicki and McGill[29] and McGill et al[30] with regard to spinal stability; they argued that no single muscle played a dominant role in the process and that the contribution of individual stability spring-like guide wires constantly changed, depending on the task.[29,30]

Scapular Stability Paradigm

One of our main purposes is to challenge the notion of scapular stability or stabilization. A key part of the challenge consists of confronting the language used to describe scapula function, along with the implicit assumptions made when that language is used. The perspective offered here is that the current paradigm of scapular stabilization is flawed.

One obvious limitation of this paradigm, already noted, is that the scapula does not conform to the definition of stability or joint stability. Although the scapula has considerable movement capability, in the normally functioning neuromuscular system, it does not move beyond its physiological limits. The scapula returns to static or dynamic equilibrium after perturbations from unexpected external forces, internal forces, or voluntary movements, with the possible exception of high-force trauma. Although this limitation could be dismissed as simply a semantic argument, we propose that the implications and assumptions associated with the term "scapular stability" influence the approach to patient care in that clarity versus ambiguity matters. These implications include the notions that normal scapula motion is critical to shoulder and upper extremity function; that dyskinesia is a sign of muscle weakness, instability, or lack of motor control; and that stabilization exercises will resolve symptoms and improve scapula motion.

A second key limitation of the scapular stability paradigm relates to the less stringent definition of scapular stability proposed earlier. The necessary antithesis of the definition that normal scapula motion indicates system stability is that scapula movement that is not normal (dyskinesia) indicates instability. Clinicians tend to use dyskinesia as the index test for stability and to initiate scapular stabilization exercises when dyskinesia is present.[5] Importantly, this connection between nonnormal movement and instability has not been established, and many people with scapular dyskinesia maintain healthy functional use of the extremity.

Admittedly, the argument that there is no connection between dyskinesia and instability is problematic because there is no established way to measure scapular stability. Without a method for quantifying stability, the ability to test for a relationship with dyskinesia is not currently possible. That said, the absence of an appropriate stability test supports the argument that the term "scapular stability" is vulnerable to being used indiscriminately.

Rethinking the Stable Base Function of the Scapula

For the scapula to manage perturbations and maintain equilibrium, it must be able to transfer forces. But how does this happen? The scapula has minimum geometric and anatomical constraints, sits on the thorax, and is suspended primarily by the musculotendinous attachment of 17 muscles in addition to some load transfer via the clavicle. Some of these muscles, such as the trapezius, levator scapulae, rhomboid, and SA muscles, are axioscapular muscles and are considered to be prime movers or pivots of the ST articulation.[3] These axioscapular muscles are the ones most targeted for rehabilitation to improve scapular stability.[6,31–33] Although there is some passive suspension support for the scapula via the acromioclavicular joint ligaments and the coracoclavicular ligaments, the minimum geometric and anatomical constraints of the scapula necessitate that forces generated in the arm must be transferred to the axial skeleton primarily through the musculotendinous attachments. Likewise, proximally generated forces needed for arm function must be transferred distally through these same musculotendinous attachments. Although the ligamentous connections do assist in maintaining acromioclavicular joint congruency and force transfer between the scapula and the clavicle, synergistic ST muscle activation is critical for force transfer between the arm and the trunk.

Given that there is no geometric base for transferring loads from the upper extremity, an appealing model for load transfer suggests that the scapula functions as the hub of a "tensegrity" structure[34,35]; in this scenario, forces coming from the arm are transferred to the axial skeleton through the soft tissues rather than through the linked bone levers. As in the design of a bicycle wheel, the scapula, suspended in the "spokes" of the attached muscles and soft tissues, could function as a hub for the arm and the thorax. In addition, the concept that

the scapula, supported within a musculotendinous sling, could transfer forces from proximal to distal or distal to proximal also is plausible. In this scenario, the stiffness of the sling elements would dictate the relative ability of the system to absorb or transfer kinetic energy.

