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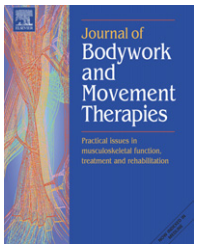

The presence of physiological stress shielding in the degenerative cycle of musculoskeletal disorders

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Summary The health of physiological tissue is governed by the continuous conversion of mechanical stimulus (stress) to bio-chemical response, a concept known as mechanical homeostasis. If this regulatory imperative becomes flawed, it may be detrimental, and consequently invoke or encourage the progression of various musculoskeletal disorders. This notion is corroborated by the quantification of altered function and irregular mechanical properties found within the articulations of such phenotypes as cerebral palsy. Although the divergence from healthy to irregular tissue behavior is likely multifactorial, the presence of imbalanced mechanical properties may promote the concept of physiological stress shielding. Extrapolating upon the stress shielding phenomenon may allow inferences to be drawn with respect to the pathomechanisms of progressive disorders. Further, recognition of this association may also provide a new platform from which to interpret the impact of conventional treatments aimed at such syndromes and, in turn, perhaps support new therapeutic avenues.

Musculoskeletal stability

Joint stability is perceived as a balance of forces stemming from a variety of tissues. Specifically, the dynamic equilibrium of joints is achieved through muscular and ligament tension, joint capsule flexion, cartilage rigidity, and bony structure reaction forces driven by gravitational forces. The health of these physiological tissues is essential for the maintenance of proper joint function and integrity. Conventional interpretation of joint dynamics is performed under several assumptions and optimization procedures that may have considerable influence on numerical interpretations (Pierce and Li, 2005). These admitted simplifications filter the redundancy caused by the immense number of unknown variables involved within their static or dynamic interpretations. Nevertheless, equilibrium analyses of joint articulations achieve reasonable agreement with electromyography (EMG) readings for healthy individuals predicted during gait (Rohrle et al., 1984).

Although detecting muscular activity through EMG has advanced our knowledge of the various activation strategies adopted during gait and other activities, it does not provide empirical interpretation of loads passively sustained within implicated connective tissues such as fascia (considered as

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ligaments, aponeuroses, joint capsules, endo-, peri-, and epi-mysium throughout this manuscript). Additionally, EMG interpretation does not detect resting muscle tone — an important form of intrinsic stress (Masi and Hannon, 2009). Interestingly enough, these commonly undetected and overlooked loads passively sustained in fascia may be important and acknowledging their role in joint dynamics may explain inconsistencies that arise when evaluating joint mechanics utilizing classical methods. This notion was demonstrated when the inclusion of fascial layers in spinal stability models rectified this previously inconsistent and indeterminate system and provided results that corroborated with physiologic measures (Gracovetsky, 2008).

Notwithstanding such revelations, the majority of conventional treatments of musculoskeletal disorders focus on muscle and bone biological and morphological adaptations — perhaps resulting from the popular use of these simplified measuring tools and biomechanical interpretations. Further, acknowledgment of such fascial loads may provide a foundation upon which to recognize several advantages put forth by rehabilitative and massage therapies that target these tissues. This recognition may also provide a more in-depth understanding of the influence of current treatments and provide insight into new innovated rehabilitative techniques.

The role of fascia in musculoskeletal stability

Although recently receiving greater interest, a tissue previously neglected that accentuates joint stability is fascia: a connective tissue that surrounds and penetrates muscles tissue; and is present throughout our entire body. Perhaps the initial documentation of its functional importance occurred in the 1940's when fascia was referred to as an ectoskeleton by Wood Jones who hypothesized the greater prominence of fascial tissues present in the lower body, compared to the upper body, results from significant greater dynamic stability demands in the lower regions (i.e. supported locomotion) (Wood Jones, 1944). This observation lends itself to the inference that the demanding environment of our locomotive musculature requires additional stabilizers — more than previously believed. Such theories where followed up and expanded by several authors who notably include works by (Rolf, 1977) who proposed manual therapy treatment avenues aimed at addressing the specifics of this structurally involve connective tissue.

