

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

Part I

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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 reviews 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 review culminates in Part II with 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 neuro-musculoskeletal problem allowing application of necessary and proper intervention.

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

La relation entre les muscles et les causes et effets de l'entité physiopathologique renvoyant à une subluxation ou à un dysfonctionnement des articulations s'avère particulièrement critique. La première partie de ce texte examine la complexité des muscles du squelette au regard de l'anatomie, de la tonicité active et passive, de la détection de la tonicité musculaire, de la neurophysiologie et du fonctionnement des muscles par rapport à une variété de modèles de subluxation ou de dysfonctionnement des articulations. Dans la deuxième partie du texte, une hypothèse est formulée pour décrire et expliquer les divers degrés de tonicité musculaire qui peuvent être rencontrés en clinique. Il est à souhaiter que la connaissance des différents degrés de tonicité musculaire de même que leurs causes aideront les cliniciens à mieux déterminer la cause sous-jacente au problème neuromusculaire du squelette, permettant ainsi d'appliquer une intervention nécessaire et appropriée.

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MOTS CLÉS : muscle du squelette, tonicité musculaire, subluxation, dysfonctionnement des articulations.

Introduction

The position paper of the Association of Chiropractic Colleges (ACC) defines subluxation as a complex of functional and/or structural and/or pathological articular changes that compromise neural integrity and may influence organ system function and general health. [Note: In order not to be repetitive, we will use the term joint dys-

function as an inclusive synonym for chiropractic subluxation, osteopathic lesion and/or somatic dysfunction.] Joint dysfunction is characterized by findings of misalignment, relative fixation, loss of normal range-of-motion and end-play, tenderness, and tissue texture abnormality.¹ Most of these characteristics of joint dysfunction can be mediated by the muscular system. The functional association of

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muscles with joint dysfunction has roots as far back as the historical “Green Books,” a series of 39 volumes published by Palmer College from 1906 to 1961. In volume 14, Stevenson states, “the muscles are the means by which subluxations occur”.² This was restated well by Schneider who wrote, “Bones are inert structures; their alignment in space and segmental range of motion are determined by active muscle contraction and soft tissue length”.³

Many hypothetical models of joint dysfunction postulate, as part of their mechanism, changes in muscle tone. These models include facilitation,⁴ central sensitization,⁵ flexor or nociceptive reflex,⁶ pain-spasm-pain cycle,⁷ gamma loop,⁸ thixotropy,⁹ and post contraction sensory discharge¹⁰ and will be examined in Part II of this article. Muscles are such a critical component of neuro-musculoskeletal dysfunction that some “manipulative” techniques focus almost exclusively on the detection and correction of muscle dysfunction. These include such techniques as receptor-tonus or Nimmo, trigger point, proprioceptive neuromuscular facilitation, muscle energy, post-isometric relaxation, and “spray and stretch”. Many of these techniques are available to the chiropractor as adjunctive procedures that may be used in combination with vertebral adjustment. These techniques are also driven by hypothetical models of muscle dysfunction, which lead to pain and/or become a functional component of the joint dysfunction.

Given the role that muscles are thought to have as a functional aspect of, and/or an effect of joint dysfunction, the question of how muscle tone becomes dysfunctional takes on greater importance. This article will outline some of the mechanisms thought to cause muscle dysfunction and changes in muscle tone. Background information on muscle structure and function, including active (contractile), as well as passive (viscoelastic) properties of muscle will be included. The article will culminate in a clinical muscle model that incorporates classifications based on type of dysfunction, active/passive properties, and chronicity.

