THE MUSCLE SPINDLE SENSORY RECEPTOR
An In-Depth Study By Tamsin Stewart

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MUSCLE SPINDLE SENSORY RECEPTORS

A. INTRODUCTION:

Wikipedia defines muscle spindles, the small nerve bodies located throughout skeletal muscles, as sensory receptors whose job is primarily to detect changes in the length of their muscle, and then convey that length information to the central nervous system via sensory neurons. From this information the brain determines the position of body parts. The spindles also regulate muscle contraction via the stretch reflex. Thomas Griner has made the study of muscle spindles his life’s work, and his description and definition differ from and expand on conventional thinking on the subject.

We'll first look at the Location and Structure of the spindle; then you'll learn about the Nerves* of the Muscle Spindle. Next we go into the tiny Intrafusal Muscle Fibers, muscle fibers actually encased within the spindle nerve body itself, (intrafusal = muscle fibers inside the spindle capsule; extrafusal = muscle fibers outside the capsule; i.e. the regular working forest of skeletal muscle fibers). Next covered is the Lymph System of the Spindle, and then everything comes together in more detail under Spindle Function – and of course, Malfunction.

Griner refers to the muscle spindle picture below as “The Runway Model”; we like her because she’s so lovely, floating in air, a thing of beauty. The picture shows one chain fiber attaching to one bag fiber, which extends out to the myofascia. Off to the side you can see the main muscle. What does that picture tell you? Not a thing. But on page 21 of this Lecture you will find a not-very-pretty working drawing by Griner, that tells much more about muscle spindles.

*(AUTHOR’s NOTE: Like the chicken/egg debate, it was difficult to decide whether to first discuss the spindle’s nerves or its muscles; the nerves won. Just read along, and all will become clear.)

B. LOCATION & STRUCTURE OF THE SPINDLE

The muscle spindle is a fusiform-shaped organ (wide in the middle and tapered at both ends), encased in its own connective tissue capsule that grows out of the perimysial connective tissue surrounding the fascicle of working muscle fibers to which that spindle belongs.

Each muscle spindle cell contains small intrafusal muscle fibers wrapped in endomysium, and each spindle cell has a single lymphatic drainage that pulls fluids both out of and in to the organ. Efferent & afferent gamma nerves enter & exit the spindle.

There are many spindles scattered all along the length of each fascicle, distributed throughout skeletal muscle bellies; the number per fascicle depends on the length and function of the muscle. They are attached to the outside of the perimysial wall surrounding the fascicles, and so are in the corridors between fascicles, along with lymph ducts, nerves, and capillaries. Spindles are actually an outgrowth of the perimysium ~ the outside of the spindle capsule is just an extension of the perimysium. The inside of the capsule is slightly separated, but perimysium still forms the inside of the capsule.

Most sources will tell you that spindles attach directly to and lie in parallel with the extrafusal muscle fibers, but this is untrue; muscles cannot attach to muscles.

Because the spindles are located in the interfascicular corridors, embedded within the perimysium of each fascicle, they are able to monitor contractions taking place inside their fascicle, and collect an average of the activity of all the extrafusal muscle cells within that bundle. One spindle collectively takes the action of all the fibers adjacent to that part of the perimysium. In other words, one spindle samples a whole group of muscle fibers. We could say
it operates on hearsay, like one monitoring scuttlebutt on the other side of a wall by holding a
glass tumbler up to the wall and pressing his ear against the glass.

Then based on what the perimysium says is going on inside its fascicle, the spindle reports the activity of the extrafusal muscle fibers to the cerebellum through small, delicate feedback nerves.

A communication breakdown occurs not in the reading the spindle takes from the perimysium, because those are surprisingly accurate; instead the problem happens in the nerve relay system carrying information to the brain, and correcting this problem is really what NeuroSoma® is all about. Now we look in more detail at muscle spindles, starting with its nervous system.

C. NERVES OF THE MUSCLE SPINDLE

| Efferent nerves exit the brain or spinal cord and travel to the muscle |
| Afferent nerves travel back to the brain or spinal cord from the muscle. |

Efferent (motor) into the spindle from the CNS:
The main nerve branch comes down from the spine and gives off the alpha motor nerve, which goes to the extrafusal fibers, plus gamma motor fibers that go to the spindle and convert electrical activity to mechanical activity (i.e. nerve pulse to muscle contraction).

Alpha Motor Nerve: the Final Common Pathway

Alpha motor nerves innervate the main skeletal muscles; they’re rapidly-conducting, pyramidal system nerves that have summated all the information coming from hundreds of dendrites along the cell body of the alpha motor neurons. Some of these dendrites are from the pyramidal nerves coming from the cerebrum, some are from the cerebellum, some from the annulospiral nerves from both bag1 and 2 intrafusal muscle fibers (you’ll learn about them next) via the spinal cord reflex arc; some are from the Golgi tendon organ, and some from nociceptors in the skin. Some are inhibitory, some are excitatory, some are involved in pain reflex arcs, and so on.