On the basis of the preceding discussion, it may be more accurate to conceptualize ST function as an energy transfer system rather than an anatomical structural base of support. In this concept, the role of the scapula is not so much to provide a stable base but to maximize the overall degrees of freedom needed to place the hand in space and to absorb and transfer energy to and from the upper extremity. In particular, Hasan[36] suggested that stability in the sense of quick resistance to perturbation often may not be necessary for the successful control of forces, energy, and movement. Instead, he proposed that movement variability creates resilience, which is more desirable than stability for the control of movement. Similarly, "robustness" may be a more appropriate term than "stability" because it describes the tolerance of a system for uncertainty, allowing for degrees of movement variability. For the scapula, catching a ball with uncertain trajectory and velocity perturbs the shoulder complex as forces are transferred to it from the upper extremity. How well the ST system tolerates that perturbation defines its robustness, and the ability to recover to the preperturbed state defines its stability.

Is Dyskinesia an Indicator of Instability?

There are several arguments against considering dyskinesia to be an indicator of instability. First, we propose that most observed scapular dyskinesia likely represents normal movement variability. In walking, many movement patterns are considered to be normal, and a single person's gait pattern is variable enough to be unique and recognizable. There seems to be much less acceptance of such individual variability at the ST joint, with all nonnormal movement being identified as problematic. Similarly, it is accepted that normal gait patterns result from the combined motions of all of the lower extremity joints, the pelvis, and the trunk. However, for upper extremity function, ST movement is often emphasized, with less regard for the comprehensive nature of the whole system.

The traditional orthopedic biomechanical model has assumed that variability is evidence of incorrect movement patterns. However, a dynamic systems approach may provide a better framework for understanding scapula movement. Dynamic systems theory[37] argues that variability reflects the variety of coordination patterns used to complete a task and suggests that variability is evidence of the flexibility and adaptability of the neuromuscular system in exploring new movement solutions. Essentially, dynamic systems theory considers coupled movement patterns and their phase relationships. In this context, dynamic stability is characterized by the stability of coupled movement patterns—such as phasing or coupling of scapular posterior tilt with other segmental motions, such as humeral external rotation—rather than any one position or angular magnitude—such as the degrees of scapular tilt or rotation.

Second, a theoretical connection among dyskinesia, scapular instability, and shoulder pathology has become widely accepted without clear evidence that such a connection exists. The concern is that the presence of dyskinesia may lead to quick conclusions about pathological mechanisms and interventions that stifle clinical reasoning and decision making. The notion that scapular dyskinesia is an indicator of instability and is linked to pathology is not supported by recent independent literature reviews. Thomas et al[38] performed a meta-analysis of 9 studies comparing scapular kinematics in people with and people without subacromial impingement. They reported a small overall effect size for decreased scapula upward rotation and increased internal rotation in people with subacromial impingement but acknowledged that the data were influenced by the inclusion of athletes and workers who used their arms in overhead positions in the review. As noted earlier, repetitive use may alter scapula position; therefore, because of the inclusion of these groups, the analysis may not have been applicable to the general population. Ratcliffe et al[39] performed a systematic review of 10 studies evaluating the link between scapula kinematics and subacromial impingement. They concluded that no ideal scapula position exists and that deviations in scapula motion do not cause or contribute to subacromial impingement.

Furthermore, Ludewig and Reynolds noted substantial evidence for scapula kinematic alterations in people with impingement, yet the type and prevalence of alterations were inconsistent, with a mix of both "negative" and "positive" alterations being reported.[1] For example, of the 23 between-group movement comparisons, only 13 (57%) were significantly different, with 43% showing negative scapula movement in people with impingement and the other 14% showing positive movement.

Overall, these reviews do not support a consistent connection between scapular kinematic changes (dyskinesia) and shoulder impingement, suggesting that dyskinesia could develop in response to pain rather than cause it, that measurements or definitions of stability currently in use are not sensitive enough to clarify an existing relationship, or that a high level of scapula motion variability may be the norm.[40]

Third, normal scapula motion—if even possible to define—is based on group data that tend to encompass the movement variability of an individual. In other words, scapular kinematic data for people who are healthy includes ranges of motion that most likely encompass those of people with nonnormal motion patterns for at least one scapula rotation. Although this dyskinesia becomes lost in the group average, if observed in an individual, it is interpreted as problematic. This point becomes apparent in a study comparing visual dyskinesia in people with and people without impingement; in that study, equal percentages of people in the 2 groups displayed visible dyskinesia.[11]

Fourth, dyskinesia and lack of stability have been said to reflect poor motor control of the scapula stabilizers,[4] but, as we

discuss below, evidence is far from conclusive.

