

## Review

# Neuromechanical coupling in the regulation of muscle tone and joint stiffness

A. R. Needle, J. Baumeister, T. W. Kaminski, J. S. Higginson, W. B. Farquhar, C. B. Swanik

Department of Health and Exercise Science, Appalachian State University, Boone, North Carolina, USA

Corresponding author: Alan R. Needle, PhD, ATC, CSCS, Holmes Convocation Center 011, 111 Rivers St., ASU Box 32071, Boone, NC 28608, USA. Tel: 828-262-4039, Fax: 828-262-3138, E-mail: needlear@appstate.edu

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The ability of the nervous system to accommodate changes to joint mechanics is crucial in the maintenance of joint stability and the prevention of injury. This neuromechanical coupling is achieved through several mechanisms such as the central and peripheral regulation of muscle tone and subsequent alterations to joint stiffness. Following joint injury, such as a ligamentous sprains, some patients develop functional instability or require surgery to stabilize the joint, while others are able to cope and display limited impairments. Several researchers have attempted to explain these divergent

outcomes, although research using proprioceptive tasks and quantifying reaction times has led to equivocal results. Recent innovations have allowed for the simultaneous measurement of mechanical and nervous system function among these subsets. The intent of this review was to explore the relationships between joint stiffness and nervous system function, and how it changes following injury. By better understanding these mechanisms, researchers and clinicians may better develop and implement rehabilitation protocols to target individual deficits among injured populations.

The maintenance of joint stability is a critical factor for daily function and the prevention of falls and injury. Throughout physical activity, the neurological and mechanical components of a joint must work collectively to both prepare for and react to injurious perturbations (Sherrington, 1911; Freeman & Wyke, 1967; Lacroix, 1981; Sinkjaer et al., 1988; Johansson et al., 1991). The result of this neuromechanical coupling is a modulation in muscle tone that can optimize joint stiffness for specific tasks, contributing toward injury prevention and improved functional performance (Sinkjaer et al., 1988; Nielsen et al., 1994). The intent of this article is to examine the combined influence of muscle tone and joint stiffness regulation with specific consideration to their roles in sports medicine practice.

## Muscle tone

The phenomenon of muscle tone is a commonly misunderstood concept when discussing injury prevention and athletic performance (Masi & Hannon, 2008). Defined as the low-level steady-state muscle contraction at rest, it exists unconsciously, and controls the tautness of a relaxed muscle. Upon passive stretching of muscles with heightened tone, the mechanical response will be increased resistance to length changes (Davidoff, 1992). Much of what we understand about muscle tone relates to pathological populations including forms of hypertonicity such as spasticity and rigidity; or *in vitro* models

of decerebrate felines (Bosma & Gellhorn, 1947; Eccles & Lundberg, 1959; Burke, 1983; Sinkjaer, 1997; Breakefield et al., 2008). However, limited research exists regarding potential mechanisms capable of altering this descending drive among healthy adults, as well as those with joint injury. This information would be clinically useful because tone may be described as the muscles' "state of readiness," which can be modified based on specific functional tasks and the individual's level of anxiety (Saper, 2000; Davis et al., 2011). Therefore, the neuromechanical properties of muscle tone have an important role for both maximizing functional performance and preventing injury.

## Peripheral mechanisms

Original theories suggested that muscle tone is mediated peripherally by the fusimotor system – the neural circuits existing between the muscle spindle, its afferent axons (Type Ia & Type II) and the gamma ( $\gamma$ ) motor neurons that act to alter spindle sensitivity by changing the length and tension of intrafusal fibers (Burke, 1983; Johansson et al., 1986; Bergenheim et al., 1995; Pearson & Gordon, 2000). This increase, or gain in the gamma motor neuron drive is associated with coactivation of the alpha motor neurons controlling the contraction of the extrafusal skeletal muscle fibers (Johansson et al., 1986; Knutson, 2000). Therefore, increased activity of the fusimotor system translates into higher levels of resting skeletal

Table 1. Classification and conditions affecting muscle tone

Condition	Description	Potential Cause(s)
Hypotonicity	Abnormal decrease in muscle tone; associated with hypoactive reflexes	Lower motor neuron lesions Posterior cerebellar lobe lesions
Hypertonicity	Abnormal increase in muscle tone with resistance to active and passive movement; associated with hyperactive reflexes	Upper motor neuron lesions Anterior cerebellar lobe lesions
Rigidity	Hypertonicity to muscles on <i>both</i> sides of a joint	Parkinson's disease and other neurological disorders
Spasticity	Hypertonicity on <i>one</i> side of a joint	Spinal cord injury above T12, head injury, cerebrovascular accident, cerebral palsy
Clonus	Uncontrolled oscillation of muscle in a spastic muscle group	Upper motor neuron lesions
Dystonia	Involuntary muscle contraction and abnormal posture in a specific part of the body	Task-specific Interruption of cortical-basal ganglia loops.