Discussion

Anatomical and physiological considerations

While a comprehensive review of muscle anatomy is not the purpose of this paper, there are a few anatomical char-

acteristics that need emphasis when reviewing muscle physiology. At the ultrastructural level of muscles, molecular chains called cross-bridges protrude off of the myosin filament and, in the presence of extracellular calcium, attach to the actin filament. Contraction binds the myosin heads to the actin filament which is then pulled or ratcheted by the cross bridges, much like pulling a canoe parallel to a dock by using your arms. While a high concentration of calcium initiates contraction,¹¹ too low a concentration causes the cross-bridges to lock into place, producing the rigidity of muscles in rigor mortis. Fitts and Metzger summarized the transformation of an action potential into cross-bridge activity as involving seven steps: 1) the sarcolemma action potential; 2) t-tubular charge movement; 3) coupling of t-tubular charge movement with Ca²⁺ release from the sarcoplasmic reticulum; 4) Ca²⁺ release from the sarcoplasmic reticulum; 5) reuptake of Ca²⁺ by the sarcoplasmic reticulum; 6) Ca²⁺ binding to troponin; 7) actomyosin hydrolysis of ATP and cross bridge cycling.¹²

Two major types of muscle fibers make up skeletal muscle; type I or slow twitch, and type II or fast twitch. Type II fibers are subdivided into IIA (oxidative, glycolytic), IIB (glycolytic) or IIX in new terminology¹³ and IIC (transitional).¹⁴ Postural muscles, also known as tonic or red muscles, are characterized by a greater proportion of type I fibers. Type I fibers are involved in slower, prolonged contraction and performance of work, are surrounded by more blood capillaries, have more mitochondria, a larger amount of myoglobin in the sarcoplasm, and contain higher concentrations of muscle spindles.¹⁵ As the intensity of muscle stimulation increases, the motor units made up of type I, type IIA and IIB fibers are progressively recruited.¹⁶ Muscles not only have different proportions of type I and type II fibers, but the geographic distribution of those fibers within the muscle may vary widely.¹⁷⁻¹⁹ This intramuscular distribution of fiber types can affect EMG studies regarding phasic versus tonic muscle activity.¹⁷

Muscles contain two general types of specialized receptors: Golgi Tendon Organs (GTOs) and muscle spindles. A schematic layout of the afferent and efferent innervation of muscle is shown in figure 1.

Golgi tendon organs (GTOs) are tension receptors²⁰ that are stimulated both during muscle contraction and as the muscle relaxes. GTOs give rise to fast conducting Ib afferent nerve fibers that enter the dorsal horn of the spinal

