Active and passive characteristics of muscle tone and their relationship to models of subluxation / joint dysfunction Part II

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The relationship of muscles to the causes and effects of the pathophysiologic entity referred to as chiropractic subluxation or joint dysfunction is critical. Part I of this paper reviewed the complexities of skeletal muscle in regards to anatomy, active and passive tone, detection of muscle tone, neurophysiology, and how muscle function fits into a variety of subluxation/joint dysfunction models. The concluding part of the review culminates in a hypothesis to describe and explain varying degrees of muscle tone that may be encountered clinically. It is hoped that knowledge of the differing levels of muscle tone and their causes will help the clinician to better determine the underlying cause of a neuromusculoskeletal problem allowing application of necessary and proper intervention.

Introduction
In Part I of this article (September issue), the anatomy and physiology of muscle function was reviewed with the purpose of establishing the cause, types, gradations and detection of skeletal muscle tone. In Part II, this back-ground in muscle physiology is overlaid on a variety of mechanisms that have been proposed to explain joint dysfunction. Finally, a guide to various types and causes of altered muscle tone that may be experienced in clinical practice is presented.

Les muscles jouent un rôle essentiel dans les causes et effets de l’entité physiopathologique couramment appelée « subluxation » ou « dysfonctionnement des articulations ». Dans la première partie du présent article, on a examiné la complexité des muscles squelettiques quant à l’anatomie, à la tonicité active et passive, à la présence de tonicité, à la neurophysiologie et au fonctionnement des muscles dans une variété de modèles de subluxation ou de dysfonctionnement des articulations. La conclusion de cet examen aboutit à une hypothèse pour décrire et expliquer les divers degrés de tonicité musculaire pouvant être reconnus sur le plan clinique. Nous espérons qu’une connaissance approfondie des divers degrés de tonicité musculaire et de leurs causes aidera le clinicien à mieux cerner les causes sous-jacentes des troubles neuromusculosquelettiques afin de permettre l’intervention nécessaire et adéquate.

KEY WORDS : skeletal muscle, muscle tone, subluxation, joint dysfunction.

MOTS CLÉS : muscle squelettique, tonicité musculaire, subluxation, dysfonctionnement des articulations.
Functional characteristics of muscle tone in models of joint dysfunction

The following section examines how the varying properties of muscle physiology may be related to myopathology in differing models of joint dysfunction, which include: facilitation, central sensitization, flexor/nociceptive reflex, pain-spasm-pain, gamma loop, sub-maximal contraction, thixotropy and post contraction sensory discharge.

Facilitation/Korr-Denslow hypothesis

This is a neurological reflex based theory whose salient points are based on the premise that unbalanced, abnormal afferent signals enter the spinal cord and cause an increase in segmental neural activity, including active α-motoneuron firing, and which result in reflexive muscle hypertonicity. Input from the now activated muscles then reflexes into the cord, exacerbating the feedback loop. Korr proposed that the segmental spinal motoneuron excitability was the result of the afferent output from muscle spindles.

Central sensitization

In this theory, an injury that causes nociceptive signals can produce increases in muscle tone that are maintained long after the nociception ceases. These findings led to a theory of spinal cord plasticity now called central sensitization, or long-term potentiation. The effect of central sensitization is to reduce the associated segmental neural thresholds prompting increased excitability and summation of the α-motoneuron(s) to threshold. Such a spinal learning mechanism has been suggested as a potential basis for osteopathic somatic dysfunction and chiropractic subluxation. It has been theorized that chronic, repetitive joint dysfunction is caused by some injury long ago whose afferent stimulation led to plastic changes in the spinal cord leading to a “neural scar”. Stimulation of this sensitized area leads to reflexive muscle contraction and signs of dysfunction.

The source of afferent input leading to central sensitization has not been adequately defined. In a recent paper, the authors write: “It is not clear how central hyperexcitability is maintained and eventually causes chronicity, but most likely an outgoing nociceptive afferent barrage is needed”. Slosberg provides a review of central sensitization and how this phenomenon may relate to joint dysfunction.