Adapted from: Gutman, S.A. 2001, Quick Reference Neuroscience for Rehabilitation Professionals, 1st edition, Slack, Inc., Thorofare, NJ.

muscle contraction or tone. When this reflexive loop becomes overactive, disorders of muscle tone such as spasticity and hyperreflexia may occur (Burke, 1983; Sinkjaer, 1997; Hallett, 1998; Breakefield et al., 2008; Hall, 2011). However, this approach to understanding muscle tone is somewhat isolated to the peripheral nervous system (PNS), but it is known that alterations to the central nervous system (CNS) can lead to pathologies related to muscle tone (Table 1) (Bosma & Gellhorn, 1947; Eccles & Lundberg, 1959; Burke, 1983).

### Central mechanisms

Several central mechanisms have been associated with heightened muscle tone including pathways from the cerebellum, basal ganglia and thalamus (Burke, 1983; Saper, 2000; Hall, 2011). These structures are capable of altering muscle spindle sensitivity by regulating inhibition to the fusimotor loop (Eccles & Lundberg, 1957). Therefore, increased intracortical inhibitory activity is responsible for decreasing muscle tone (Eccles & Lundberg, 1957; Rymer et al., 1979; Ridding et al., 1995; Hallett, 1998). Much of what we know about this relationship comes from observations among patients with CNS disorders that, for a variety of reasons, have difficulty regulating inhibitory and excitatory signals within the CNS. This leads to an outward appearance of altered muscle tone, including spasticity and dystonia (Breakefield et al., 2008). Findings of decreased intracortical inhibition are consistently observed in spastic and dystonic populations, as well as increased motor excitability suggesting a loss of cortical mediation where the excitatory pathways are left uninhibited, leading to continuous increased motor neuron firing (Ridding et al., 1995; Devanne et al., 1997; Hallett, 1998). Because of these complex interrelationships among the PNS and CNS, very few studies have attempted to quantify subtle differences in muscle tone among healthy or athletic patients, although preliminary links have been observed between the mechanical stiffness of the joint and these cortical measures (Needle et al., 2013a). To comprehend this relationship and

connect tone to the muscles' "state of readiness," we should consider the process of events necessary for altering tone from a resting, "baseline" state.

If the nervous system had to be "turned on," such as awaking from a deep sleep, the sequence for generating muscle tone would proceed as such. The reticular formation will emit excitatory signals to arouse the brain and trigger awareness across the cerebral cortex. Signals originating in the brainstem and cerebellum will travel to the gamma motor neurons along extrapyramidal pathways including the reticulospinal, vestibulospinal and rubrospinal tracts. Muscle tone will then begin to spread through extrapyramidal pathways that include the reticulospinal, vestibulospinal and rubrospinal tracts originating in the brainstem and cerebellum travelling to the gamma motor neurons (Moruzzi & Magoun, 1949; Siegel et al., 1983; Saper, 2000; Hall, 2011). This causes the spindle to become taut, as if the muscle were suddenly stretched, stimulating the intrafusal spindle sensory organ. The intrafusal fibers generate an excitatory afferent signal that is transmitted back to the spinal cord through fast sensory nerves. These sensory fibers directly synapse with  $\alpha$  motor neurons, resulting in a monosynaptic stretch reflex and contraction of the same muscle containing the spindles (Pearson & Gordon, 2000). Together, with signals from the corticospinal tract, with modification by the basal ganglia, the alpha-gamma co-activation will establish baseline levels of muscle tone (Burke, 1983). This would initially be mediated simply through the fusimotor loop; however, as this process occurs, the anterior and posterior spinocerebellar tracts will transmit new proprioceptive information to the cerebellum (Saper, 2000). The result leads to a feedforward/feedback loop such that the cerebellum modifies activity of the brainstem and basal ganglia to control tone (King, 1948; Saper, 2000; Hall, 2011). Without inhibition from higher cortical structures, the original monosynaptic stretch reflex is so potent that an individual would exhibit hypertonicity or spasticity, and movement becomes abrupt and erratic (Breakefield et al., 2008; Hall, 2011). In circumstances where individuals become anxious, the cortex normally decreases inhibitory influences, allowing for an increased stretch

reflex and heightened muscle tone. This may serve to maximize muscular responsiveness and protect the individual under stressful conditions (Davis et al., 2011). However, if muscle tone is increased excessively, such as when an individual becomes startled, it could disrupt normal agonist – antagonist co-activation and inhibition (Freeman & Wyke, 1965, 1967). This leads to erratic movements and subsequent errors in coordination that decrease functional performance and increase risk of injury (Iversen et al., 2000; Swanik et al., 2007; Moffit, 2008; Mobbs et al., 2009).

### Measurement of tone

Our understanding of muscle tone, in otherwise neurologically healthy individuals, has been limited by the absence of instruments to concurrently measure both neural and mechanical contributors with sufficient temporal resolution to couple events. Quantifying joint stiffness by applying a sudden perturbation has been previously utilized to quantify tone (Boiteau et al., 1995; Lorentzen et al., 2012); however, limitations exist as these characteristics are dependent on the velocity of the perturbation, and the almost instantaneous superimposition of reflexive muscle responses (Sinkjaer, 1997). While several investigators have constructed customized devices to measure muscle tone, no device has been used consistently throughout the literature, potentially because of poor reliability (Boiteau et al., 1995; Lorentzen et al., 2012). Therefore, some researchers have focused on understanding the brain's relationship to resting muscle activity and stiffness.