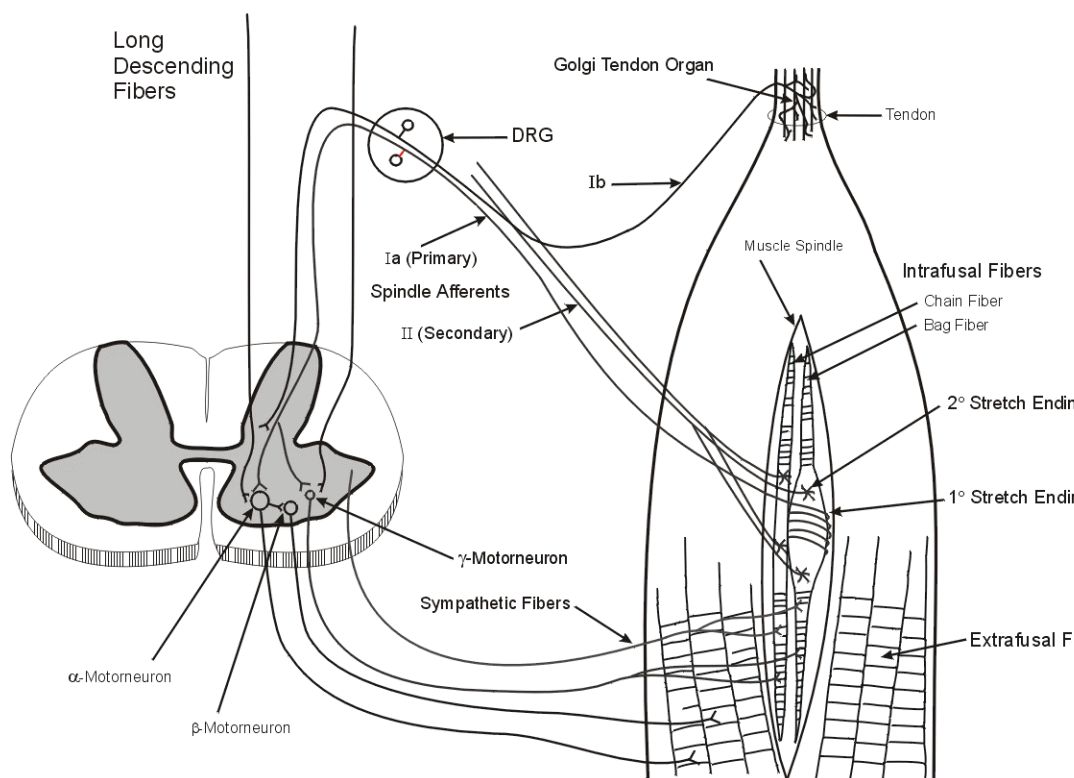


Figure 1 A schematic representation of the neurological connections between muscle and the spinal cord. Afferent fibers lead from the specialized Golgi Tendon Organs and muscle spindle receptors. Efferent neurons lead from the gray matter in the ventral horn of the spinal cord to the extrafusal and intrafusal muscle fibers. Reproduced with modifications and by permission from: Duke, A. Neurology Module 1, Upper Cervical Diplomate Program, Sherman College of Straight Chiropractic. (DRG = dorsal root ganglion)

cord. The Ib fiber then branches into two directions, sending fibers into the dorsal or posterior column which signals higher centers regarding tendon tension, and fibers to the dorsal horn where they synapse on an inhibitory interneuron which then synapses on the cell body of the α -motoneuron serving the same muscle. Thus, the general function of GTO excitation is to inhibit the contraction of the muscle within which it is found²¹ – this is referred to as “autogenic inhibition” and helps prevent damage due to overloading of the tissues. GTOs are quite sensitive, and can respond to the contraction of a single motor fiber.²⁰ Recording of Ib afferents showed their responses to be depressed or abolished during fatiguing muscle contractions, with a slow recovery, the exact mechanism by which this happens, however, is not known.²²

Muscle spindles are a complex subdivision of muscle anatomy about which whole books are devoted.

Kinesthetic sense²³ as well as a variety of neurologic reflexes²⁴ obtains afferent input almost exclusively from muscle spindles, illustrating their importance. Essentially, muscle spindles are length-measuring receptors²⁰ that are embedded in the bulk of the muscle tissue. The muscle fibers outside the spindle are termed “extrafusal” and the small muscle fibers inside the spindle are termed “intrafusal”.

While the nerve supply to the extrafusal muscle fibers is via α -motoneurons, the intrafusal muscle, inside the spindle, is innervated by gamma (γ) fibers. β -motoneurons supply both the intra and extrafusal muscle^{20,25} and receive monosynaptic inputs from both Ia and II afferent fibers²⁵ which are sensory fibers from the muscle spindle. The Beta system ensures a fixed and inflexible co-activation of extra and intrafusal muscle fibers so that the spindle does not become unloaded during muscle contraction.²⁶

The central, non-contractile portion of the muscle spindle gives rise to the Ia and II afferent nerve fibers. When a muscle is stretched, both extra and intrafusal muscle fibers are stretched in tandem. Stretch of the intrafusal muscle leads to spindle sensory excitation and Ia and II output. Spindles can be excited independently of extrafusal muscle stretching via γ -motoneurons which innervate the contractile ends of the intrafusal fibers. Stimulation of the gamma efferents causes the ends of the spindle to contract independently of the extrafusal fibers, stretching the central region of the spindle and exciting the spindle sensory Ia and II fibers.