Flexor or nociceptive reflex response to injury

Wyke, as reported in a review by Slosberg, found that activation of nociceptors by irritation of the joint capsule generated an intense, non-adapting muscle hypertonicity to guard the joint. The musculoskeletal reflexes triggered by nociceptive input are termed “nocifensive reflexes”. The prototypical nocifensive reflex is the flexion reflex, in which stimulation of nociceptors causes muscular contraction – less than a spasm – of the flexor muscle and inhibition of the extensor muscle about the associated joint. Woolf and McMahon found that the prolonged nociceptive stimulation changed the reflexive muscular response such that, “Essentially, the high-threshold phasic flexor withdrawal response has become a low-threshold tonic one”. This change in muscular response was thought to be due to “a remarkable degree of functional plasticity of the system”. A group of interneurons in lamina 4–6 of the dorsal horn of the spinal cord has been shown to encode the muscular contractions in nociceptive reflexes, and this may be where plasticity of the nervous system occurs.

Muscular reactions to nociceptive input may also be learned and stored in the cerebellum. Kottke has suggested that movement is based upon the development of cerebellar engrams that are acquired through learning. The nocifensive reflex can be a conditioned reflex, and the muscular reactions to a nociceptive stimulus have been shown to be learned by, and stored in, the cerebellum. Engrams occurring at the reflex level contain the codes for exciting and inhibiting the appropriate muscles. Such findings give support to a hypothesis that the cerebellum learns nocifensively induced muscle reactions to acute joint dysfunction, and could account for a relatively constant misalignment pattern.

There is much evidence to show that the effect of injury to the mechanoreceptors of a joint can cause loss of awareness of joint position (loss of kinesthesia). McLain, for example, found few mechanoreceptors in the facet capsule of the joints in the cervical spine, and commented that the loss of any of these receptors could lead to denervation of the joint with the potential for loss of protective muscular reflexes. This loss of kinesthetic sense allows the joint to be moved into positions that, prior to injury, protective muscle contractions would have prevented. In an injured joint this feedback system loses sensitivity, and the joint can be forced into harmful (nociceptive) positions. Should this happen, the memory stored in the spinal cord inter-
neurons or cerebellar engram reacts with the pattern of muscle contraction learned from the prior nociceptive event. Hence, a pattern of joint dysfunction or postural distortion could be established.

Whether flexor or nociceptive reflex muscle contractions can be maintained for long periods of time is controversial. Animal research indicates that a prolonged pathological nociceptive input from deep tissues abolishes the capability of muscle to facilitate the flexor reflex for extended periods of time.21 A further problem with the flexor reflex model is that, in back pain, hypertonicity is often in the extensor muscles, not flexor muscles. Mense suggests that the activation of the γ-motor system by muscle nociceptors must be postulated to account for extensor hypertonicity.22

**Pain-spasm-pain positive feedback loop**
The “vicious cycle” hypothesis of pain and muscle tone was proposed by Travell in 1942 who wrote, “According to this view, limitation of motion is primarily a reaction to pain rather than the result of structural lesion. If muscle spasm causes pain, and pain reflexly produces muscle spasm, a self-perpetuating cycle might be established ...”.23 Roland provides a review of evidence for the pain-spasm-pain cycle, arguing very generally that pain can cause muscle spasm and that muscle spasm can cause pain.24 However, subsequent to that review there have been several studies examining what happens when a painful stimulus is applied to a muscle. While a linkage between muscle pain and increased muscle spindle sensitivity has been found, there was no effect of muscle pain on α-motoneuron excitability.25 In their review, Matre et al. found that “… a large number of well-controlled studies have shown no statistical significant difference in resting EMG activity between painful and non-painful muscles”.26 Indeed, a painful, inflamed muscle has been found to have lower than normal tone.27

If a muscle becomes hypertonic – spasmed – as a reflex, that spasm, while it may or may not be painful, does not seem to cause a positive feedback loop that results in further spasm. Roland points out that if the pain-spasm-pain theory is correct, eliminating pain with analgesics or muscle spasm via biofeedback, physical therapy, or muscle relaxant drugs should interrupt the putative feedback loop.24 Although this type of positive feedback loop may be active in some cases of musculoskeletal pain, many patients seeking chiropractic/manipulative evaluation have already been treated using methods that should abolish this hypothetical cycle, presumably without effective relief of their complaint.