One tool for investigating this relationship, transcranial magnetic stimulation, is often employed because of its versatility as techniques allow for understanding both excitatory and inhibitory mechanisms of cortical motor neurons (Hallett, 2007). This technique involves administration of brief magnetic pulses over various areas of the cerebral cortex, sometimes with multiple pulses applied rapidly, and measuring the size of a motor response from a targeted muscle (Devanne et al., 1997; Hallett, 2007). Several transcranial magnetic stimulation-derived variables have been linked to disorders of muscle tone. Among patients with hand-grip dystonia, separate studies have reported a higher gain in the stimulus-response properties, indicating that for a given size stimulus, there exists a more rapid recruitment of additional cortical motor neurons. These findings indicate stronger connections between the brain and the muscle, and perhaps a disorder of cortical modification to the alpha motor neurons (Ridding et al., 1995). While these excitatory connections are an important link, it is important to recall that inhibitory mechanisms within the cortex are largely responsible for downregulating the stretch reflex and muscle tone. Thus, accordingly, suppression of inhibitory pathways (*disinhibition*) is present among hypertonic populations (Ridding et al., 1995; Hallett, 2007). The

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inhibitory mechanisms observed from these measures have been associated with the corticofugal projection fibers to the basal ganglia and thalamus – the same pathways leading to hypertonicity (Hallett, 2007). Identifying these CNS correlates of muscle tone creates the opportunity to study how neuromechanical coupling may be affected among injuries common to sports medicine practice.

While much remains unknown about muscle tone, it appears that this property provides a strong link between the PNS, the CNS and the mechanical properties of the joint. Therefore, when one considers the phenomenon of joint instability as consisting of both mechanical and neurophysiological deficits, it warrants questions by both clinicians and researchers regarding how this quality may affect the ability to protect the joint while maximizing functional performance.

### Muscle and joint stiffness

Modification of muscle tone is one component contributing to a joint's stiffness properties. Defined as the resistance of the joint and its supporting structures to stretch, these stiffness properties would determine the amount of force required to cause injury (Latash & Zatsiorsky, 1993). This has important clinical implications as higher joint stiffness may represent better joint stability and a failure to appropriately regulate stiffness contributes to injury (Johansson et al., 1991; Wilson et al. 1991a). Alternately, to improve performance, stiffness must be optimized, using potentially less stiffness or greater compliance to allow for greater absorption of load and storing elastic energy. Selective muscle recruitment may therefore be necessary to allow individuals to operate within an ideal performance envelope and avoid injury (Bach et al., 1983; Wilson et al. 1991a; Gottlieb, 1996). Often represented by a load-deformation curve that represents the amount of displacement per unit force, these properties may predict the force at which a tissue would become damaged. Additionally, by studying the “toe region” during which the joint moves from a shortened state to its resting length, we may understand the threshold at which embedded mechanoreceptors might be stimulated (Sinkjaer et al., 1988; Wilson et al., 1991b). While each tissue possesses its own stiffness characteristics, the overall joint stiffness is representative of the combined resistance from the surrounding joint capsule, ligaments, muscle and skin. All of these structures have their own intrinsic properties, but muscular stiffness would additionally be modulated by the amount of contraction and formed crossbridges.

### Passive stiffness

Muscular contraction adds a degree of complexity to the joint's stiffness, creating a link between the joint's resistance to load and neuromuscular function (Sinkjaer

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et al., 1988; Latash & Zatsiorsky, 1993). In a relaxed state, with volitional muscle activity absent, stiffness is regulated largely by the elastic components of the surrounding joint capsule, ligaments, the parallel and series elastic components from the musculotendinous unit, and resting muscle tone (Sinkjaer et al., 1988; Toft et al., 1989a, b). If a joint perturbation is of enough speed and magnitude, a reflexive muscle response may, almost instantaneously, aid in increasing joint stiffness. As muscle spindles mediate the monosynaptic reflex, the amount of muscle tone and subsequent sensitivity of those receptors could potentially increase the likelihood of a reflexive response (Nielsen et al., 1994). For instance, if muscle tone were higher, this would imply a higher gain to the muscle spindle, and therefore an increased sensitivity to stretch. While passive stiffness has been studied in sports medicine research, the exact contributions of muscle tone are unclear as this resting activity is not apparent on electromyography.

### Contribution of volitional contraction

Volitional muscle activity has a profound effect on the stiffness of the joint, leading to more than fourfold increase in measured joint resistance simply by maintaining a baseline level of precontraction throughout a perturbation (Huxel et al., 2008; Thomas et al., 2013). This increase from muscular stiffness represents the individual's ability to regulate the reverse cycling of the actin-myosin crossbridges (Nielsen et al., 1994; Huxel et al., 2008; Thomas et al., 2013). Furthermore, as an individual experiences a potentially injurious load, length changes in the surrounding muscle would initiate reflexive muscular activation, which may serve to create a more drastic increase in joint stiffness to protect musculoskeletal structures. The relationship between muscle activation and joint stiffness therefore represents the capacity to integrate mechanical and neurophysiologic mechanisms to improve performance and prevent injury.