With respect to stability, it has been shown that the health or tensional properties of this membrane partially regulates the contractual ability of the encapsulated muscle (Street, 1983; Huijing, 1999). Further, Huijing et al. experimentally showed (Huijing et al., 1998; Huijing, 1999; Huijing et al., 1999), and supported via simplified finite element analysis (Yucesoy et al., 2001), that the fascial membrane may allow for inter and intra muscular force transmission between adjacent or synergistic muscles. This would otherwise not be possible via conventional interpretations, since it isolates muscle behavior. In recent works, it has been speculated that epimuscular myofascial force transmission may occur in antagonistic muscles (Huijing, 2007; Yucesoy et al., 2008). Although experimental limitations of such studies (i.e. *ex-vivo* and non-physiological forces) must be noted, these novel findings are believed to hold true in daily activity. Using *in-vivo* cat models, it was demonstrated that removal of crural-fascias attachment reduces biceps-femoris torque by up to 50% (Carrasco and English, 1999). Also, passive elasticity in the femur tibia joint of locusts, in combination with viscous damping, has been shown to be strong enough to realign flexed articulation without conscious stimulation (Zakotnik et al., 2006). This passive force is likely generated by pre-stresses invoked on involved fascial connective tissues in addition to resting muscle tone. With regards to humans, over several notable communications and recently summarized, Gracovetsky demonstrated the distinct involvement of lumbodorsal fascia in spine biomechanics (Gracovetsky, 2008). In addition, *ex-vivo* human lumbar fascia behaves in a matter suggesting the presence of smooth muscle cells (Yahia et al., 1993). Moreover, the educated hypothesis of Schleip et al., (2005) suggests that these contractile cells may alter tissue pre-tension, therefore actively modifying the muscle's performance and biomechanical influence on the musculoskelal system.

These findings, amongst many others, support the notion that fascia plays an important role in joint strength and stability. Therefore, if weakened and/or rendered deficient, fascial membranes may distort regular joint dynamics through the impedance of regular and effective tensional implications. Such alterations would alter the stability and structural integrity of our bodies which is maintained by a balance between passive (fascia) and active (muscle contraction) tensional forces with a complementing hydrostatic (cartilage, intervertebral discs, and visceral pressure) and compressive resistance (bone). Translation of this principle to joint dynamics directly implicates fascia, since it is involved in passive or intrinsic tensional force and may partially regulate the contractile performance of muscles. Application of the aforementioned description of musculoskeletal stability, may explain how local instabilities lead to distant responses and/or pain, as recently observed between instable ankles with injured subtalus joint and irregular tensor fascia-lata muscle force duration (Zampagni et al., 2008). It therefore comes as no surprise, based on the above scientific contributions, that various fascial treatment avenues have been elegantly identified and exploited in practice (Chaitow, 1999; Myers, 2008; Paoletti, 2006; Schultz and Rosemary, 1996). In addition to its biomechanical implications, fascia is highly innervated consisting of nerve endings and encapsulated receptors as identified in the deep fascia examined from 20 upper limbs (Stecco et al., 2007). In a recent review, the implication of ligament innervations was linked to motor control and the structural integrity of the joint (Solomonow, 2006). Similar findings also emphasized this importance, as proprioceptive defects are found in, and may be linked to, disruptive limb control within the irregular structures of patients with cerebral palsy (CP) (Wingert et al., 2009). Although the presence of inadequate neuro-control mechanism undeniably plays a role in the disruptive ambulation of CP patients, the irregular mechanical environment of their distorted musculoskeletal system may impose additional and restrictive directives to their function.

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Consequences of instability in musculoskeletal disorders

Perhaps the most prominent demonstration of joint instability is observed within the heavily distorted musculoskeletal system of severe CP patients (i.e. gross motor function classification type 4 & 5 — non-ambulatory). Distinctively, articulations within this CP phenotype are often associated with spastic or hypertonic muscles in conjunction with weak or hypotoned muscles. Such muscle tone and associated force imbalance, has a detrimental influence on the developmental function and performance of affected joints often leading to painful contractures and subluxations. The existence of these biomechanical irregularities influences the musculature under consideration while having important implications on the health of involved fascial tissues.