Applying Motor Control Theory to Scapula Control

Before the evidence is evaluated, the parameters within which motor control theory operates should be defined. One parameter is that neuromuscular control is synergistic, which means that control elements such as muscle forces vary considerably in response to perceptual information. For example, errors in force generation in one muscle element are automatically compensated for by other muscle elements to perform a particular task.[41] These compensations are reflexive and do not occur within any voluntary time frame. In the context of synergistic motor control, scapula movement variability becomes a necessary and advantageous strategy for movement.

Another parameter is that the motor system has built-in redundancy for motor degrees of freedom. Especially in the upper extremity, the motor system has many combinations of actions and movements available to achieve a given goal. Therefore, it is possible that an exact scapular orientation is associated with multiple ways in which a given state can be achieved and multiple ways in which muscular kinetics can vary. This notion of redundancy has been referred to as the "degrees of freedom problem," as outlined by Bongaardt and Meijer.[41] Movements can have different trajectories, velocities, and muscle activation profiles to achieve the same end result. In this regard, the notion of presuming normal motor control of the scapula as a reflexive link within a kinematic chain is a weak axiom.

With regard to scapular function, a construct similar to redundancy is the idea of optimization* of movement-related criteria.[42–44] For human motion, the question to ask is, "What needs to be optimized?" In some cases, it may be energy expenditure, and in other cases, it may be energy transfer or absorption. An emphasis on scapular stability may not be a functionally relevant optimization strategy of the motor system.

In general, the higher-order structures within the nervous system have a greater association with movement synergy than with individual muscle control. This is the principal rationale for why attempts at isolated muscle contraction exercises, such as vastus medialis obliquus muscle exercises for patellofemoral pain syndrome, multifidus spinae and transversus abdominis muscle exercises for low back pain, and any isolated scapular muscle exercises, have been associated with a lack of strong biologic plausibility. This idea is not without exceptions, however. Holtermann and colleagues[45,46] showed that it is possible to use intense EMG biofeedback training to selectively activate the UT, LT, and SA muscles. However, these observations have not been shown to transfer to any real-life functional task performance, making the practical relevance to scapula control or stabilization unclear.

Studies evaluating scapular motor control have involved comparisons of ST muscle activation between groups or after a training intervention. A frequent assessment of motor control involves comparing EMG activation levels in the ST muscles, with lower UT and higher SA and LT muscle activation levels being considered optimal. Larsen et al[47] investigated "neuromuscular control of scapular muscles" in patients with subacromial impingement syndrome (SIS) and a matched control group and found no differences in muscle activation onset or SA/UT and UT/LT muscle amplitude ratios between the groups. Worsley et al[48] compared the effects of a 10-week "motor control training program" that consisted of learning optimal scapular orientation at rest and during active muscle-specific exercises for the LT and SA muscles in a group of patients with impingement syndrome (n=16) and a control group of people who were healthy (n=16). Electromyographic timing deficits and improved scapular posterior tilt in the patient group suggested that the motor control training program was effective. However, EMG timing relied only on visual inspection; data from the control group, which also may have changed in an equivalent way, were not collected at 10 weeks; the motor control training program included other interventions commonly used in clinical practice to manage symptoms; and the only observed scapular kinematic change involved posterior tilt, which may be affected by simple improvements in posture.