### Stiffness measurement techniques

Several researchers have investigated joint stiffness in healthy and injured populations. Methods include handheld devices such as joint arthrometry (Fox et al., 1985; Kovaleski et al., 2002, 2008; Bahk et al., 2007), application of joint perturbations (Huxel et al., 2008; Zinder et al., 2009) and use of motion analysis and inverse dynamics to calculate joint stiffness during activity (McGinnis et al., 2013; Shamaei et al., 2013). Arthrometry involves the manual application of translational or rotational stresses of a known load, allowing investigators to study the contributions of passive structures to joint stiffness across various ranges (Fox et al., 1985; Kovaleski et al., 2002; Bahk et al., 2007). These devices are designed to passively quantify stiffness and

laxity changes in the capsuloligamentous structures, as well as resistance from the relaxed muscle (Kovaleski et al., 2002; Needle et al., 2013a). An advantage of these devices is their ability to identify deficits across discrete loading ranges, and offer a unique opportunity to study the "toe region" where early mechanoreceptor stimulation occurs (Needle et al., 2013b). Investigators have used free oscillating platforms or motors to apply a rotational perturbation to a joint, while the resistance the joint provides is measured through a load cell. These devices allow for measurement of stiffness over a variety of controlled conditions, with the ability to manipulate the amount of volitional muscle contraction (Thomas et al., 2013; Zinder et al., 2009). Furthermore, perturbations applied with a motor offer the advantage of controlling the exact magnitude, velocity and acceleration of the perturbation to closely replicate injurious mechanisms, whereas oscillatory platforms derive stiffness from the dampening response of the device (Otis et al., 1983; Latash & Zatsiorsky, 1993; Oatis, 1993; Swanik et al., 2004; Blackburn et al., 2006; Huxel et al., 2008). Finally, calculating joint stiffness through inverse dynamics during motion analysis has been used to quantify changes while performing an activity, but these values may not reflect loads associated with injury (McGinnis et al., 2013; Shamaei et al., 2013).

### Joint injury pathway

As muscle tone and the subsequent joint stiffness demonstrate a coupling of the nervous system and joint mechanics, they have an important role in the preparation and responses to joint perturbations. Prevention of joint injury is often addressed through two separate mechanisms: feedback (closed-loop/reactive) and feedforward (open-loop/preparatory) neuromuscular control (Lacroix, 1981; Dunn et al., 1986). To understand these concepts, it is helpful to address the cascade of events that lead to injury from the start of a perturbation.

### Application of a load

As the joint is loaded and stress is applied to the supporting tissues, a complex sensorimotor response at multiple levels of the nervous system is triggered. Initial detection of the load is dependent on the peripheral mechanoreceptors embedded in the skin, joint capsule, ligaments and the musculotendinous unit (Freeman & Wyke, 1965, 1967; Johansson, 1991; Hutton & Atwater, 1992; Michelson & Hutchins, 1995). These receptors include both slow- and fast-adapting receptors that detect stretch, tension and load (Michelson & Hutchins, 1995). Arguably, the most important of these receptors are the muscle spindles, as afferents from other joint mechanoreceptors act to modify fusimotor activity through polysynaptic reflex pathways (Johansson et al., 1986, 1991). Some of the most compelling evidence for

the role of muscle spindle activity on peripheral sensation was derived by Johansson et al. (1986) in a series of studies that severed the feline anterior cruciate ligament and observed altered afferent spindle activity. It was hypothesized that as muscle spindle sensitivity was modifiable by ascending and descending pathways, these spindle afferents transmitted a final common input of sensory activity to the nervous system. Furthermore, severing the anterior cruciate ligament and thereby removing its afferent input had a profound effect on spindle sensitivity, and therefore could influence the reflexive responses in muscles surrounding the joint (Johansson et al., 1991).

The input from the muscle spindle to the spinal cord may be enough to trigger a monosynaptic reflex providing some degree of joint stabilization. However, the afferent stimulus will continue to ascend in the CNS via the dorsal column-medial lemniscal and spinothalamic tracts to the thalamus, internal capsule, and ultimately the somatosensory cortex, with parallel tracts (spinocerebellar and spinoreticular) conveying sensory information to the medulla, pons and cerebellum (Prud'homme & Kalaska, 1994; Hall, 2011). While these pathways may be enough to induce reflexive responses from higher centers, largely through the cerebellum's role in the maintenance of tone, a volitional response to stabilize the joint will form in the primary motor cortex (Kolb et al., 1997; Ehrsson et al., 2007). To initiate this response, projection fibers from the somatosensory cortex will extend toward the prefrontal cortex, supplementary motor areas, premotor areas and, ultimately, the primary motor cortex. This is where a volitional muscle activity will be triggered by motor neuron bodies in the cortex, with their axons forming the descending corticospinal tracts, leading to contraction (or potentially deactivation) of stabilizing muscles (Hall, 2011).