Muscle control limitations in patients with CP are conventionally attributed to an inadequate reflex control system that provides continuous exaggerated contractile reflex information. Therefore, targeted muscles are forced into a long term shortened and "immobilized" state. Muscle then becomes rigid and confined, whereby this status invokes several underlying changes to the functional units biomechanics. An example of this force imbalance is shown as spastic wrist flexors demonstrate preferred biomechanical and chemical involvement when compared to wrist extensors (Pontén et al., 2005). Further, other scientists identified a correlation between biochemical and biomechanical measures of joint contractures in connective tissue of immobilized rabbit knees. As speculated by the authors, these findings suggest a lack of adequate stimulus (joint forces) to maintain tissue health (Akeson et al., 1974). Complementarily, several authors have quantified an increase of passive modulus, i.e. resistance to stretch, in spastic muscles. In particular, several distinguished studies clearly demonstrated this phenomenon using impressive montages and numerical analyses to isolate and quantify passive muscle forces in CP patient compared to control values (Kearney and Stein, 1997; Mirbagheri et al., 2001; Sinkjaer and Magnussen, 1994).

Upon revision of internal muscular biomechanics, several factors need to be considered: passive muscle resistance arises from a combination of stretching muscle fibers' cross-links; the extension of non-contractile proteins of sarcomeric cytoskeleton; and the modulus of the involved fascial layers (epi-, peri- and endo-mysium). Isolated muscle fibers from spastic individuals proved twice as strong as regular or healthy cells and had a significantly shorter resting sarcomere length (Lieber et al., 2003). However, repeating tests with spastic muscle bundles, although stronger than an individual cell, proved approximately three and a half times weaker than healthy muscle bundles (Fridén and Lieber, 2003). Upon comparison of these two analyses one might speculate that the extracellular matrix in a healthy bundle of muscle cells is stronger or more efficiently organized. However, as stated, under physiologic conditions, passive resistance (due to passive tissues, such as fascia, while discounting initial intrinsic stiffness and reflex-mediated contractions from muscle) of spastic muscles are more resilient to stretch. The difference between these findings occurs because, under in-vivo conditions, the presence of fascia is implicated as a surrounding tissue. Thus, the altered resistance to stretch may arise from the outer fascial tissue (epimysium and perimysium) that has undergone a form of remodeling and has become more resistant to stretch.

It is becoming clear that fascia adds an important aspect to joint dynamics, while spasticity invokes remodeling of both the involved muscle and implicated fascial tissues. It appears that fascia has a role in: passively resisting stretch during joint dynamics; providing tensional forces in order to release stored energy in the form of pre-stress; providing musculoskeletal proprioceptive feedback; and offering a feasible platform for force transmission between one or several muscles that may be actively adjusted via pre-tension.

Correlations between muscle and elastic force from fascia have not been effectively established, nor, to date, have there been studies of the remodeling process of the fascial membrane. Also, the passive distribution of load within the muscle and the connective tissue is not well defined. However, with reference to the publications discussed herein, one can reasonably assume that its presence is significant. It is known that spastic muscles have significantly more resistance to elongation. Therefore, one may deduce with respect to CP patients, both the muscle and the fascial layer have higher mechanical properties that define its resistance to stretch, while the hypotoned stabilizer or weakened co-contractor will likely have a reduced modulus, due to disuse. Previous emphasis of biomechanical remodeling has focused attention on musculature and bones. However, although less evident to the naked eye, implicated fascial tissues also undergo remodeling due to irregular stimulus. Such remodeling is of concern as connective tissues provide an important role in governing the capabilities of the musculoskeletal system. Fascia does not possess the capability to effectively alter its tensional properties as witnessed in skeletal muscles. Therefore, alterations of its intrinsic mechanical properties via remodeling may have considerable implications determining internal stress allocation (tissue stimulus) within musculoskeletal disorders under the stress shielding phenomenon. In the authors' opinion and the key concept to this manuscript, such a mechanical imbalance plays a decisive role in manipulating internal load allocation in any musculoskeletal disorder whose stabilizing structures have yielded and undergone partial or complete remodeling.