Think about that alpha motor nerve for a minute. Realize that in order to get to a skeletal muscle fiber, this nerve has pierced through the epimysium ~ the fascia surrounding the muscle ~ to get into the spaces between the fascicles, then through the perimysium ~ the fascia surrounding the fascicle ~ to get into the fascicle itself, and then it travels between the individual muscle fibers, along with the capillaries. Finally it pierces into the endomysium ~ the fascia surrounding the individual fiber ~ in order to reach the muscle fiber. The alpha motor nerve is the only thing that pierces the endomysium. And where is the sarcolemma (muscle cell wall) relative to this? The sarcolemma is underneath the endomysium, and the motor end plate is on the sarcolemma, which itself is nerve tissue, and conducts the nerve pulse along the length of the cell and down into the interior of the cell via the transverse tubules.

In the illustration by Thomas Griner of the muscle spindle on Page 21 herein, note that the nerve branch coming down at the top of the page gives off 1) the alpha motor nerve to the extrafusal fibers, 2) the gamma1 motor fiber (which goes to the dynamic stretch reflex bag1 fiber), and 3) gamma motor nerves to bag2 and chain fibers. There has to be a gamma efferent axon to each end of the intrafusal fibers, because they have no actin or myosin in the middle, only nuclei; so both ends of the fibers must be stimulated to contract. Although these gamma
Gamma efferent motor nerves from the CNS into the spindle determine the amount of tension, or stress in the spindle organ; gammas use the extra-pyramidal nervous system (see Lecture 1 Page 15).

In the section on intrafusal muscle fibers below, you'll learn that the tiny striated intrafusal muscle fibers within the spindle organ have to be innervated just like any other muscle, even though their middle portions contain no actin and no myosin, and it is only their ends that contract. Just as motor nerves innervate the extrafusal muscles, so do smaller ‘gamma’ versions enervate the little intrafusal fibers. The large alpha motor nerve branching to the extrafusal fibers (designated by a little ‘a’) gives off smaller gamma efferent nerves to the spindle (designated by a little ‘y’). These thinner, slower gamma motor nerves form synaptic connections, similar to motor end plates, on both ends of the spindle’s intrafusal fibers. These efferent motor nerves coming into the muscle spindle are

- Gamma 1 (to dynamic bag1 fibers)
- Gamma 2b (to static bag2 fibers)
- Gamma 2c (to static chain fibers)

All these gamma efferents excite mechanical contraction of their intrafusal fibers just like the alpha nerves excite contraction to the extrafusal fibers. Gamma nerves have smaller, slower impulses than the rapidly conducting alpha nerves.

There is always stress in the spindle because it always maintains at least resting muscle tone. In the section on intrafusal muscle fibers below, you'll learn that when the chain fibers release their stress and transfer it to the bag 2s, muscle tone increases, producing positive feed forward from the bag2 annulospirals. Healthy muscle tone is developed by 5 pulses per second, but hardly anyone has that except in their flat muscles (and if all our muscles were flat, our top speed would be 9 miles per hour instead of 26 MPH). Ideally of course, our round muscles would also send out 5 pulses per second. But in a hypertonic muscle, the nerve pulse can go off the chart; there is no standard reading for it. The core of a round muscle, where all contraction begins, can actually go into physiologic contracture, wherein the fibers cannot ever relax (akin to rigor mortis), so produce no nerve activity at all.

Afferent (sensory) gamma nerves out from spindle to CNS

The muscle spindles contain 2 types of nerve end organs (aka ‘endings’) that convert mechanical activity into electrical activity (i.e. muscle contraction back into nerve pulses). These are the flowerspray end organs and annulospiral end organs.

Each of the gamma afferent nerves leaving the spindle begins with nerve end organs attached to the intrafusal muscle fibers within the spindle. What is an end organ? It is a transducer, which converts stimuli, either chemical or mechanical (for example vibration, pressure, temperature, taste) to an electrical impulse. This electrical impulse is then transmitted by the afferent gamma nerve fiber that comes off the end organ. Both the annulospirals and flowersprays are nerve end organs and both are mechanical transducers.

Note that the muscle spindle sensory receptor is the only organ in the body able to convert electrical signals into mechanical action, and mechanical action into electrical signals!