Muscle spindle intrafusal fibers are thought to receive innervation from branches of the sympathetic nervous system as well,^{27,28} an idea which is finding experimental verification.^{29,30} Sympathetic stimulation was shown to reduce Ia and II output.³⁰ One study found that an increase in sympathetic outflow depresses the feedback control of muscle length, "... which provides evidence for a reduction in proprioceptive capacity under conditions of muscle fatigue, which is always associated with sympathetic activation".³⁰ Sympathetic action on muscle spindles – including the fight-or-flight reaction – may be one of the mechanisms by which physical and emotional stress modify the muscular system. In effect, precision and fine control are transiently sacrificed for stability and reliability of fast running or fighting movements.³⁰

The spindle intrafusal muscle fibers vary in their anatomy, containing variations of bag₁ (b₁), bag₂ (b₂) and chain (c) fibers. Bag₁ fibers are innervated by dynamic or velocity sensitive γ -motoneurons and give rise to Ia spindle afferents.³¹ Bag₂ and chain fibers are innervated by static γ -motoneurons which mainly monitor muscle length, and give rise to type II spindle afferents.³¹ The brain does not have completely separate control of the static bag₂ and chain fiber intrafusal system,³² a finding that may be important in the establishment of reflex spinal muscle hypertonicity. It has been discovered that as many as one-third of spindles in the cervical muscles of the cat lack the bag₁ intrafusal fiber – making them bag₂, chain fiber (b₂c) spindles.³³ These b₂c muscle spindles have also been found in the neck muscles of humans.³³ These spindles represent a novel form of muscle stretch receptor with significantly different responsiveness to muscle stretching than limb muscle spindles.³¹ The purpose of the b₂c spindle has not been definitively determined, but it is thought

they may be utilized as a comparator or reference base signal for fine muscle control.³⁴

The spindle index or density is the number of muscle spindles per gram of muscle. In cats, the spindle density in the soleus is 23 spindles per gram of muscle, in the dorsal cervical muscles 120,³⁵ and in the upper cervical paravertebral muscles, 500 per gram.³⁶ Human spindle density ranges from 50/g in the rectus femoris, 150–200/g in the suboccipital muscles and 200–500/g in the intertransverse muscles.³⁷ It has been suggested that because of their density in the intervertebral muscles, muscle spindles act as a complex information gathering system,³⁸ and the spindle-dense muscles themselves may actually function more as sensory receptors than muscles.³⁹

Muscle spindle afferents are thought to make up a proprioceptive chain, from the eye to the foot, which are involved in controlling human posture and stance; and, by their cortical projections, contribute to the sense of position.^{23,40} In a review of the somatosensory system of the neck, Bolton describes connections between cervical afferents and the cervicocollic, cervico-ocular and tonic neck reflexes.²⁴ The input for these reflexes is primarily from the muscle mechanoreceptors – spindles and Golgi tendon organs – as activity from the zygapophysial joint afferents is rare during natural movement of the vertebra.⁴¹

This short review of select aspects of muscle anatomy and neurology leads to a topic of muscle physiology more controversial and pertinent to the myopathology of joint dysfunction – muscle tone.

Muscle tone

Muscle tone is defined as the stiffness or resistance of the muscle to passive movement.⁴² Even when resting quietly, muscles have some tone. A common misconception, still taught by Guyton and others, is that, "Even when muscles are at rest, a certain amount of tautness usually remains. This is called muscle tone. Because muscle fibers do not contract without an action potential to stimulate the fibers (except in certain pathological conditions) skeletal muscle tone results entirely from a low rate of nerve impulses coming from the spinal cord".⁴³ However, careful research has found no electromyographic activity in normal resting muscle, indicating lack of active α -motoneuron contractile activity.^{42,44} Simons & Mense¹⁹ cite research by Clemmensen (1951), Ralston and Libet (1953) and Basmajian (1957) which failed to find EMG activity in the resting

muscles of normal subjects. Basmajian and DeLuca wrote in 1985, “Muscular “tone” is a useful concept if we keep in mind that at rest a muscle relaxes rapidly and completely. This has now been common knowledge among neurophysiologists for more than a decade. If one keeps one’s hands off a resting muscle, it shows no more neuromuscular activity than one with its nerve cut”.⁴⁵ Lakie et al. could find no reduction of muscle tone in a group of 14 patients anaesthetized with a mixture of intravenous anesthetics and muscle relaxants,⁴⁶ again indicating that resting muscle tone is not maintained by active α -motoneuron stimulation. Walsh provides a historical review behind the misinterpretation that resting muscle tone depends on a low-level tonic discharge of α -motoneurons.⁴⁴