Selective load distribution in musculoskeletal disorders

Selective load distribution is hereby speculated to occur in all relevant musculoskeletal disorders with altered internal mechanical properties. However for clarification and argument sake, patients with severe CP will continue to be utilized to demonstrate this principle. In congenital CP patients, irregular and distorted load bearing configurations assumed by their limbs are governed by previously mentioned inadequacies of their functional stabilizers. This is clearly evident upon analysis of these patients' activities,

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such as disrupted gait patterns triggered by irregular muscle activation strategies — identified via EMG analysis (Dietz and Berger, 1995). Mechanically, this adopted selective-load support means that severe CP patients will attempt to perform load bearing activities while favoring the hypertonic muscle groups since they are "more fit" to sustain loads. In support of such notion, Tynan et al. identified enlarged peroneal compartments relative to anterior portion when forefoot pes cavus were compared with controls via magnetic resonance imaging. In addition, the authors speculate that this muscular imbalance will continue to provide overaction of the peroneus longus and thus play a role in pathogenesis of symptomatic cases of forefoot pes cavus (Tynan et al., 1992).

The transition from regular muscle tone to hypertonic and/or spasticity may be part of the regressive musculoskeletal function identified in severe CP patients using temporal analyses of their function (Hanna et al., 2009). This transition does not occur in the short term. It is a progressive disruption of regular joint mechanics caused by irregular loading conditions imposed within the joint. Although common belief has it that neurological lesions play a dominant role in its etiology, and thus initiate this process, the transition or progression of muscles into heavy spastic and hyper-toned phenotypes may not be attributed to a non-progressive inadequate reflex control system. This is because, in order to alter the mechanical performance of both the muscle and fascial tissues involved in a spastic contraction, one must stimulate these tissues via dynamic or cyclic stresses. For example, attempts to improve biceps strength would not achieve by holding a weight in one position. Instead, repeated loading (biceps curls) would induce changes in the ability to perform arm flexion. These requirements apply for CP patients or any other progressive musculoskeletal disorder.

The initial onset of hypertoned muscles, as mentioned, involves muscle shortening and/or immobilization. That is, there is an increase in positive reflex muscle stimulus often triggering flexion or pronation, which restricts functional use, leading to disuse. However, initially, articulations may still be forced through what is quantified as a regular range of motion. Referring to Figure 1, this would be defined as Transition 1 ($T1$), or by passing from healthy muscle and connective tissue rigidities to hyper or hypotoned muscle groups. In regular joint articulations, passive rigidities of co-contractors are similar. Therefore, mediated contractions dictate controlled motion and internal stress stimulus. Further, in a healthy joint, it is known that intentional stimulus of co-contractions occur. Although not mechanically efficient, as it imposes restrictive moments or reduces functional torque on the selected motion, this co-contraction is necessary to both stabilize and assume accurate control over the desired motion. A child who has transitioned to T_1 due to inadequate neurological control, and/or disproportion of muscle and bone growth, will undergo disuse atrophy, or weakening of the extensor or antagonistic. This event may play a role in the reduced ability to perform accurate and controlled motion as frequently observed in CP patients.

As a result of these alterations, balanced or healthy mechanical properties previously present in articulations will be offset. This would entail weakened properties of the

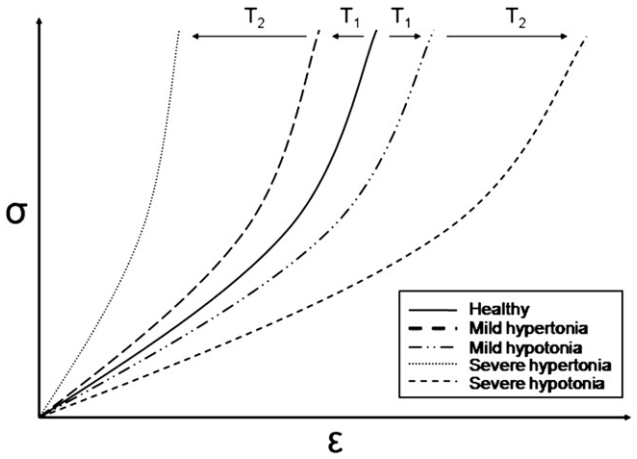

Figure 1 Depiction of the transition from healthy mechanical properties to the irregular or offset relationship between agonistic and antagonist muscle groups in patients with cerebral palsy.