**Gamma loop**
Recently, another type of feedback loop has been proposed and investigated with a series of experiments.25,28–31 Active muscle contraction generates metabolites of fatigue, including bradykinin, which stimulates group III and IV chemoreceptive nociceptors, or metaboreceptors.32 Group III and IV metaboreceptors synapse with and excite γ-motoneuron cells. Depolarization of γ-motoneurons excites homonymous muscle spindles which causes an increase in Ia and II output. The Ia and II input to the cord then stimulates α-motoneurons to summation causing further muscle contraction generating more metabolites, completing the positive feedback loop. Any increased activity in secondary (II) spindle afferents, which project back to the gamma system, constitutes a second positive feedback loop which may perpetuate the muscle contraction in absence of the group III and IV input.29 Interestingly, as mentioned previously, there is incomplete cortical control of the static bag and chain spindle fibers,33 which give rise to the secondary (II) spindle afferents. Once a static gamma → spindle II → static gamma positive feedback loop has been created, cortical inhibitory signals may not be able to break the cycle.

Considerable research has demonstrated evidence for this mechanism28,29,34–41 however, some has not.42,43 It may be worth noting that the two studies with negative results were done on the lumbar spine muscles of the cat, whereas most of the other studies were conducted on muscles of the head and neck. Perhaps the concentration of γC spindles in the cervical spine and their predominant association with type II afferents is a factor in the conflicting findings. Further information regarding the gamma positive feedback loop hypothesis as proposed by Johansson/Sojka can be found in a recent review.44 Muscle stiffness associated with ischemia and fatigue – the establishment of a gamma positive feedback loop – would result in lowered nociceptive thresholds to mechanical stimulation.45 This has been confirmed in studies done on some muscle in the human using the chemicals of ischemic contraction.46,47 If lowered mechanical pain
thresholds occur with spinal joint dysfunction the associated paraspinal muscles would be painful to touch. Palpation for abnormal pain thresholds has been shown to be a reliable characteristic of joint dysfunction.48–50

The gamma positive feedback loop itself has spinal cord connections by which a central sensitization phenomenon may occur. There are neurons connecting to the group II/γ-motoneurons synapses which release noradrenaline (NA) and serotonin (5-HT). NA strongly depresses synaptic actions of group II afferents on gamma motoneurons thus effectively depressing the positive feedback from group II afferents to the gamma motoneuron. 5–HT has the opposite effect – to enhance activity of γ-motoneurons.51 Jankowska and Gladden conclude, “Changes in the balance between opposite effects of NA and 5–HT releasing neurons on γ-motoneurons may be used to adjust the effectiveness of positive feedback to these neurons to the needs of different movements”.51

According to Pedersen et al., “The [gamma loop] hypothesis implies that sustained muscle contractions, inflammation and/or ischaemia may lead to activation of chemosensitive group III and IV muscle afferents which increases the stretch sensitivity and the discharge rate of muscle spindle afferents. If the excitatory load on the γ-muscle-spindle system is high enough, as a result of a massive input from, for instance, group III and IV muscle afferents due to reduced muscular circulation in combination with inflammation and/or muscle contractions, this may turn the system into a ‘vicious circle’”.28 This positive feedback loop could then become the source of the “afferent barrage” required for central sensitization.8 Over time, the spinal cord neural influence on the group II/γ-motoneuron synapse is altered to favor the excitatory action of serotonin (5–HT). This creates a central excitatory state able to enhance the influence of any signal from spindle group II afferents. This central excitatory state would remain in effect even if the initiating event and the ensuing positive feedback loop has been eliminated. Such a central, lowered threshold to re-establishing the positive feedback loop (causing muscle hypertonicity) could account for recurrence of joint dysfunction at the same joint. If this hypothesis proves to be true, a complete correction of spinal joint dysfunction would involve breaking the segmental positive feedback loop and keeping it from recurring during the time it takes for the spinal cord serotonin neurons to shift back to their normal – pre-positive feedback loop – state. Speculatively, these goals may be facilitated by having the patient rest post manipulation and through the use of rehabilitation techniques.