Annulospiral End Organs & Their Feed-Forward Nerves

The large, sensory annulospiral nerve end organs (‘primary endings’) wrap themselves around the very center of the nuclear receptor areas of both bag1 and bag2 fibers. The 1a gamma afferent nerves coming off these end organs travel directly to the spinal cord through the posterior nerve route, where they synapse with alpha motor nerves that then fire back into

efferents are not attached to the extrafusal fibers in any way, there is some adaptation between the alpha and gamma nerves in that when the alpha nerves contract the extrafusal fibers, the gamma nerves also contract the intrafusal fibers. This allows the spindle organ to track with the skeletal muscle’s voluntary movement. When the spindle does not track together accurately with the main muscle, there will be a difference in the speed between cerebellar control and cerebral control of the muscles. This mismatch between control of the extrafusal fibers using the pyramidal nervous system, and the intrafusal fibers using the extra-pyramidal system is known as cerebral palsy.

Gamma efferent motor nerves from the CNS into the spindle determine the amount of tension, or stress in the spindle organ; gammas use the extra-pyramidal nervous system (see Lecture 1 Page 15).
the main extrafusal muscles associated with that particular spindle, causing them to contract. Therefore the annulospirals generate feed-forward, to activate contraction in skeletal muscles.

And although they are called ‘group 1a’ gamma afferents and are considered afferent because they travel back to the spinal cord, because they synapse directly onto the alpha motor nerve in the spinal cord, and generate nerve impulse to activate the skeletal muscles to contract, they are in reality efferent, “feed-forward” nerves, driving contraction into skeletal muscles to nullify the stretch that started the whole business. This is called a reflex arc.

All annulospirals nerve endings convert mechanical stimuli, whether from intrafusal or extrafusal muscles, into electrical activity. However, the bag1’s annulospiral nerves are stimulated much more powerfully when the muscle and its spindles are stretched than from stimulus caused by the bag2’s static response. The bag1 annulospiral responds extremely actively to the stretch, instantly summatting the mechanical action of the stretch and converting that information into action potentials that are transmitted through its annulospiral nerve into the alpha motor nerve cell body in the spinal cord. This excess stimulus of the primary ending – the dynamic stretch reflex response – is well known and yet is widely ignored by therapists and trainers alike.

These little gamma nerve fibers transmit sensory signals to the spinal cord as rapidly as any type of sensory nerve fiber in the entire body. Only 1/5th of the annulospiral endings come from bag1 fibers and are involved in activating that powerful, dynamic stretch reflex contraction. 4/5ths of the annulospiral endings come from bag2 fibers and are involved in activating the static, smooth, constant contraction of muscle tone.

**Flowerspray End Organs and Their Feedback Nerves**

The flowerspray end organs (‘secondary endings’) are also transducers, converting muscle activity into nerve pulses. They are located the chain fibers, and possibly on the bag2 fibers; their tiny little flower-like buds at the ends of their gamma afferent nerve fibers give them their name. The flowerspray nerves travel directly back to the cerebellum to provide feedback information the cerebellum needs in order to maintain proper muscle tone.

Although these little flowerspray feedback gamma nerves do convert muscle activity into nerve pulses, they only provide information specific to the low-level, automatic contraction of muscle tone existing in their skeletal muscles while in a resting state; they measure the stress or force endured by their fascicle’s forest of extrafusal muscle when at rest, then take that information directly to the cerebellum, which compares that nerve signal with the 5 pulses-per-second, pre-programmed “set value” that it, the brain, is required to maintain. Any variation from that set value causes a variation in the efferent gamma motor nerves from the cerebellum back to the intrafusal fibers.

If the incoming feedback signal is too low, the cerebellum inhibits or reduces nerve output to the chain fiber through its gamma efferent (y2c) fibers, thereby relaxing the chain fibers. This adds the stress the chains were carrying to the next in line, the bag2 fibers to which the chains are attached. Remember, the stress is along the length of the fibers, so whatever the chains release, the bag2s have to take up. So we have reduced activity to the chain fibers and increased stress on the static bag2 fibers. The static bag fibers’ annulospiral nerve end organs then start driving into the extrafusal fibers via the spinal cord reflex arc.

And of course when things are working properly, if the signal from the flowersprays into the cerebellum is too high, the brain increases nerve output to the chain fibers, contracting them and reducing stress to the bag2 fibers next up the line. Any annulospiral activity coming from the bag2s decreases, allowing the extrafusal muscle fibers to relax. This is a major component of maintaining the muscle tone system.