antagonist, with elevated stiffness in the agonistic group. Currently, the second transition (T_2) is poorly understood, as a consequence of the pathomechanism of hypertonic muscles being a difficult phenomenon to study. However, it is recognized that different muscle groups are more susceptible to: a quicker transition to spasticity; a reduced range of motion; and an associated rigid joint. Further, as mentioned, selective load bearing configurations are undertaken in joints having transitioned to T_1. This new mechanical environment imposed on joints induces a stress shielding effect on the antagonistic group. In other words, because the hypertonic muscle group is more rigid than its antagonistic hypotonic group, it will assume the majority of the load. This occurs naturally within internal load distribution and is hypothesized to be dominantly involved in the transition to T_2, a more degenerated and detrimental state than T_1.

Under the stress shielding phenomenon, the possible reason for this occurrence is that distribution of loads will occur via the most suitable mechanism in order to achieve stability. Although the concept of stress shielding is conventionally reserved to describe how the introduction of rigid prosthetics shelters the surrounding bone from regular stimulus, this principle may be extended to describe how connective tissues and/or muscle units that are more rigid than its counterpart will undertake the majority of the load. In a healthy individual one may regulate their stabilizers stiffness, through controlled muscle contractions, in order to achieve affective and energy efficient stability. However, the stress shielding phenomenon may, in part, describe how an injury to any lower leg stabilizer leads to limping for example (conscious selective load bearing). Although pain likely governs the act of limping, one may entertain the idea that this irregular gait may be provoked by injured tissues not being mechanically fit to sustain regular loading. In a patient with CP, implementation of the stress shielding phenomenon is more apparent since the conscious adjustability of muscle tone is hindered and, therefore, loads are likely distributed to stiffer parties. Figure 2 coupled with mechanical interpretation further

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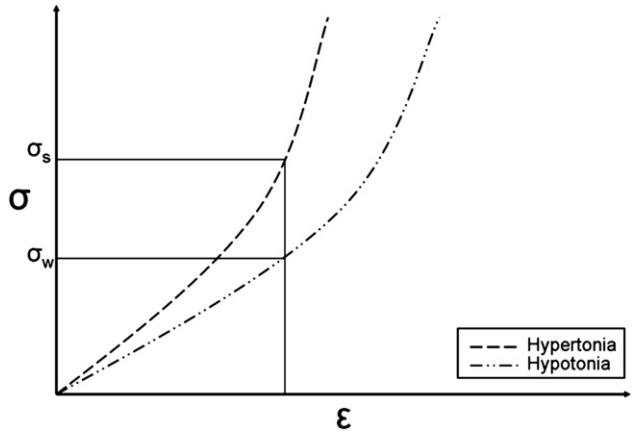

Figure 2 Stress shielding phenomenon governed by mechanical properties of musculature.

rationalize this phenomenon. As previously described, the hypertonic and hypotonic muscle groups have different rigidities (instantaneous slopes of non-linear moduli) and, therefore, different stress-strain relationships. However, they are both present when joint stability is achieved. For argument sake, imagine that the joint attempts to collapse in a manner that elongates the involved muscle groups equally. As a consequence of one muscle being more rigid than the other, the elongation of the weaker group (ε_w) will be restricted by that of the stronger muscle (ε_s), as shown in Eq. (1). Further, fundamentals show that strain, or normalized elongation (ε), is dependent on the ratio of stress (σ) over its rigidity (E) (Eq. 2). Therefore, the amount of stress experienced in the weaker or hypotoned muscle group (σ_w) will be proportional to the ratio of rigidities between the weak (E_w) and the strong (E_s) tissues. Therefore internal stress shielding will occur (Eq. 3).

$$\varepsilon_w = \varepsilon_s \tag{1}$$

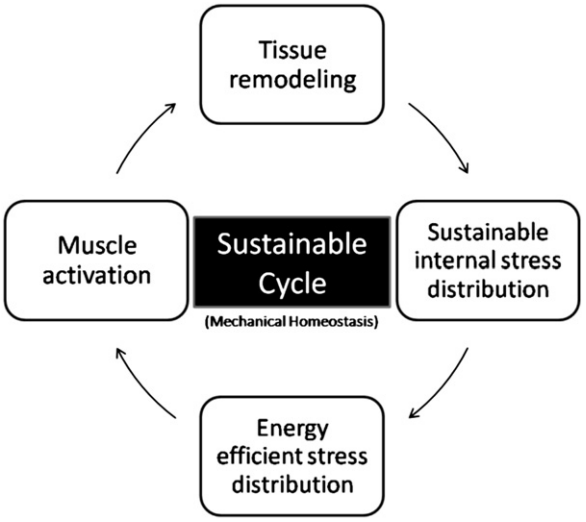

Figure 3 Physiological tissue mechanical homeostasis cycle (sustainable cycle).