### Preparing for injury

As the speed at which these events occur is extremely fast, limited instrumentation is capable of investigating this chain of events *in vivo* to determine the loading stimulus intensities and velocities necessary to elicit responses at each level, or precisely how the CNS optimizes muscle tone and joint stiffness for the prevention of injuries. However, multiple studies have investigated the “input” and “output” of the system in isolated situations. This involves perturbing the joint and measuring the relative timing and amplitude of the muscle response or stiffness change following a joint perturbation (Konradsen & Ravn, 1991; Konradsen 2002b; Myers et al., 2004; Delahunt, 2007). While the effect of joint injury on this ability has led to conflicting results, researchers have hypothesized that the fastest reflexive muscle responses (approximately 30–80 ms) may not be rapid enough to prevent ligamentous injury (occurring as quickly as 50 ms) (Freeman & Wyke, 1967; Sinkjaer

et al., 1988; Konradsen 2002b; Swanik et al., 2004). Therefore, if the quickest response is not enough to protect the joint, how would a volitional response (180–250 ms) provide stabilization to the joint?

One answer to this timing dilemma is found in the preparatory, or feedforward, muscle activity. Before each joint load, whether landing from a jump or taking a step, muscular tension is produced to prepare the joint for impact, with the amount highly modifiable to account for any unforeseen perturbations or unanticipated events (such as landing on someone's foot or stepping on an uneven surface) (Dunn et al., 1986; Huxel et al., 2008; Gutierrez et al., 2009). This serves two main purposes. First, joint pre-activation will increase the muscle activity and subsequent stiffness of the joint, meaning a higher amount of load would be required to perturb the joint (Latash & Zatsiorsky, 1993; Huxel et al., 2008; Thomas et al., 2013). Second, this pre-activation will increase the aforementioned fusimotor activity for the muscles surrounding the joint through alpha-gamma coactivation, allowing for quicker sensation of length change, with faster and larger reflexive responses (Dietz et al., 1981; Johansson et al., 1986; Bergenheim et al., 1995). This pre-activation therefore serves to both protect the joint prior to load and facilitate a response in the case of an unanticipated perturbation.

Muscular pre-activation may also be modified by several factors. These include memory, visual cues, vestibular cues and planning (Gottlieb, 1996; Hermsdörfer et al., 2008; Jackson et al., 2009). This means that signals from the frontal, temporal and occipital lobes act together to change activity from the primary motor cortex and supplementary motor areas. In many cases, the integration of information may serve to optimize levels of pre-activation based on visual cues, such as seeing an uneven surface or recalling successful movement strategies from previous experience. However, these complex interconnections could also serve to negatively impact joint stability. Increased cognitive loading and anxiety may lead either to a decreased amount of pre-activation and altered tone, or alternately a rapid, mistimed activation of various muscles that could negatively impact joint stability (Moffit, 2008). Some researchers have suggested that these associations may contribute to a negative feedback loop following injury that contributes to recurrent problems such as reinjury and instability (Wikstrom et al., 2013).

Limited studies have investigated the ability of the CNS to rapidly negotiate an appropriate response to joint perturbations. Currently, changes in sensorimotor control that occur centrally are quantified using functional magnetic resonance imaging (fMRI) (Kapreli et al., 2009) or electroencephalography (EEG) (Baumeister et al., 2008). Both of these techniques have various strengths and limitations, as fMRI offers high spatial resolution and low temporal resolution, with the converse true of EEG. One notable study by de Graaf et al. (2009) used fMRI to

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examine activity of the primary and secondary motor and sensory areas before and after a perturbation, with instructions to either resist or allow for a passive perturbation. An increase in primary motor cortex and somatosensory cortex activity in preparation for the activity was observed, suggesting both an increase in motor pre-activation and preparatory sensation where the brain may be anticipating how a perturbation should feel. This demonstrates the role of the cortex to not only increase joint stiffness prior to a load, but also reflexively plan potency of muscular reflexes in a feedforward manner (Henderson & Dittrich, 1998; Santello, 2005; de Graaf et al., 2009).

## Joint instability

This review has addressed the normal processes behind the regulation of muscle tone and joint stiffness and how these work together to prepare for and react to joint injury, but the question remains regarding how ligamentous injury affects these processes. Complaints of functional joint instability, where a patient experiences repeated sensations of “giving-way”, are common in the ankle, knee and shoulder joints (Yeung et al., 1994; Anandacoomarasamy & Barnsley, 2005; Clayton & Court-Brown, 2008; Murray et al., 2012). This may manifest as recurrent ankle sprains, loss of function and frequent falls, and repeated subluxations of the shoulder joint and, furthermore, have all been attributed to early onset post-traumatic osteoarthritis (Valderrabano et al., 2006; Clayton & Court-Brown, 2008). As these may arise following ligamentous injury, it was originally believed that the repeated injuries were secondary to increased joint laxity (Freeman, 1965). However, laxity did not correlate with sensations of instability as highly lax joints may function normally, and previously injured, unstable joints may heal and present with no mechanical insufficiencies (Eastlack et al., 1999; Bahk et al., 2007; Wikstrom & Brown, 2013). It was therefore suggested that following injury, the ligament itself might heal, but the capsuloligamentous mechanoreceptors may remain damaged, limiting the proprioceptive information to the nervous system, altering both preparatory muscle activity and delaying reaction times (Freeman et al., 1965; Johansson et al., 1991; Lephart et al., 1997; Myers et al., 2004). However, these proprioceptive and neuromuscular deficits inconsistently correlate with sensations of joint instability (Lephart et al., 1997; Arnold et al., 2009; Munn et al., 2010; Wright & Arnold, 2011; Gokeler et al., 2012; Laudner et al., 2012). As a result, the etiology and more importantly optimal treatment of joint instability continue to elude clinicians and researchers.