The system uses this feedback loop, as opposed to the reflex arc of the annulospirals going to the spinal cord to synapse with the alpha motors in the dynamic stretch reflex. But both utilize the mechanical synapse aspect of the spindle – turning muscle contraction (mechanical activity) into an electrical signal. Muscle tone isn’t a dynamic response, as would come from the bag1 fiber, in response to outside forces, but a static response to inside forces. But just like those on the bag1 fibers, the bag2 annulospirals go right back to the spinal cord to synapse with and drive the alpha motors back into the main extrafusal fibers.
If all goes well, the muscle tone system and the dynamic stretch reflex work perfectly; the trouble is that often, even typically, all does not go well; there is a glitch in the way we’re made that can create havoc when the system of muscle tone runs amok.

D. INTRAFUSAL MUSCLE FIBERS

Overview
There are 3 kinds of intrafusal fibers within muscle spindles: bag1, bag2, and chain fibers. Please note that only the ends of these tiny intrafusal muscle fibers actually contract; unlike extrafusal fibers, their central portions do not contain actin or myosin filaments; they contain nuclei only, and therefore the intrafusal muscle fibers function as sensory receptors rather than contractile units. All intrafusal fibers are innervated at their ends with gamma motor nerves, which fire concurrently with the alpha motor nerves driving into extrafusal muscles.

Just as each extrafusal muscle fiber is wrapped in its own fascial layer of endomysium, so is each intrafusal muscle wrapped in its own endomysium.

All 3 types of intrafusal muscle fibers attach within the spindle differently: bag1s are the spindle’s main attachment to the perimysium that wraps the bundle of extrafusal muscle fibers (fascicle) to which that spindle belongs; i.e. the bag1 fibers pierce the spindle organ’s own perimysial capsule, then attach directly into the perimysial fascia. Bag2s attach to the ends of bag1 fibers, and both short and long chain fibers attach to the ends of bag2 fibers. In each spindle there are more intrafusal bag2 fibers than bag1 fibers, and the intrafusal chain fibers are even more numerous.

Both bag1 and bag2 intrafusal muscle fibers are so-called because a large number of nuclei are congregated into an expanded bag in their central receptor areas. Bag1 fibers generate the dynamic (myotatic) stretch reflex response, and are slow-twitch fibers. (Dynamic means force; power; constantly changing; movement. ‘Myotatic’ specifically refers to the dynamic stretch reflex response in muscles, so in our context, we can use the words interchangeably.)

Fast-twitch Bag2 fibers have 3 jobs, which are 1) generate the static stretch reflex reaction, 2) assist the chain fibers in controlling muscle tone, and 3) maintain fine motor control. Primary annulospiral gamma nerve endings wrap the central, nuclear portions of both bag1 and bag2 intrafusal muscles; nerve fibers issue from these nerve endings and travel to the spinal cord through the posterior route.

Fast-twitch chain fibers are so-named because their central receptor areas have nuclei aligned in a chain. Their only job is to monitor and relay information on muscle tone. They sense how much automatic contraction (‘muscle tone’) is occurring in their fascicle of extrafusal skeletal muscle when it is resting, and then relay that information to the cerebellum. Secondary gamma afferent flowerspray nerve endings (they have little flower-like buds at their ends) wrap the central, nuclear portion of the chain fibers; ‘feedback’ nerve fibers issue from these endings and travel directly to the cerebellum.

The little chain muscles are about half as long as the bag2 fibers to which they attach, and to complicate things even more, there are both long and short chain fibers (don’t ask why, I don’t know). The interfusal muscle fibers that deal with muscle tone – fast-twitch bag2s and chains – operate continually (there is always stress in the spindle, because it always maintains at least resting muscle tone), whereas slow-twitch, dynamic stretch reflex bag1 fibers operate intermittently.

Again, only the ends of all 3 types these intrafusal muscle fibers contract; their central nuclear portions function as receptors. Each intrafusal fiber is wrapped in its own endomysium, and each has a specific job: Bag1s create the dynamic stretch reflex, almost 20% of the spindle’s function. Bag 2s help the chain fibers maintain the system of muscle tone, they create the static stretch reflex against gravity, and they maintain fine motor control. Chain fibers monitor and maintain muscle tone, and this job comprises almost 80% of the spindle’s function.

All chain fibers are muscle tone fibers; some bag fibers are muscle tone fibers and some are myotatic stretch reflex fibers. Bag2s for muscle tone are fast-twitch fibers, and bag1s
for myotatic stretch reflex are slow-twitch fibers. Both kinds of bag fibers have annulospiral
nerves, because they both drive into the main muscle to make it contract.