$$\varepsilon = \frac{\sigma}{E} \tag{2}$$

$$\sigma_w = \sigma_s \left(\frac{E_w}{E_s}\right) E_w < E_s \therefore \sigma_w < \sigma_s \tag{3}$$

This creates elevated stress levels (σ_s) in the stronger group and reduced stress levels (σ_w) in the weaker ones (Figure 2), when compared to regular conditions of a healthy individual.

Important implications of physiological stress shielding

Physiological stress shielding is of great importance since the integrity or health of all physiological tissues responds

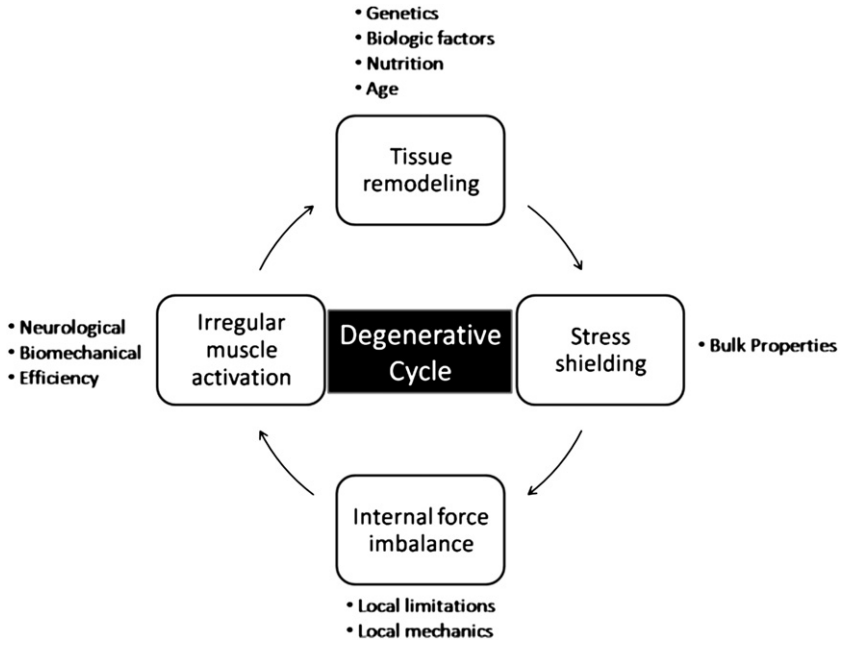

Figure 4 Degenerative cycle of physiological tissue in musculoskeletal disorders.