Along with these potential positive feedback loops, muscle fatigue has also been shown to dramatically decrease the inhibitory afferent input to the α-motoneuron pool from Golgi tendon organ receptors.52 This cycle of fatigue, gamma stimulation, alpha stimulation and Golgi inhibition has been forwarded as a hypothesis to explain exercise associated muscle cramps.53

While nociception from a muscle due to tissue injury or inflammation leads to inhibition of that muscle,27 nociception from muscle ischemia and the products of metabolism may indeed lead to positive feedback loop-induced hypertonicity. This positive feedback loop would stabilize the muscle being hypertonic yet in dynamic equilibrium with its vascular supply. This dynamic equilibrium would likely be at or about the critical force of the muscle, a concept discussed next.

Sub-maximal contraction

In most cases of active muscle contraction and hypertonicity, the decisive factor for the occurrence of fatigue and pain seems to be the vascular environment of the muscle. A muscle will compress its own blood vessels if it contracts with a force above approximately 30% maximal contractile force.54 40% in back extensor muscles.55 In lower contraction forces, fatigue is not as certain.

α-motoneurons appear to fire in order of increasing size. Input to the motoneuron pool first excites to threshold the smallest, most excitable α-motoneurons connected to slow-twitch, fatigue-resistant type I muscle fibers.56 Muscles, particularly postural muscles, are composed predominantly of type I fibers, which, as we have seen, are suited for sustained contraction. One study suggested that during sub-maximal contraction – less than 40% maximum voluntary contraction (MVC) – of the erector spinae, the metabolic effects of fatigue may be counteracted or delayed by the rotational recruitment of additional motor units. Such a compensatory mechanism may reflect a functional requirement of the back muscles to maintain static “postural” contractions for long periods of time.57 The phenomenon of rotation of slow twitch (type I) motor unit contraction may help explain the ability to sustain low-level active contraction.58

All of the above factors may be responsible for what is
called the critical force of a muscle. Critical force is around 15 to 20% of a maximum voluntary contraction (MVC), and is that point below which an isometric contraction can be maintained for a very long time without fatigue. To get a feel for this level of muscle contraction, a 10% MVC is approximately the force used to maintain the arms in a horizontal working position. The critical force point of a muscle is a controversial concept. Others have found that despite adequate blood flow, in long term contractions, a muscle can become fatigued although not exhausted. Figure 1 provides a graphic representation of active muscle contraction relative to intramuscular blood flow.

Related to submaximal contraction is the so-called “Cinderella Hypothesis” (referring to Cinderella who worked continuously) proposed by Hagg in 1991. This hypothesis states that type I fiber motor units are at risk of overload in conditions of low level, prolonged and sustained activation which causes fiber damage and pain. The hypothesis is based on the neurophysiological finding of ordered recruitment of motor units (Henneman principle) with increasing force and the finding of abnormal morphological characteristics in trapezius myalgia. There has been some research supportive of this hypothesis. Kadeffors et al. suggests the possibility that sustained, monotonous contraction may result in a reduction of the neurophysiological activation threshold, making eventual relaxation of the muscle difficult to achieve. If muscle activation is kept below the critical force level, there is no metabolic (lactic acid, bradykinin) build-up to activate nociceptors to cause immediate pain despite the sustained contraction. Repetitive long-term contraction, however, may lead to physical damage of the muscle fibers resulting in pain.

Mense and Stahnke found that while contractions of moderate force are an effective stimulus for some muscle mechanoreceptors (group III and IV) another sub-population of these slowly conducting afferents is activated during ischemic work. This second population of group III and IV receptors (metaboreceptors), “… is not excited or is only weakly excited by contractions without ischemia.” Pushing a muscle to a level of contraction past the critical force and into an ischemic contraction would generate enough metabolites to depolarize group III and IV metaboreceptors and be experienced cortically as pain.