**What is the difference between static stretch reflex and dynamic stretch reflex? This
differentiation is not often made, but Griner defines static stretch reflex as a steady,
constant, and balanced contraction, in equilibrium; static doesn’t change with time, it’s
fixed. As one contracted fiber lets go, another immediately and smoothly contracts to take
its place. It is an inside force against another inside force, and shows up on an EMG as
background noise only, regardless of how many fibers are involved. In fact, the term “static
stretch reflex” is an oxymoron, because ‘stretch’ is a dynamic term; what is usually meant
by that term is ‘muscle tone’, a basal metabolic function, operating even while asleep, and
it doesn’t change much from that point. We also use “muscle tone” in lieu of static stretch
reflex. The word dynamic means constantly changing; the dynamic stretch reflex is a
powerful inside response to an outside force (i.e. tapping the knee with a hammer) and
shows up on an EMG as a sudden, sharp spike of activity. Even though it is only 20% of
the spindle’s function, the dynamic stretch reflex causes powerful reactions compared to
muscle tone. It can easily override the constant, low-level muscle tone, unless the level of
muscle tone has ratcheted up high enough to nullify the need for dynamic contraction.**

**Bag1 Fibers:** How the bag1 intrafusal muscle fibers control the dynamic stretch reflex mechanism:
As stated previously, both ends of bag1 fibers pierce the spindle capsule and connect directly
into the perimysium surrounding that particular spindle’s fascicle of extrafusal fibers; this allows
bag1s inside the spindle to respond to changes in the length of the extrafusal muscle fibers;
they are the 1st level, so to speak. When the extrafusal fibers are stretched, the perimysium
surrounding that bundle of fibers also stretches. If the stretch lasts long enough, the
annulospiral nerves wrapping the spindle’s intrafusal bag1 fibers simultaneously fire their
gamma nerve signals into the spinal cord; that incoming signal then reflexly fires the alpha
motor nerves back into the working forest of extrafusal fibers within the fascicle to which that
spindle attaches, creating a contraction in that stretching fascicle. The stretched muscle is
automatically contracted! Almost 20% of the spindle’s function is in controlling this dynamic,
powerful muscle contraction. And these bag1 intrafusal muscles to which these stretch reflex-
activating annulospiral endings are attached are slow-twitch, rapidly-adapting muscles. Bag1
fibers are slow-twitch fibers. One would think that bag1 muscles, acting quickly & intermittently,
would be fast-twitch anaerobic fibers, and that bag2/chain fibers acting continuously would be
slow-twitch aerobic, but it’s just the reverse.

There are 3 reasons why dynamic stretch reflex bag1 fibers must be slow-twitch fibers:

1) **Bag1s must be fatigue-resistant and always ready to go – a required characteristic for
dynamic reactions.** Slow-twitch fibers are easier to excite at a slow metabolic rate, and
so are encouraged to perform most of the action; that dynamic bag fiber is always there
waiting, ready to go if needed, even when we’re sending out a low number of nerve
pulses. *(But be aware that even in basal metabolism ~ when asleep for example ~
some fast-twitch fibers are contracting to maintain muscle tone; which is why even
during sleep there is a basal metabolic level of lactic acid in the bloodstream; and yes,
it has been measured!)*

2) **Bag1s need to stay a beat behind the main, extrafusal muscles (which are 50% slow &
50% fast) to which they are attached, in order to sense changes in those muscles and be
able to react to their action.** Remember that dynamic bag1 fibers have contractile
portions on their ends so they can adapt to the changing length of the extrafusal fibers.
When the skeletal muscles contract (shorten), stress is mechanically reduced to the
spindle, causing the intrafusal fibers to relax. When the skeletal muscles are stretched
(lenthen), stress is mechanically added to the spindle, which causes the contractile
portions of the bag1s to contract and their annulospirals to fire into the spinal cord,
thereby firing the alpha motors into and contracting the extrafusal fibers in the dynamic
stretch reflex response. *(Note that adding or reducing stress within the spindle is a
mechanical reaction, as opposed to the result of neural stimulation.)*
Remember that each motor unit innervates both slow & fast-twitch fibers. In basal metabolic, when a low number of nerve pulses are being generated, the slow-twitch extrafusal fibers out in the skeletal muscles will be more excited to contract at that slow rate and encouraged to do a majority of the action. Inside the spindle, the bag1s need to lag behind ALL the extrafusal fibers – fast & slow – so they can sense and monitor any change (or stretch), and if needed, create the powerful stretch reflex response to that stretch.

3) **Bag1s must be must be more neurologically active and sensitive than fast-twitch fibers, to be able to adapt quickly and not set off the stretch reflex under short-term stimuli.** Again, one motor unit innervates a combination of slow and fast-twitch fibers; fewer spikes-per-second of longer duration will excite slow-twitch fibers much faster than it will excite fast-twitch fibers. Slow-twitch fibers are more excitable both because of their structure: they are thin fibers ~ and their nature: they are ATP-driven. Like all slow-twitch fibers, bag1s for the dynamic stretch reflex adapt rapidly to incoming nerve pulses, meaning there's a time lag between the incoming (efferent) gamma nerve pulse and this muscle fiber's afferent (annulospiral) response. You could say their stretch reflex trigger acts something like a slow-blow fuse; and here's why that works well for our purposes:

Think of an electrical power system: in an electrical line, sudden "transient" pulses of voltage can temporarily take the amperage over 20 amps, which will blow a standard 20-amp fuse. But if you put in a 20-amp 'slow-blow' fuse, it will adjust to these sudden transient spikes, even one of 50 amps. A 50-amp transient has to sustain for a period of time in order to heat the element enough to blow a 20-amp slow-blow fuse.