Others have invoked tonic fusimotor (γ -motoneuron) activity as the underlying cause of resting muscle tone. Tonic γ -motoneuron stimulation would hypothetically cause spindle output that reflexes back to α -motoneurons causing them to fire, leading to normal resting muscle tone. However, Hagbarth reviews several microneurographic (insertion of tungsten microelectrodes into peripheral nerves) studies that conclude fusimotor activity is absent or negligible in human muscles kept in a totally relaxed state.²⁰ Davidoff also cites research on passive, relaxed human muscles that demonstrates a low and irregular rate of spontaneous firing from muscle spindles which was unchanged after a peripheral block of fusimotor fibers.²⁶ Such evidence suggests that fusimotor fibers to relaxed human muscles do not subject the spindles to significant background drive.

Characteristics of passive – viscoelastic – muscle tone

If tonic α - or γ -motoneuron activity is not responsible for the tone in normal resting muscle, what is? The resistance to a change in length of a normal resting muscle – its stiffness – is caused by the inherent viscoelastic or mechanical properties of muscle tissues.⁴² These included the viscoelastic properties of tendons,⁴⁷ titin^{48–50} and the actin-myosin cross-bridges. Due to their ability to variably change the stiffness or tone of the muscle, the actin-myosin cross-bridges are the most promising to examine.

Skeletal muscle fibers, intra- and extrafusal, exhibit a physiologic property known as thixotropy. A thixotropic substance behaves as a solid below a certain applied shear force, and as a fluid at higher shear force.⁵¹ Paint, tomato ketchup and human blood all show thixotropic properties.

After an initial, threshold movement, their viscosity decreases, often dramatically. Ketchup, as an example, resists being poured from a bottle that has been at rest. Upon shaking – applying shear force – the ketchup pours easily.

A widely accepted explanation for thixotropy of muscles is a tendency for the actin and myosin filaments to stick together when inactive for a period of time.⁵¹ After movement, the actin-myosin cross-bridges reform or reset to the new, contracted or lengthened state.^{52–56}

Postural or anti-gravity muscles exhibit high degrees of thixotropy that are believed to allow for the maintenance of a given posture without active contraction and the expenditure of energy.⁴⁴ Because muscle thixotropy is non-active, the resistance to stretch of a resting muscle can be higher than that muscle’s normal resting tone – in the absence of contractile activity. For example, if the fingers grip an object tightly for several seconds – active contraction – then release and relax, the tone of the finger flexor muscles, now at rest, will be higher than prior to the active flexion. For a review of the thixotropic behavior of skeletal muscle and muscle spindles see Proske et al.¹¹

Categories of active – α -motoneuron activity – muscle tone

Muscle tone ranges from paralysis to spasticity. While paralysis, a complete lack of any volitional α -motoneuron activity, is not often cared for in a chiropractic office, other muscular tensions in the wide continuum of muscle tone are seen clinically. These differing levels of tone need to be defined and described.

Hypotonia or weakness is often mistaken as a decrease in the normal active contractile tone of a resting muscle (based on Guyton’s remark that resting muscle tone involves active contraction). Due to this misconception, hypotheses are forwarded that in a resting position, the normal active tone of agonists can overcome their “weak” or hypotonic antagonists, thus moving joints into positions of abnormal flexion or extension. An example of such a hypothesis would be the notion that hypertonic neck flexors might overcome weak extensors to produce a postural abnormality of forward head posture. However, as reviewed above, resting muscle shows no continuous contractile activity, so flexors of normal tone cannot overcome hypothetically “weak” or hypotonic extensors because both the agonist and antagonist muscles are inactive when resting.