Impaired muscle circulation has been found in cases of chronic neck and shoulder pain, is suspect in low back pain, and has been shown to occur when spinal biomechanics change from lordosis to kyphosis. Ashton-Miller et al. quote a study which showed that muscle contraction at a level of as little as 4% MVC combined with work loading caused pain and significant increases in sick leave due to musculoskeletal complaints.

Sub-maximal contraction on the border of ischemia
may also lead to decreased performance of muscle fatigue characteristics.71–74 Mannion, in reviewing various causes of back pain writes, “… the evidence implicating highly fatigable back muscles in the development of low back pain is somewhat more substantial”.75 While the underlying cause(s) of the abnormal fatigability in back pain have not been adequately described, chronic sub-maximal contraction resulting in limited blood flow affecting muscle performance is seen as a possibility. McGill notes that, “… complete relaxation of the low-back muscles is necessary to avoid compromising performance and an increased risk of musculoskeletal disorders”.72

Additional indirect evidence for the hypothesis of long term, sub-maximal contraction is the finding by Roland that, on balance, chronic back pain patients have increased spinal muscular activity at rest or following exercise, but reduced muscle activity during spinal movement.24 When at rest, muscle contraction at or just above the critical force level would not be opposed by stretching or movement that would promote intra-muscular blood flow. Inactivity would allow for build-up of metabolites, activation of metaboreceptors, and be experienced cortically as pain. Exercise would tend to quickly fatigue these already contracted muscles, also leading to ischemia and pain.76

A critical force contraction phenomenon may also be responsible for the long-term muscular hypertonicity associated with the asymmetric tonic neck reflex.77 This postural reflex causes changes in muscle tone that do not attenuate as long as the reflex is left alone.77–79

Finally, the stimulation of type III and IV metaboreceptors with the by-products of muscle metabolism has been shown to cause changes in skin blood flow patterns,80–83 increased heart rate,84–86 blood pressure,85,86 rate of breathing,80 and renal86 and splenic86 sympathetic nerve activity. All of these reflexive effects in response to metaboreceptor stimulation work to increase blood flow to the ischemic muscle(s), and are elements of what is known as the pressor reflex.

Given the intimate connection of muscles to joint dysfunction, one might then expect that manipulation would be found to have an effect on some of the noted physiological effects of metaboreceptors. Although no direct link between manipulation and a decrease in metaboreceptor activity has been studied, there have been many studies, some controlled and blinded, showing a correlation between manipulation and a significant decrease in blood pressure.87–90 Study of other physiological effects of metaboreceptors and manipulation for joint dysfunction may be fruitful areas of investigation.

Thixotropy

As was noted previously, there is a tendency for the actin and myosin filaments to stick together when inactive for a period of time. If a muscle has been lengthened or shortened for a period of time, cross-bridges form at that length and/or tone, changing the resting properties of that mus-

Figure 2  Point A is normal resting muscle tone. As the muscle actively contracts (alpha excitation) muscle tone increases. From point B to C the muscle is being held in an actively contracted state. At point C, alpha excitation stops, and the muscle fibers relax. At point D, the muscle tone stops decreasing due to cross-bridges in the muscle fibers remaining in their prior contracted position. This is muscle – intra- and extrafusal – thixotropy. Note the muscle tone is increased despite no active alpha excitation. A rapid stretch of the muscle, point E to F, brings the muscle tone back to its normal resting value.
cle. Because muscle thixotropy depends on passive muscle physiology – the resting positions of actin-myosin cross-bridges – a thixotropic muscle may be hypertonic without EMG activity. In the standing human, the center of gravity of the upper body is anterior of the ankle joint, suggesting that some constant force in the gastrocnemius or soleus muscle is required to counter the forward moment and prevent toppling. Basmajian and Deluca, however, noted that the posterior calf muscles are electromyographically silent on quiet standing, except for occasional bursts of corrective activity. Simon and Mense used the passive thixotropic property of muscle to account for the non-contractile forces that must be generated by the calf and other postural muscles. They estimate that the thixotropic bonds formed when a muscle is actively contracted, then relaxed, can stiffen the muscle up to ten times its normal resting tone.