Similarly, because bag1 fibers adapt rapidly, they will not fire their annulospiral nerve endings if the stimulation (stretch) is narrow (short) enough, even if that stimulation comes in rapid repetition. Instead, these slow-twitch fibers that adapt rapidly to nerve pulses coming down the line, will just sit and watch a pulse go by ~ if the pulse is short enough. In order to make the bag1s contract and set off the dynamic stretch reflex, any stimulation must sustain for a period of time ~ for example, those long, slow, deep strokes along the grain of the muscle in deep tissue massage. That's why we stroke across the grain of the muscles, strumming them like a guitar, so are only on each fiber for a fraction of a second. Be clear that this analogy to a slow-blow fuse refers not to the force of the twitch, but to the speed of the nerve pulse coming down the line.

**Bag2 fibers** are fast-twitch, slowly adapting muscle fibers. There are twice as many static bag2 fibers in every spindle as there are dynamic bag1 fibers; bag2s are shorter in length than bag1s, and they attach to the ends of the longer bag1 fibers. As stated earlier, they participate in 3 of the spindle's 4 functions: 1) they generate control of muscle tone in concert with the chain fibers; 2) they generate the static stretch reflex; and 3) they generate stimulation for fine motor movements. This is covered in detail later in this paper under Functions of the Spindle.

It is necessary that there be separate bag fibers for the dynamic stretch reflex (bag1) and the static stretch reflex/muscle tone (bag2) for 2 reasons: 1) chain fibers attach to/hang off the bag2 fibers, and therefore inhibit the reaction of bag2s, and 2) both the chain and bag2 fibers control the continuous, static operation of muscle tone, whereas dynamic bag1 fibers control the intermittent operation of dynamic stretch reflex.

Although static bag2s are fast-twitch, slowly adapting fibers, they perform a continuous, static operation. How do they do that? In 2 ways:

1. **There are more of them.** Although, like all fast-twitch fibers, the static-stretch reflex-against-gravity bag2 fibers are large, creatine-driven, rapidly-fatiguing fibers, there are twice as many of them as bag1 fibers, so they can replace each other as they tire.

2. **Reverse Elasticity, or Creep:** More importantly, the muscular, contractile ends of the bag2s have an elastic property that the bag1s do not have, even though, like bag1s, they are wrapped with annulospiral end organs in their central portions. However, instead of increasing elasticity, this elastic property in the bag2 fibers actually decreases elasticity! This reverse elasticity, known as “creep”, helps the bag2s maintain their length when the
chain fibers attached to them contract; it prevents them from stretching. Were it not for this reverse elasticity, when the flowerspray nerves on the chain fibers were stimulated, instigating the cerebellum to contract the chains in order to relax muscle tone, the annulospirals on the bag2 fibers would also fire their annulospirals, and would consequently be working against the chains.

a. It is lucky for us that the bag2 fibers do have this ability to maintain their length; that gives us our ‘IN’ to the system. It means that during a NeuroSoma treatment, our short pulses of stimulation, our limited pressure cross-fiber strokes that produce quick, short pulses to the fibers – the manual NeuroSoma stroke (and by the way, the BioPulser resonate vibration) that causes the flowerspray end organs on the chain fibers to fire – will not stimulate the annulospirals on either the dynamic slow-twitch bag1 fibers, or on the static fast-twitch bag2 fibers. If we are working correctly, we stimulate only the flowerspray endings on the chain fibers.

Because both the chain and bag2 intrafusal muscle fibers are fast-twitch, they are both slow to adapt to brief changes in neural stimulation. The flowerspray endings on the chains will fire with very short pulses of stimulation, even when the annulospirals on the dynamic bag1 fibers will not. The annulospiral nerve endings on the bag2s do fire every time a pulse of current comes down the line, regardless of the brevity of the pulse, but bag2 fibers do not contract because of creep, as discussed above.

Further, being slow to adapt to short pulses, both chains and bag2s are less responsive to the extrafusal fibers/skeletal muscles. Because their job is to maintain static, constant muscle tone, these fast-twitch intrafusal fibers don’t want to respond to changes outside the spindle, as the bag1 fibers do. Instead of being a beat behind and responding to changes, they keep right up with the movement of the extrafusal fibers.