Roy and Iqbal[44] assessed the effects of motor control and strengthening exercises on shoulder function in a case series of 8 people with SIS and reported reduced pain and improved function; however, they did not assess scapula muscle strength or activation. Conscious correction of scapula orientation during 4 different exercises designed to properly activate the UT, LT, and middle trapezius (MT) muscles did not increase favorable UT/MT and UT/LT muscle activation ratios in athletes who used their arms in overhead positions and had dyskinesia.[12] However, visual biofeedback increased the amount of posterior tilt and decreased UT/MT, UT/LT, and UT/SA muscle activation ratios during scapular stabilization exercises in people with SIS.[49] Similarly, visual EMG feedback resulted in the ability to selectively activate the LT muscles in approximately 50% of people with and without SIS,[49] and visual feedback of the scapula position with real-time video was shown to increase UT and SA muscle activation during arm elevation in people with scapula winging.[50]

Although some of these studies suggested that scapular stabilization exercises influence ST muscle activation, it is not known whether increases in ST muscle activation or changes in activation ratios translate to any lasting kinematic pattern improvements. In total, there is little evidence to suggest that scapula motor control training can functionally affect scapula muscle activation. Learning to consciously control scapula position and using visual biofeedback appear to be good methods for immediately altering ST muscle activation or motion, yet the long-term clinical significance and transferability to daily functional tasks remain unknown. Whether these strategies conform to the previously

* In biomechanics, "optimization" is a term used to imply that there is some parameter, such as minimum energy expenditure or maximum force, that the body tries to optimize for the most efficient function.

described ideas of synergy, redundancy, optimization, and individual muscle control should also be considered.

Finally, with regard to dyskinesia and implied instability, exercises to improve scapula control or strength do not appear to normalize scapula motion. The general idea is that if scapular muscles are appropriately strengthened, then observed abnormal kinematics or dyskinesia can be corrected, leading to improved scapular stabilization. Only one clinical trial favored the addition of "scapular stabilization exercises" for patients with SIS[51]; significant increases in scapular muscle strength and joint position sense were reported for a scapular stabilization group (n=20), relative to a traditional shoulder exercise group (n=20), after 6 weeks of training. Unfortunately, a static measure of scapula position was used as an outcome measure.

Several studies have involved the evaluation of athletes or workers who use their arms in overhead positions because of their increased prevalence for scapular dyskinesia and shoulder pathology. In one assessment, a 6-week scapular stabilization exercise intervention did not alter dynamic 3-dimensional scapula kinematics or ST muscle strength in swimmers.[52] In another study, a 6-week intervention involving 4 ST exercises previously determined to "optimize" muscle activation levels was evaluated.[53] After the intervention, athletes who used their arms in overhead positions and had SIS had lower UT/SA muscle activation ratios during arm elevation in a standing position.[54] In a subsequent study, after participants were given instructions on how to consciously control scapula position, 2 of the 4 exercises (side-lying external rotation and prone extension) resulted in increased UT, MT, and LT muscle activation ratios but no change in ST muscle activation ratios.[54] In a similar study of people who worked in offices and had neck pain and scapular dyskinesia, maintaining a learned correct scapula position resulted in greater LT muscle activation during typing.[55] Two other studies failed to show any change in scapular 3-dimensional kinematics after a 4-week[10] or a 6-week[56] scapular training program. Neither of these studies included quantification of scapular stability or reporting of the presence of scapular dyskinesia in participants before the interventions, undermining the ability to evaluate how the interventions may have influenced ST stability. In summary, it does not appear that exercise programs focused on correcting scapula movement are consistently supportive. Many studies reporting changes in scapular dyskinesia have been hindered by equivocal reliability of the measurement of scapular dyskinesia[9,12] and the fact that scapular dyskinesia is a poor predictor of shoulder pain.[10]

Where Is the Evidence Leading Us, and on What Should We Focus?

This perspective article is meant to challenge thinking and clarify concepts related to scapula dysfunction and therapy. Although there is not yet sufficient evidence to support a new approach to treating shoulder pathology, on the basis of the principles discussed earlier, we can suggest a new approach for therapists and some new research directions.

Because the ST joint is located proximally, the muscles that control movement are innervated by the ventromedial descending motor control system.[57] This descending system has limited potential for precise voluntary control of the individual muscles it recruits and instead organizes activation into more global synergy. This information suggests that exercises intended to recruit and strengthen individual muscles are not likely to be effective and that global recruitment of ST muscles should be emphasized. If this approach is coupled with the idea that manipulating length/force relationships can enhance muscle stiffness, then perhaps facilitating muscle recruitment with the scapula away from its "ideal" position may be most effective. This notion is contrary to the current paradigm, which suggests that people must learn and maintain the ideal neutral scapula position before beginning any exercises.