Hypotonia or weakness of a muscle is perhaps better thought of in terms of an active contraction which generates less force and is quicker to fatigue than normal, and not as a decrease in tonic α -motoneuron activity.²⁶ This kind of muscle weakness has been demonstrated in cases of muscle injury/inflammation,⁵⁷ joint injury,⁵⁸ facet injection⁵⁹ and due to the effects of tonic neck reflexes.^{60,61}

Hypertonicity is greater than normal resting tone, often referred to using the generic term “spasm”. Emre defined muscle spasm as an “involuntary and inappropriate, reversible, prolonged bracing of a muscle or group of muscles, attributable to overactivity of motor units or changes of excitability of muscle fibers”.⁶² Simons and Mense add that muscle spasm exhibits electromyographic activity that is not under voluntary control, is not dependent upon posture and may or may not be painful.⁴²

Hypertonicity is most often thought of as active muscle contraction due to α -motoneuron activity. As an example, “spasm” of the biceps would flex the elbow, the biceps would be hard (less compliant) and if we tried to extend the arm, the resistance would be increased – hypertonicity. This is alpha hypertonicity. Muscle tone, however, can also be affected by the stretch reflex. Reflex mediated muscle stiffness is determined by the excitability of the α -motoneuron pool^{63,64} which depends in part on the activity in the γ -motor system.^{65,66} For example, stretch reflexes contribute to about 50% of the stiffness of the ankle flexor and extensor muscles in man.⁶⁷ Such stiffness is an important factor that can dominate the mechanical behavior of the ankle joint during everyday motor tasks.⁶⁷ The gamma system acts like a rheostat trigger, decreasing the firing threshold of the alpha system, causing muscle fibers to contract and increase the resistance to stretch at varying levels of sensitivity. Revisiting the elbow joint example again, with the joint in a resting, neutral position, both the biceps and triceps are normally compliant with no evidence of hypertonicity. Suppose the gamma signal to the biceps is increased. Now when the biceps are stretched – and hypertonicity is the resistance to stretch – the muscle reacts to the stretch (the stretch reflex) with more power (resistance) than normal. This would be gamma hypertonicity bias.

Given the importance of increased muscle tone, the lack of an agreed upon descriptive terminology for varying types of muscle hypertonicity is surprising. Janda has outlined five various types and characteristics of increased

muscle tone.⁶⁸

- 1 Limbic dysfunction – muscle hypertonicity due to dysfunction of the limbic system that is usually not spontaneously painful. Muscle tension from emotional stress is an example.
- 2 Impaired function at the segmental (interneuronal) level – characterized by an altered balance between physiologically antagonistic muscles. Muscle tension asymmetry in joint dysfunction would fall into this category.
- 3 Impaired coordination of muscle contraction – this type leads to increased tone in part of a muscle where the rest of the muscle remains normal. Trigger points (TrPs) may be included in this category.
- 4 Response to pain irritation – a defense reaction meant to immobilize an injured part of the body. Splinting spasm from viscera and flexor or nociceptive reflexes are examples.
- 5 Overuse – muscle tightness, involved in “muscle imbalance” syndromes. Janda believes this type of hypertonicity is due to long-term overuse and finds the active muscle fibers are replaced by non-contractile tissue.

Paradoxically, a muscle can be hypertonic – an increased passive resistance to stretch, yet weak – an inability to sustain contraction. This phenomenon can be demonstrated in a contracted muscle which begins to build up fatiguing metabolites. While the muscle has higher than normal resting tone, further activation (stretch) of the muscle produces less and less power, i.e., the muscle is weak.