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to stress input. Stress is the factor that triggers the appropriate mechanotransduction, which in turn regulates the tissue performance and defines its mechanical homeostasis or sustainable cycle as summarized in Figure 3. However, if sheltered from regular stresses due to physiological stress shielding and/or internal force imbalance and/or irregular muscle activation, neglected tissues will resorb or disappear. In contrast, if excessively stimulated, the tissue will become stronger. This has long been quantified in bone, under Wolff's Law (Wolff, 1892), and integrated into various feedback algorithms that demonstrate the theories feasibility in predicting disuse atrophy caused by the presence of rigid prostheses (Hart et al., 1984). Further, the notion of tissue-health stress dependence has been quantified to regulate cartilage, ligaments, tendons, and joint capsules (Von Reyher, 1874; Weichselbaum, 1878; Parker and Keefer, 1935; Salter and Field, 1960). This well known tissue health dependence on stimulation allows one to conceive of the flawed or non-sustainable cycle that occurs in subjects with qualitatively evident and quantitatively documented irregular musculoskeletal stimulus. This unsuitable cycle may be envisioned as a temporal transition from mechanically balanced or healthy properties to those "stability inappropriate" properties of CP patients (Figure 1). In parallel, Figure 4, an inversion of the mechanical homeostasis cycle, provides an appropriate depiction of a regressive musculoskeletal system or degenerative cycle under the stress shielding phenomenon. Although the etiology or origin of this degenerative cycle may arise from a number of factors detailed on the extremities of Figure 4, once initiated this iterative control system will likely continue unless otherwise interrupted via the appropriate treatment. However, it is reasonably safe to conclude that a dominant player in the restrictive transition, from healthy functional characteristics to a distorted and non-functional state such as T_2 (Figure 1), is the role physiologic stress-shielding plays in irregular mechanical environments. Such stress-shielding phenomenon was previously demonstrated to occur in spinal columns of patients with scoliosis since the presence of local physiological rigidity increases were shown to cause an augmentation in stress levels when compared to regular conditions and, as a result, encouraged the progression of the scoliotic deformation as predicted via finite element modeling (Driscoll et al., 2009). In a similar fashion, the hypothesis portrayed herein suggests stress shielding plays a significant role in the deterioration of the CP musculoskeletal system.

Specifically with regards to CP patients, the onsets of the phenotypic associated deficiencies are conventionally attributed to neurological shortcomings. However, one must not neglect the likelihood of multifactorial causes nor

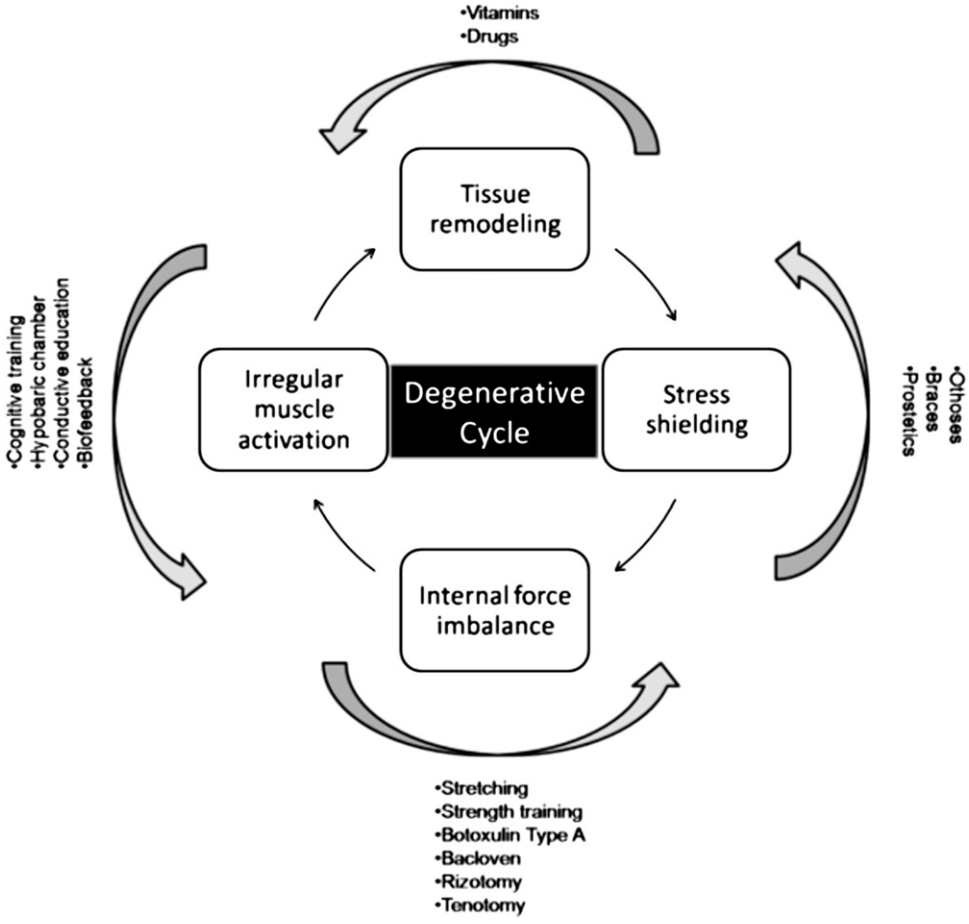

Figure 5 Specific therapeutic attempts to impede, halt or reverse the degenerative cycle in patients with cerebral palsy.