Chain fibers are fast-twitch, and as you know by now, are shorter than the bag2 fibers from which they hang. The chain fibers actually sense all the activity going on in the spindle because they are 3rd in line and see everything in front of them, and they summate all that information with their flowerspray endings. Then the flowerspray nerves coming off those endings, which have an internal and direct connection with the cerebellum, feed that information back to brain, reporting on how much stress the muscle in the section of the fascicle they monitor is enduring at any given moment, and they tell the cerebellum what commands to send down on the gamma efferents.

This is how the cerebellum knows whether or not it is correctly asking for those 5 pulses-per-second of automatic contraction required for proper muscle tone in that muscle. But this system works only as long as the flowersprays are operating properly.

If the signal coming from the flowersprays is weak, and the brain decides it needs to increase the amount of muscle tone, IT DOES SO BY APPLYING MORE TENSION TO THE STATIC BAG2 FIBER, which can be accomplished either by [1] relaxing the tension on the chain fibers, OR by [2] directly increasing the tension on the bag2 fibers. Either action activates the bag2 annulospirals, which fire out to the spinal cord and synapse with the alpha motor nerves.

1. **Decrease** gamma 2c input to (thereby decrease the tone of) the chain fibers, so they will then relax and transfer more of the tension, or stress they have been carrying across the static bag2 fibers, but leave the dynamic bags unaffected. When the shorter, smaller chain fibers relax, their tension/stress is transferred to the next in line, the bag 2 fibers from which they hang. In a nutshell, when the chains give up their action, their tension spreads across the bag2s, creating hypertonicity.

   a. Remember, the stress is along the length of the fibers, so when the chains stop absorbing the stress, that leaves the bag 2 to absorb it. Whatever the chains let go of, the bag 2s have to take up.

   b. Then the bag2’s annulospirals, which go right back to the spinal cord just like bag1 annulospirals, start driving the extrafusal fibers through the reflex arc

   c. This produces hypertonus out in the extrafusal muscles

   1) The annulospirals always drive the main extrafusal fibers.
2) This is not really a myotatic response, which is more for outside forces; this is an inside response to an inside force.
3) But it doesn’t matter if it’s external or internal, the result is the same: more hypertonus.

2. **Increase** gamma input directly to (and thereby increase the tone of) the static bag2 fiber’s end muscles, to stretch the bag in the middle; this decreases the tension on the bag1 because bag2 is physically taking up some of the stress that would ordinarily go through bag1 *(bag2s act on bag1 fibers much like chain fibers act on bag2s)*
   a. Even though stress is taken off the bag1s, in the early stages of hypertonicity, the dynamic stretch-reflex is exaggerated, not decreased. In order to cause the original hypertonus in what has been a healthy muscle, tension is first added to the dynamic bag1 fiber in a temporary response; the only function of the dynamic bag fiber, remember, is to drive into the alpha motor nerves.
   b. Later on as the vicious cycle of muscle spasm sets in, and as the tone of the skeletal muscles and static bag fibers increases, the dynamic stretch reflex actually decreases; the muscle becomes more and more contracted, so there is less opportunity or even need for the reflex guarding reaction of the bag1 fibers to dynamically contract the muscle.

Remember that the static stretch function of static bag2 fibers is driven by gravity; that’s why bag2 fibers exist. When discussing the static portions of the muscle spindle, all most texts talk about is the static stretch reflex; they don’t mention muscle tone or fine motor control. Most discuss the static stretch reflex only in terms of what happens after the dynamic stretch reflex has ended, mentioning a ‘trail-off’ response (because bag2s do respond to dynamic actions, and so have to recover. Even though static bag fibers are inhibited by chain fibers, they still react dynamically). And although some texts do mention the 5 pulse-per-second signal coming out of the spindle to drive muscle tone, they never explain where it comes from.

**SO the tension on static bag2 fibers is what sets the tone level for any muscle.** Essentially, static stretch reflex is muscle tone. When you are lying down experiencing no real gravity (other than what holds you to the planet of course), muscle tone still exists; because without it you would disjoint. And when you stand up, muscle tones increases with gravity. Muscle tone is still the static stretch reflex ~ it’s still the same mechanism ~ except this muscle tone is driven by external forces instead of by gamma efferent nerves increasing the tension of the intrafusal spindle muscles.

Can pain drive the hypertonus reaction? No. Pain can cause a splinting reaction, which is entirely different and more superficial. All sensory pain nerves impact directly on the alpha motor nerves; they do not go to the muscle spindle. So-called muscle relaxants will work to relieve pain from a splinting reaction, because that is a spinal cord reaction; blocking those synapses at the spinal cord will turn the reaction off. But they only block splinting/guarding reactions at the anterior cord level, they do not reduce muscle tone.