In most hypothetical models of joint dysfunction, muscle hypertonicity is an active agent in the cause, maintenance, and effects of the pathologic complex. In the area of muscle hypertonicity associated with joint dysfunction, Denslow and Hassett described three categories:⁶⁹

- 1 Normal – soft, resilient and not tender
- 2 Minor lesions – less rigid and tender, subjects are often unaware of the abnormality
- 3 Major lesions – considerable degree of abnormal firmness, rigidity and tenderness

Active muscle hypertonicity also includes other diagnostic/descriptive categories, including focal dystonia, antalgia, trigger points, cramp, spasticity and rigidity.

Focal dystonia is defined as hyperactivity in a single

muscle without a clear source.⁷⁰ While the cause of dystonia is unknown, the basal ganglia,⁷¹ abnormal processing of information from muscle spindles⁷² and the fusimotor system⁷³ have been implicated. Grünewald et al.⁷² suspect that dystonia involves an abnormal reliance on group II instead of Ia spindle afferents for information on muscle movement. Type II afferents, again, are thought to be more responsible for information relating to muscle length rather than movement. Abnormal stimulation of muscle spindle type II afferents has been proposed to be part of the gamma-loop mechanism of muscle tone dysfunction (see “Gamma-loop” section, Part II). Further, the inherited neurologic disease Charcot-Marie-Tooth (CMT) Type Ia involves selective degeneration of larger nerve fibers including muscle spindle primaries (group Ia), but not secondaries (group II).⁷⁴ CMT subjects can suffer from significant and severe back pain, spasms of muscle after rapid movements, difficulty in releasing grip, on releasing the grip the hand slowly flexes again, the inability to relax muscles, and legs that stiffen when attempting to run.^{75,76} Perhaps, as proposed by Grünewald, it is the loss of muscle spindle Ia afferents and the dependency on type II afferents that disposes CMT sufferers to these types of muscle dysfunction. There has been a case report of manipulative management of a particular focal dystonia.⁷⁷ Focal dystonia includes spasm of the sternocleidomastoid – cervical dystonia or torticollis.

Antalgia often involves a group of muscles that become hypertonic in order to position the body away from pain. Antalgic posture itself may or may not be painful. Antalgic postures are often exhibited as a stabilizing effect for disc injury.^{78,79}

Trigger points (TrPs) are palpable, hypertonic and hyperirritable localized tender points in skeletal muscle lying within a taut band of muscle fibers.³ TrPs are thought to be caused by muscle trauma, micro and macro,³ and may also be an attempt to meet excessive demands on muscle strength⁶⁸ and overwork fatigue.⁸⁰

Donaldson reports that on a general basis, individuals with a unilateral TrP demonstrate a significant bilateral difference of surface electromyography (sEMG) amplitude generated during the primary movement of the muscle. This finding differentiated sufferers with TrPs from those without in 80% of cases; unilateral TrPs corresponded 100% of the time to the higher sEMG reading.⁸⁰ Given these studies, increased active muscle tone – hyper-

tonicity – does seem to be a quantifiable aspect of trigger points.

Interestingly, Donaldson proposes that TrPs are caused by the unusual physiologic properties of muscle spindles. He hypothesizes that when a muscle is stretched upon its length with sufficient force while that muscle is contracting – for example in an auto accident when muscle bracing (contraction) is overcome by opposing forces (stretch) – the interaction between the muscle spindle fiber and the length of the muscle becomes “dysregulated”. By dysregulated, Donaldson means the intrafusal representation of the extrafusal muscle length no longer matches the actual length. This dysregulation alters the afferent signal sufficiently to adversely affect the normal feedback at the motoneuron pool.⁸⁰

Cramp is a sudden, painful muscle spasm, which may or may not be electromyographically active. Cramps generally occur after, rather than during, exertion, or may come on when a muscle is apparently at rest.⁸¹

Spasticity commonly refers to muscle spasm observed in clinical conditions of upper motor neuron lesion and is associated with hyperactive stretch reflexes. Spasticity can be thought of as occurring at the spinal level because of loss of supraspinal inhibition.⁴² Interestingly, some studies have found no continuous motor activity in the muscles of spastic patients.⁸² Hufschmidt and Mauritz state, “Muscle tone at rest and during slow passive movements is exclusively due to intrinsic muscular properties. There is no electromyographic evidence that it is sustained by continuous motor neuron activity. This is also true in spastic patients and is confirmed by the results presented here”.⁸²