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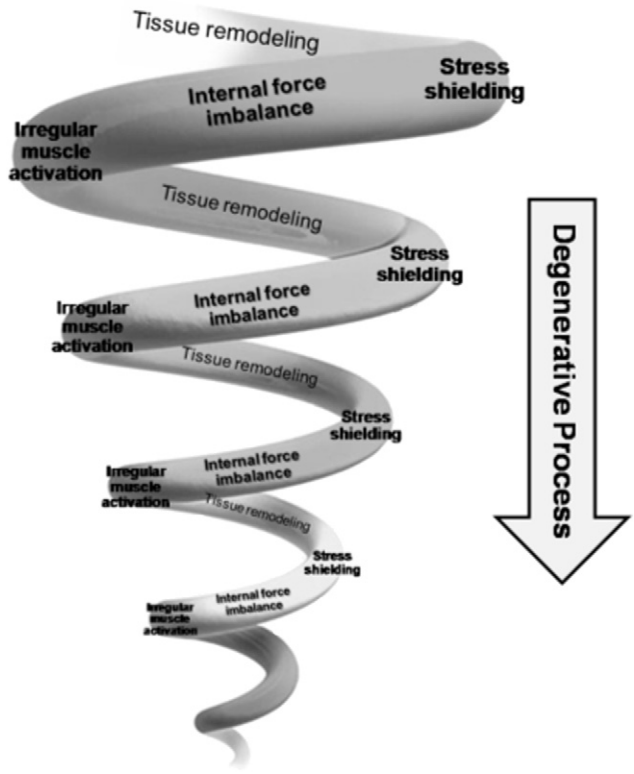

Figure 6 Degenerative process of untreated musculoskeletal deficiencies.

must they discredit the role of altered biomechanics in the deterioration of a CP musculoskeletal system. Having recognized the significance of such factors, many rehabilitative methods have been adopted in attempt to restore mechanical homeostasis (i.e. reverse the degenerative cycle) as portrayed in Figure 5. Moreover, in the authors' opinion, this degenerative cycle will become a degenerative process and thus the longer left untreated or neglected the further ones mechanical imbalance or musculoskeletal shortcomings may progress as depicted in Figure 6. These illustrations highlight two important concepts. Firstly, they demonstrate the need to combine therapeutic interventions to fully address the available rehabilitative spectrum of musculoskeletal disorders. Secondly, and perhaps most importantly, the degenerative process emphasizes the need to intervene promptly in order to most effectively restore adequate and regular musculoskeletal function. In other words, this process insinuates that there lies an immense rehabilitative challenge for severely affected CP individuals as they may have "spiraled down" several levels within the suggested degenerative process.

Conclusions

Joint mechanics of a CP patient are disrupted and defined by fascial muscle groups that are strong, over used, hypertonic, and/or spastic, which are often coupled with co-contractors that are hypotonic, under stimulated, and weak. The diverging mechanical characteristics of these two groups lead to asymmetrical load distributions governed by the differences in mechanical properties, a phenomenon known as stress shielding. Further, this principle coupled with the lack of adequate neurological control over strong and weak tissues observed in CP, complements the regressive transition of their musculoskeletal system defined by a degenerative cycle, which are expressed through functional limitations observed throughout the degenerative process. With this under consideration, one may now critically analyze current treatments that seek to address the restrictive phenotypes associated with CP. Moreover, the acknowledgment of physiological stress shielding highlights a therapeutic avenue that requires additional recognition — how to actively transfer stress to the otherwise neglected tissues in order to stimulate beneficial remodeling or self healing.

In closing, this article proposes the concept of physiologic stress shielding having a role in the pathomechanism of regressive musculoskeletal disorders such as CP. If neglected, physical fundamentals will continue to shield deficient tissues, thus, sheltering them from what may be characterized as a Darwinian competition for stimulus. In contrast, recognition of this aspect may lend insight into undeniable benefits that arise from therapeutic or massage methods that actively induce stimulus to the physiologically shielded tissues, which play a passive but important role in joint integrity.

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