## Challenges in current research

Some problems currently exist with investigations into joint instability. First, changes in proprioception (the sensation of joint position, movement and force) and

neuromuscular control most often use indirect methodology: tests that require motor output (depression of a switch, contraction of a muscle) following a proprioceptive cue (joint movement or loading) to presume changes in the nervous system (Riemann & Lephart, 2002). However, many neurophysiological processes occur in the latency between onset of the cue and the motor output, making it difficult to detect impairments (Konradsen 2002a; Munn et al., 2010). Second, current research utilizing more direct measures of neurophysiological control does so without quantifying mechanical joint stability (Baumeister et al., 2008, 2011; Kapreli et al., 2009; Pietrosimone et al., 2012). Therefore, it is difficult to determine how much muscle tone or joint stiffness is normal for that patient, and how coupling between the mechanical joint properties and the PNS and CNS become disrupted. Lastly, research frequently employs study designs that compare unstable joints with the unaffected limb or with a group of uninjured subjects; but, only recently have studies incorporated groups of “copers” who suffer a joint injury but do not develop recurrent instability as a secondary control group (Eastlack et al., 1999; Courtney et al., 2005; Wikstrom & Brown, 2013). This cohort of subjects may be capable of providing valuable insight into mechanisms through which proper healing occurs, or how the nervous system may optimize itself and allow for healthy return to activity.

To understand the mechanisms behind joint instability, it must be restated that episodes of rolling an ankle, the knee “giving-way” or a shoulder subluxation represents a failure to properly regulate joint stiffness (Wilson et al., 1991a). While research exists supporting the role of increased laxity of the capsuloligamentous structures in complaints of joint instability (Lentell et al., 1995; Hubbard et al., 2004), an equal or greater amount of literature to the contrary is also present (Eastlack et al., 1999; Kerkhoffs et al., 2001a). This therefore implies that injury to the static joint stabilizers affect dynamic restraint mechanisms, leading to failures in stiffness regulation and subsequent instability.

## Dynamic restraint mechanism and instability

Many theories regarding altered dynamic restraint focus on the peripheral deafferentation that may occur in capsuloligamentous receptors following injury. The most direct manifestation of this would be a delayed *detection* of an impending episode of giving-way, leading to a delayed muscular response. However, as previously mentioned, proprioception has been inconsistently correlated with history of injury or instability. Furthermore, this proprioceptive deficit may only affect the feedback loop of neuromuscular control, with no change in the equally important feedforward (preparatory) control (Konradsen 2002a; Gutierrez et al., 2009). However, researchers have proposed altered mechanisms

by which peripheral deafferentation may impair feedforward neuromuscular control, and subsequently the planning of future movements. As Johansson et al. (1991) observed a *potent* effect of ligamentous afferents on fusimotor activity and therefore muscle spindle sensitivity, one may recognize how damage to those ligamentous receptors could subsequently decrease fusimotor drive, and therefore amplify sensory deficits and lower muscle tone and stiffness.

### CNS changes following injury

Throughout the evolution of joint instability research, evidence has emerged supporting segmental and cortical changes following joint injury. Some of these earlier findings concentrated on the Hoffman reflex, a combination of sensory and motor activity, measuring spinal excitability and fusimotor function. These data suggest that among unstable joints, this spinal excitability is diminished (McVey et al., 2005; Sefton et al., 2008; Kim et al., 2012). Two proposed mechanisms have been suggested to explain these changes, where decreased afferent activity from the joint receptors lowered fusimotor activity; or decreased cortical drive alters fusimotor function (Sefton et al., 2008; Palmieri-Smith et al., 2009). The use of microneurography (peripheral nerve recordings) has identified that muscle spindle afferents have a decreased response to joint loading among functionally unstable ankles (Needle et al., 2013b). The authors suggested this fusimotor dysfunction was a result of peripheral deafferentation, providing the first direct measurements of a sensory deficit in this population. However, limited research has studied cortical changes among these populations.