Finally, rigidity, though caused by muscle spasm, involves opposing muscle groups equally and throughout the range of motion. In a review of the literature, Murthy writes that Parkinson’s rigidity may be attributed to lesions in the substantia nigra that leads to a selective depression of dynamic fusimotor neurons and activation of static fusimotor neurons.⁸³

Detecting changes in muscle tone

Joint dysfunction can be characterized by the muscle-mediated findings of tenderness, asymmetry of joint position, restriction in range of motion, and tissue texture abnormality (TART).¹ Detection of these characteristics is most often accomplished by the art of digital palpation, which Janda calls the “best” method of evaluating muscle tone.⁶⁸

However, in order to provide objective measurements of tone, other methods have been developed, including EMG, sEMG and the use of a tissue compliance meter.

Electromyography reflects electrical activity occurring within a muscle – active contraction. In a number of studies, EMG has shown active muscle involvement in low back pain,^{84–88} has been used as a confirmatory sign of joint dysfunction,^{69,89} and a monitor of effects of manipulative treatment.^{90–93}

Surface electromyography to detect muscular activity of the spine has some supporting research.^{80,94,95} Kent⁹⁶ reviews EMG and sEMG associated with subluxation, while Meyer⁹⁷ presents a critical look. Research to clarify the reliability of sEMG in identifying areas of spinal joint dysfunction continues.⁹⁸ Some recent research points to sEMG as a tool to detect low-level muscular activation in chronic low back pain.^{99–101}

Muscle tone, as applied to clinical practice, is measurable as stiffness, which is the resistance to passive movement in the absence of contractile activity,⁴² and compliance, or the compressibility of muscle. The use of a compliance meter to measure muscle resistance, much as is done in manual palpation, may be a more accurate way to test muscle tone because, as we have seen, some increased and decreased tone is not due to active or electrical contraction. Such compliance devices have been built and tested^{102–105} including for cases of suspect joint dysfunction.^{106–108} There is evidence that manual compliance devices may be unreliable and inaccurate,¹⁰⁹ however, automated meters to assess soft tissue compliance show promise^{110,111} in determining changes in compliance.

Other, higher tech, non-invasive methods have been developed to determine muscle function, including cortical somatosensory evoked potentials (SEPs) and near-infrared spectroscopy (NIRS). SEPs record latency (for conduction velocity) and/or amplitude (for volume of receptors available at stimulation) of a magnetic stimulus at a distal part of the body (the lumbar paraspinal muscles for instance) with recording electrodes at the cortex. The amplitude of SEPs has been investigated relative to the relationship to muscle spasm and low back pain.^{112,113} These studies found unilateral changes in muscle physiology in selected groups of low back pain patients. It is suspected that the noted spasm of the erector muscles causes (or is caused by) stimulation of muscle spindle afferents, reducing the number of afferents available to be excited by the SEP

stimulation. As the spasm decreases, and the presumed muscle spindle afferent input decreases, the amplitude of the SEP increases. One of the studies found that manipulation normalized the SEP response; however there were no controls and the number of patients studied was small.¹¹² The authors do call for, "... a closer examination of the role of muscles in patients with low back pain".

NIRS is based on the variable absorption of light in the near-infrared spectrum by oxygenated and deoxygenated forms of hemoglobin and myoglobin. NIRS enables noninvasive monitoring of oxygenation of muscle tissue, and hence active muscle contraction at levels as low as 2% of maximal voluntary contraction (MVC).^{114,115} Such low-level muscle contractions, which may play a role in some types of back pain^{115,116} (see "Sub-maximal contractions" section, Part II) may not be easily demonstrable using EMG or SEMG.

The paper will resume with Part II, describing functional characteristics of muscle tone and models of joint and muscle dysfunction. (December issue)

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