From a motor control perspective, the position of the scapula and its equilibrium very likely are low priorities for the central nervous system. Instead, movement planning and execution are focused on the hand and how it will interact with the environment to complete a specific task. The scapula, in all likelihood, is subservient to the hand for most tasks. For example, several studies have evaluated the effect of performing scapular stabilization exercises on an unstable surface, hypothesizing that this approach will increase ST muscle activation.[58–61] This strategy does not increase ST muscle activation, suggesting that stiffness at the interface between the surface and the hand is the primary emphasis for these tasks.

For evaluation, assessing scapula motion remains important, but dyskinesia should not be automatically regarded as impairment; dissimilar scapula motion patterns bilaterally could suggest normal variability rather than impairment. It is known that holding a weight during arm elevation often elicits dyskinesia, but the decision to define this dyskinesia as problematic should be based on the required accuracy of the functional task, not the specific scapular orientation. In other words, the focus needs to change from trying to determine precise scapula movement deviations that lead to isolated interventions, such as stretching the pectoralis minor muscle for lack of posterior tilt or strengthening the lower trapezius muscle, with the notion that such interventions will improve scapula upward rotation.

Interventions that require the shoulder complex to function as a force absorption and transfer unit and muscle activation in functionally relevant positions should be emphasized. Functionally relevant training should be data driven, criterion based, and patient specific to meet particular activity demands. Using various levels of resistance, speed, or both can be considered to challenge the robustness of the system to perform tasks. Adding perturbations to any of these activities also will train the system to adequately respond to external forces (improving stability). Approaches that incorporate plyometric training may facilitate stiffness adaptations via the stretch reflex.

Ultimately, this perspective article does not advocate for a radical change in the treatment approach for the ST articulation as much as it challenges how clinicians think about, rationalize, and justify the approach that is used. For example, rather than attempting to change scapula posterior tilt by 5 degrees, clinicians should work to ensure that the scapula has the maximum potential for movement (by stretching tight tissues or improving thoracic mobility), the maximum capability of movement (through global muscle activation and strength), and the maximum ability to assist the hand in performing precise functional tasks. Scapula movement variability should be embraced as a central nervous system optimization strategy rather than as a pathological factor, and variability should be considered in terms of coupled movement patterns rather than absolute singular scapular positions or angles. An emphasis on managing perturbations through the manipulation of external loads, scapula position, or movement velocity to ensure robustness should be an underlying construct during treatment. This approach represents a shift from a scapula-based movement paradigm to a muscle-based functional paradigm. Although clinical metrics for the assessment of stiffness control are not currently available, it is possible to develop stability assessments that are not just kinematically defined (dyskinesia) but are performance defined. For example, new assessments could be devised to require maintaining the arm in a functional position and testing the ability of the arm to reestablish the position (with time and endpoint location indicators) after unanticipated perturbations.

For researchers, direct application of dynamic systems theory and muscle synergy decomposition analytical techniques may provide better insight into normal and abnormal coupling and neuromuscular coordination. Direct scapular stiffness experiments to define stiffness behavior under various conditions could provide needed outcome measures to evaluate interventions that propose to change neuromuscular control through the modification of muscular stiffness. When a change in motor control is desired, the primary control parameter to target is muscle stiffness, yet the ability to change ST muscle stiffness has not been demonstrated and remains an important construct to explore. Simple experiments to evaluate passive and active force/displacement or torque/angular displacement behavior could provide baseline metrics for interventions that purport to alter ST muscle stiffness behavior.

In conclusion, this perspective article suggests that the current clinical scapular stabilization paradigm is ambiguous, is flawed, and has limited support from current evidence. The notion that there is an ideal scapula orientation or that isolated ST muscle strengthening will be effective for people with dyskinesia is also unsupported. Alternatively, ideas about muscle synergy and stiffness, motor redundancy, and coupled movement pattern variability should be at the forefront of considerations of the contribution of the scapula to shoulder movement and pathology. Finally, the metrics of scapular stability should de-emphasize the scapula and be defined by the ability to return to or recover an orientation or to resume a movement trajectory of the entire upper extremity.

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