How muscle thixotropy could be related to the pathophysiology of joint dysfunction is not difficult to imagine. Nociception may cause a slowly fatiguing reflexive muscle contraction. When the nociceptive input and active contraction cease, the reflexively contracted muscle may not return to a normal relaxed length and tone, but remain in a shortened, hypertonic state due to the cross-bridge bonds formed during the previous contraction.

Clinically the effect of thixotropy on the resting length and/or tone of muscles is largely unexplored. One report has presented evidence that the cause of lagophthalmos (incomplete closure of the eyelid) in facial nerve palsy (e.g. Bell’s Palsy) is due to thixotropy of the levator palpebrae muscle. Treatment consisted of passive closure of the affected eyelid followed by manual stretching of the upper eyelid in a downward direction as far as possible, stretching the muscle to break down the “stuck” cross-bridges. Other researchers speculate that “limbering-up manoeuvres commonly employed by athletes, dancers and physiotherapists”, involve the loosening of the thixotropic muscle (intra and extrafusal) bonds that have been re-set due to prior contraction. Others find that passive and active stretching does not seem capable of re-setting the “hung up” intrafusal fibers. In these cases, sudden stretching of sufficient velocity and magnitude break down the re-formed intrafusal fiber cross-bridges. Adjustment may provide the sudden stretch of sufficient velocity and magnitude to re-set thixotropic muscles.

**Post contraction sensory discharge**

During a contraction of extrafusal muscle fibers, related muscle spindles are temporarily unloaded, and Ia and II discharge decreases. With Ia and II input to the spinal cord inhibited, the gamma efferent signal to the muscle spindles is increased; Matthews calls this “automatic gain compensation”. This gain compensation is used to establish a new “normal” muscle length and/or tension or “fusimotor set”.

Greater γ-motoneuron activity causes increased (over the normal, resting muscle) Ia and II spindle output. Experiments in cats have shown that in sampled Ia fibers, discharge rates after muscle contraction had increased by 60%, and a number of these receptors had been silent prior to the contraction. This increased Ia and II muscle spindle sensory output is known as post contraction sensory discharge (PCSD). The PCSD phenomenon allows for continued muscle tension, shortened length, and mostly Ia (with some II) spindle output.

Eldred, Hutton and Smith provide a concise summary of the effects of PCSD:

1. A persisting increase in discharge at a maintained muscle length and in response to stretch appears after a muscle has undergone contraction, under the condition that the efferent background is otherwise quiet.
2. Fusimotor activation alone, i.e., contraction of intrafusal fibers, can produce this effect, though extrafusal contraction also seems to contribute.
3. The enhanced discharge, as it was recorded, arises to a major extent in Ia afferent fibers.
4. The cause of the effect is probably mechanical in nature.

The mechanical nature of PCSD is thought to be due to re-setting of the intrafusal actin-myosin cross-bridges in the contracted/lengthened muscle. Given that kinesthetic sense is almost exclusively from muscle spindles enhanced muscle spindle output via PCSD might be expected to alter muscular and/or postural balance. PCSD as an hypothesis to explain the myopathy in joint dysfunction and the effects of manipulation was first proposed by Buerger in 1983 who wrote, “... because post-contraction sensory discharge can be relieved by sudden stretching and/or sagging muscles, it is also an attractive heuristic model for the physiologic mechanisms which may underlie many forms of manual therapy”.

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A similar mechanism of spindle dyregulation, where the intrafusal representation of the extrafusal muscle length no longer matches the actual length, was proposed by Donaldson\textsuperscript{111} to explain the rapid recovery of patients with chronic back pain.\textsuperscript{112} Rapid response of patients with chronic back pain to manipulation has also been reported.\textsuperscript{113} Because the recovery in these chronic cases was so rapid, degenerative muscle changes are not likely to be involved and the mechanism appears to be neurological rather than muscular in nature. The post contraction sensory discharge phenomenon involving “hung up” intrafusal fibers after long term contraction or stretch is a similar “dysregulation” leading to chronic but reversible increases in muscle tone.