Using techniques such as fMRI and EEG, researchers have found discrete alterations among patients with a history of joint injury and instability. Kapreli et al. (2009) found that during simple knee flexion and extension, patients with a history of anterior cruciate ligament injury had an increased dependence on joint sensation and planning for performance of the task when compared with healthy patients, evidenced by increased presupplementary motor area, posterior secondary somatosensory cortex and posterior inferior temporal gyrus activity. Similarly, a pair of studies by Baumeister et al. (2008, 2011) found higher frontal cortex activation among anterior cruciate ligament-deficient knees during proprioceptive tasks, suggesting higher reliance on attentional control and working memory in this subset. This is in line with clinical studies investigating the relationship between neurocognitive function and risk of injury (Swanik et al., 2007). While this research has not been replicated at other joints, studies have investigated changes in the motor cortex among unstable ankles. The studies have found decreases in cortical excitability in patients with ankle instability (Pietrosimone et al., 2012), as well as altered tuning of excitability to laxity

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in this subset (Needle et al., 2013a). These findings suggest that the nervous system is tuned based upon the mechanical properties of the joints, and more importantly this relationship is altered by injury. While cortical research has only recently begun in these patient populations, data suggest that neuromechanical coupling may occur between the joint and nervous system, and ligamentous injury may cause neuroplasticity. Most notably lacking from this line of research is comparisons of cortical changes in the context of alterations in joint laxity to better understand the coupling of these components.

Few studies have incorporated direct neurophysiologic measures while quantifying mechanical laxity. However, those that have integrated these variables are observing links between the PNS and CNS, and joint excursion (Needle et al., 2013a, b). This suggests that innate asymmetries in laxity exist among the uninjured population; however, the nervous system is tuned to that innate laxity. Following injury, several adaptations can occur to disrupt this relationship. For instance, while joint laxity might increase, it may not be pathological; but the nervous system is no longer able to negotiate the increased joint excursion. Alternately, the nervous system might be affected following injury, whether from peripheral deafferentation or inflammatory changes, and therefore the nervous system is no longer calibrated with the joint's mechanical properties (i.e., stiffness). This *neuromechanical decoupling* may explain the highly variable results observed across populations of unstable joints, and could also potentially explain why some patients do not develop instability following injury, as the nervous system activity in “copers” may remain accurately tuned to mechanical constraints of the joint (Fig. 1).

### Treatment

Following injury, a cascade of mechanical, proprioceptive and neuroplastic changes appear to occur on an individual basis, leading some to develop sensations of joint instability. As researchers begin to understand how these changes arise, studies must shift toward clinical recommendations to prevent instability and subsequent disability among these patients. This goal becomes difficult when research cannot suggest any single factor that equivocally changes following joint injury. Rather, it may be more important to evaluate patients on an individual basis for mechanical insufficiencies, sensorimotor and fusimotor changes, and cortical alterations; and tailor treatment programs based on those specific changes.

### Mechanical insufficiencies

Mechanical insufficiencies could potentially be the most difficult to modify following injury, without the use of

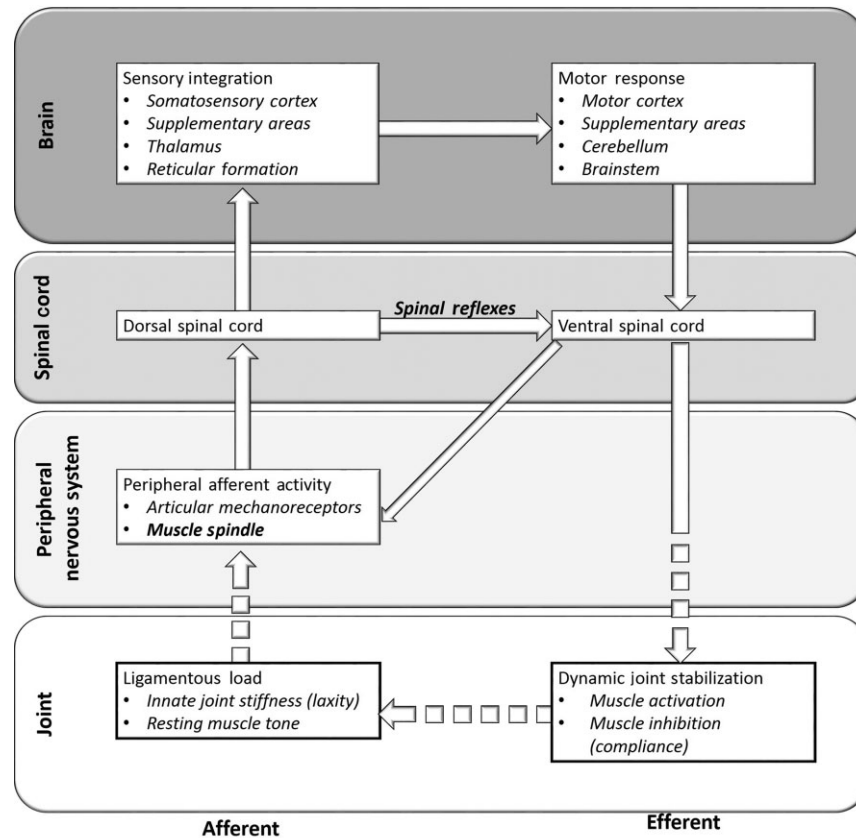


Fig. 1. Sensorimotor pathway from loading of capsuloligamentous structures (bottom left) to stabilization of the joint by the musculotendinous unit (bottom right). Neuromechanical decoupling or dissociations between the structures that provide static and dynamic joint stabilization and the nervous system (dashed lines) contribute to sensations of instability of the joint.

surgical procedures. At the ankle joint, Lamb et al. (2009) has suggested that casting for as long as 3 weeks may provide the best long-term outcomes following joint injury, although evidence suggests this may not be ideal for returning to functional activities (Kerkhoffs et al., 2001b). While reducing mechanical laxity of the joint following initial treatment may only be achievable through surgical intervention, it remains to be determined how other conservative approaches may improve the passive stiffness characteristics of the joint. While both preparatory and reactive muscle contraction is able to alter mechanical stiffness, it remains unclear if muscle tone would be modifiable following injury.