We have noted that neural commands and feedback depend on muscle spindle signals,\textsuperscript{109,114} and muscle hypertonicity has been associated with evidence of increased spindle output.\textsuperscript{115,116} The phenomenon of PCSD could explain what Panjabi had noted regarding spinal stability. “The active musculoskeletal subsystem may develop deterioration of its ability to receive and/or carry out the neural commands, to provide accurate feedback of muscle tension information to the neural control unit, or to produce coordinated and adequate muscle tensions”.\textsuperscript{117} This loss in the integrity of the stabilizing capacity of the spinal support system could be a precursor to back pain.\textsuperscript{117,118} Panjabi writes, “One example of the kind of error that might occur is that one or more muscles may fire in a manner that is undesirable; too small or too large forces and/or too early or too late firing. This may happen either due to the faulty information transmitted from the spinal system transducers or due to the fault of the control unit itself. Such an error may cause excessive muscle tension, resulting in soft tissue injury and pain. This may explain some of the instances of acute low back pain initiations where negligible or marginal loads are involved (e.g., while picking up a piece of paper from the floor)”.\textsuperscript{117}

No one particular model of joint dysfunction and its muscular component has reached consensus. One, some,
Muscules and Joint Dysfunction

Somatic Injury
Joint Injury
Visceral Pain

Acute flexor reflex contraction
Asymmetric/restricted motion

Motoneuron Pool

Inhibition wins out

Excitement wins out

Increased intramuscular pressure
Activation of type III and IV nociceptors/metaboreceptors

Equilibrium of muscle demands
and vascular supply established

Hypertonicity after attenuation of nociception
Post contraction sensory discharge
Thixotropy
Pain due to ischemia

Residual hypertonicity
Muscle wasting due to ischemia, pain
Transformation from type 2 to type 1 fibers
Fatty infiltration

Acute HVLA stretch decreases intramuscular pressure, resets extra and intrafusal cross-bridges

Manipulation, Strengthening Rehabilitation

Figure 4 The characteristics of muscle function and their relationship to joint dysfunction and manipulative therapy.
or none of the above models may be correct. It is possible, if not likely, that more than one model involving active and passive aspects of muscle tone will characterize joint dysfunction. Perhaps, for example, nociceptive muscular reflexes predominate in the acute phase of dysfunction with ischemia, mechanoreceptor, thixotropic and sub-maximal contraction mechanisms in chronic phases. Figure 4 shows a flow chart outlining the varying characteristics of muscle function and how they may relate to joint dysfunction.

**Altered muscle tone – clinical application**

Given the complex inter-relationship of muscle physiology and joint dysfunction, we would propose the following hypothetical assignment of abnormal clinical muscle tone variations and associated findings. It is important to note, especially in acute presentations, that hypertonicity of a muscle is due to some factor other than an injury/inflammation in that muscle; injured muscles are inhibited.27 As Mense noted, teleologically, inhibition induced by myositis would be an advantage, because it could reduce the forces acting on the damaged muscle.119 Involuntary muscle hypertonicity is more often a secondary reflex reaction to other injury, or, potentially a sign of primary pathologic dysfunction. Also, each of the below variations of muscle tone might lie on a continuum from mild to severe.

- **Full muscle spasm hypertonicity** – Full active muscular contraction. Spasmodic torticollis and other dystonic syndromes, cramps, antalgic reactions. Some therapeutic intervention to reduce the spasm may be indicated, although full contraction cannot be maintained for very long due to ischemia and fatigue.

- **Reflex spasm hypertonicity** – Active contraction of muscle due to reflex reaction. For example, a nocifensive or flexor reflex as a splinting response to joint/disc injury. Reflex hypertonicity results in muscle tension less than maximal spasmodic contraction. If the reflex contraction is sustained, ischemia and pain from the muscle itself will result. Movement often aggravates reflex hypertonicity. This kind of hypertonicity could be seen in acute injury and joint dysfunction.