### Functional deficits

In the case that mechanical insufficiencies appear within normal limits, several interventions have been proposed to improve peripheral sensation and neuromuscular control at the joint. Among these techniques are modalities such as joint mobilization (Hoch & McKeon, 2011), stochastic resonance (Ross et al., 2007), massage (LeClaire & Wikstrom, 2012), as well as a variety of training techniques (Fitzgerald et al., 2000; Swanik et al., 2002). However, each one of these may have separate roles in improving neuromechanical coupling and

stiffness regulation, contributing toward improved outcomes among unstable joints. Benefits of joint mobilization include improved range of motion in the joint as well as improved balance, potentially through an increase in afferent input toward the fusimotor system contributing to better proprioception (Hoch & McKeon, 2011), although it is unclear if these effects last over a prolonged treatment protocol (Hoch et al., 2014). Ross et al. (2007) has incorporated similar principles in the treatment of functional ankle instability, by utilizing stochastic resonance – subthreshold vibratory stimulus that would serve to enhance the transmission of mechanoreceptor activity and subsequently improve afferent feedback from the joint. This technique has demonstrated effectiveness in addressing balance deficits among patients with functional ankle instability (Ross & Guskiewicz, 2006; Ross et al., 2007). Similar improvements have been observed using a massage intervention that could serve to enhance tactile feedback at the joint, although proprioceptive improvements were only observed with visual feedback, making it unclear at which level of the nervous system this would be effective (LeClaire & Wikstrom, 2012).

While modalities demonstrate promise in correcting specific patient deficits, certain training techniques may serve to better correct long-term deficits. While strength



training is traditionally included into rehabilitation protocols, their effectiveness in correcting proprioceptive deficits is limited (Smith et al., 2012). Strengthening exercises have been effective at improving stiffness at the shoulder joint, but it is unclear if this carries over to the ankle and knee joints (Laudner et al., 2013). Rather, current recommendations indicate balance and perturbation training, or plyometric training may better serve these populations. Balance and perturbation training are tied to improved detection of joint loading secondary to altered Golgi tendon organ and muscle spindle sensitivity (Fitzgerald et al., 2000), while plyometric training would act to limit the inhibitory effect to the Golgi tendon organ and subsequently improve proprioception (Swanik et al., 2002; Chimera et al., 2004). While each of these has been observed to improve symptoms among patients with joint instability at the ankle, knee and shoulder; they would best serve among patients with evidence of decreased proprioception and neuromuscular control at the joint (Swanik et al., 2002). Only recently are cortical changes following injury being discussed in the literature (Kapreli & Athanasopoulos, 2006), but studies have not yet evaluated whether the aforementioned techniques would act to “recalibrate” the cortex to the joint’s mechanical properties or whether tasks requiring higher cognitive loads would be required to initiate changes in the CNS.

### Perspective

As muscle tone and joint stiffness remain misunderstood and understudied in sports medicine research, it is important for clinicians to consider these factors in the context of injury in order to appropriately guide the diagnosis and treatment of joint instability. The PNS and CNS both serve to alter fusimotor control surrounding the joint, providing a further link between joint injury and the nervous system (Burke, 1983). Morphological and neuroplastic changes have both been reported among

patients with joint instability, although these changes appear to be variable. With growing evidence describing neurophysiologic changes in response to therapeutic interventions and training techniques, clinicians should better individualize treatment following joint injury.

Joint instability is a multifaceted problem that may include alterations to joint laxity, peripheral sensation, neuromuscular control and cortical alterations. However, changes to any of these may be present in varied magnitudes, and therefore patients should be assessed on an individual basis. Several links have been observed that may predict the stiffness of a joint through measurement of nervous system parameters. Evidence is emerging and suggesting that neuromechanical decoupling may occur following injury, where some patients exhibit mechanical changes for which the nervous fails to adapt; while others have a nervous system that has adapted while the joint remains mechanically stable. Research must continue to utilize direct neurophysiologic techniques in conjunction with measures of mechanical function to truly understand this relationship, as well as incorporating these direct neurological measures with treatment interventions to individually guide each patient.

Several factors remain unknown as research continues to investigate this highly variable pathology. While links are consistently observed between joint stability and the PNS and CNS, it is unclear how these factors are coupled in a normal population, limiting our understanding of how these factors become decoupled following injury. The brain remains a new frontier for joint instability research, and its continued investigation could drastically increase our knowledge of this pathology, as could a group of previously injured subjects that have not developed instability (copers). Including these subjects in future studies is crucial to understanding, preventing, and treating this phenomenon.

**Key words:** joint instability, neuroplasticity, muscle spindle, corticospinal excitability.

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