If the nociceptive input from the initiating injury fades rapidly, the hypertonicity would decrease, perhaps leaving a passive shortened, hypertonic muscle via thixotropy (and the effects of PCSD). The reflex spasm hypertonicity may lead to the establishment of a gamma positive feedback loop, maintaining increased muscle tension and increased spindle output. Muscle stretch, via manipulation or other methods, post nociception could re-set the contracted muscle to a normal passive tone by re-establishing coordination between the intra- and extrafusal fibers. The gamma feedback loop reaction to chronic contraction is plausible for type I fiber and spindle rich postural muscles, especially those rich in b2c spindles. Larger, multi-joint spanning muscles with fewer fatigue resistant fibers exposed to chronic contraction may develop trigger points. In chronic cases of reflex spasm, degenerative changes – muscle wasting, fiber transformations, elastic component transformations - may require muscle strengthening and rehabilitation.

- **Critical force hypertonicity** – Active muscle contraction at a level of hypertonicity where there is a dynamic equilibrium of muscle demands and vascular supply. On one side of the dynamic equilibrium where the vascular supply is adequate, muscle hypertonicity may cause postural abnormalities but no immediate pain. On the other side of the area of equilibrium, contraction with ischemia causes pain and fatigue. This kind of hypertonicity would often be temporarily relieved by movement/stretching, but aggravated by inactivity, such as sleeping, where there is no resistance to the chronic muscle contraction. Any contractile effort which engages the muscle past the critical force point would also cause pain (similar to the leg pain in intermittent claudication). Such hypertonicity could be responsible for non-fatiguing muscular hypertonicity seen in pathologic tonic neck reflexes and result in long term postural distortions such as pelvic torsion, leg length alignment asymmetry, loss of lordosis, spinal curvatures, etc. Full spasm and reflex spasm muscular contractions may end in this type of active sustainable hypertonicity via gamma positive feedback loops.

While this type of hypertonicity can last indefinitely, any biomechanical problems caused by postural distortions could lead to acute joint dysfunction and reflexive spasm hypertonicity. Critical force hypertonicity could be the result of reaction to chronic stimulation of pathologic reflexes, and the source of this abnormal stimulation should be sought. Ischemia has been shown to cause muscle wasting and muscles that have been in critical force contraction may need rehabilitation.
• **Thixotropic hypertonicity** – Thixotropic properties involve resetting of muscle towards a shortened length and increased tone. There would be no muscle pain due to its passive non-contractile nature. Thixotropic hypertonicity may be caused by assuming awkward postures, or may be the final, sustainable outcome of a reflexive spasm. Thixotropic hypertonicity results in a loss of normal ROM that could become acutely painful and may be responsible for transient joint fixation that occurs in everyday life. This type of muscle hypertonicity can be acute or chronic, and may be eliminated by exercise, stretching, massage as well as high velocity manipulation.

• **Hypotonicity** – An inability of the muscle to produce force or the unwillingness to contract. Hypotonicity can be due to the influence of facet joint stimulation, joint injury, muscle inflammation or tonic neck reflexes

**Conclusion**

The muscular system is characterized by complexities of anatomy and physiology allowing for extreme plasticity to meet changing conditions. Differing levels of muscle tone, causes of increased and decreased tone, effects of tone changes, types of muscle fibers and spindles, degrees of hypertonicity and the time frame of tone changes are all variables in somatic dysfunction. Muscle physiology and attendant neurophysiological reflexes seem to explain, at least in part, some of the signs and symptoms of the musculoskeletal component of joint dysfunction as seen in clinical practice. Using knowledge of the differing levels of muscle tone and their causes will help to determine the cause of a musculoskeletal problem and allow for a better application of intervention. Hopefully this overview, with its condensation of a wide array of information, has provided a glimpse into the complicated workings of the muscular system and has stimulated thinking that will further research. Such direct clinical research will aid in the diagnosis, analysis and treatment of patients.

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Muscle tone