The only drug that has an effect on excess muscle tone is curare, but it’s not relaxing the muscles, it’s just shutting them down/cutting them off; and curare shuts off all the nerves it gets to – it’s not selective. When it wears off, the muscles come back on.

To decrease muscle tone, the cerebellum contracts the chain fibers, which takes stress off the bag2 fibers, thereby stopping their annulospirals from firing.

Remember that a majority ~80%~ of the spindle’s function is to control muscle tone, using the chains and bag2s to do the job. The static stretch reflex system drives muscle tone, maintains contraction against gravity, and controls fine motor-movement; 3 of the muscle spindle sensory receptor’s main jobs are managed by this system. Bag1 fibers only operate the dynamic stretch reflex; but also please remember that the intrafusal bag1 fibers don’t just respond to the chain fiber’s action via the bag2s; they also respond to their main attachment to their skeletal muscle’s perimysium: the actions of the extrafusal fibers. They are driven from 2 directions: the extrafusal fibers above and the bag2s below.
Also remember, chain & bag2 fibers are fast-twitch fibers because theirs is not just a static stretch reflex system; the muscle tone system maintains static muscle tone against gravity, drives muscle tone, and controls fine motor-movements. When we make little delicate movements with our hand, the alpha motor system can’t handle it; so it’s done through our real master movement controller ~ the cerebellum ~ using the muscle spindle. That’s why the hand fatigues readily when doing fine motor movements: it is using fast-twitch fibers.

Griner saw an article about an assembly plant where the employees were doing a particular assembly operation. The study actually had monitors on their gamma nerves to show when the gamma action was decreasing (meaning they were fatiguing their fast-twitch, fine motor-control spindle muscles) and the employees would take a break. That’s why when you move rapidly, you don’t have the same coordination as when you move slowly. You’re moving too fast for the muscle spindle, with its gamma nerves, to keep up with you. When you really want to do something serious, you slow down.

When we learn something physical, we learn it slowly and eventually speed it up. All of that issues from the little cerebellum, which knows the movements to make and informs the motor cortex what is available, so the motor cortex is aware of the movements being made. Although we’re told that the motor cortex controls movements, it does not; it signals the cerebellum to control the movements. Additionally, all of the muscle’s nerve feedback is going directly to the cerebellum.

So even though the gamma afferents leaving the spindle are slowly-propagating nerves, they have the advantage that the feedback information is not traveling all the way up to the cerebrum, but only to the cerebellum, giving them a fast turnaround for corrections. Therefore the information comes down rapidly, using the extra-pyramidal nerves. It is only when the nerve impulse reaches that part of the spinal cord where the gamma nerve takes off that it slows down, and from then on it moves slowly in the gammas.

The extra-pyramidal nerves are smaller than the pyramidals (the latter are the largest nerves in the body), but they’re not gamma-small. They’re like alpha 2’s. A gamma is 5 microns while an alpha is 14 microns. An extra-pyramidal is somewhere in between. A gamma nerve isn’t a gamma until it leaves the spinal cord.

It’s very difficult to separate the gamma motor nerves going to the dynamic bag1 fiber from the gamma motor nerves going to the static bag2 and chain fibers. Difficult, but it can be done. However, there are not just gamma 2 fibers, there are gamma 2b and gamma 2c. One is for the chain fibers, and one for the bag fibers, and it is really impossible to separate those two. You cannot detect or separate the action of one from the other.

E: LYMPH SYSTEM OF THE MUSCLE SPINDLE

Conventional thinking says that skeletal muscles do become inundated with lactic acid after strenuous use, but with enough time post-exercise, the flow of blood eventually flushes the lactic acid out of the tissues. In fact not all of the lactic acid gets flushed out, regardless of resumed blood flow or amount of time lapsed; some remains trapped in the spindle cells.

Each muscle spindle has one lymph duct attached to it ~ a single lymphatic drainage ~ which pulls fluids out of the spindle; but it also pulls fluids into the spindle through the perimysium and endomysium. Think of it this way: you wouldn’t want to have the air circulator intake on your car next to the exhaust.

Remember that the lymph system is part of the circulatory (and immune) system. The circulatory system utilizes skeletal muscles to pump venous blood back towards the heart. It is true that once the level of lactic acid has receded in the surrounding muscle tissue, the duct then acts to help flush the spindle and dilute any acid inside the spindles.

Nevertheless, during intense activity, injury, or lack of activity to the point of seriously reduced circulation, levels of acid can accumulate to the point that muscle contraction becomes set in and locked for a period of time, particularly in people whose muscles are already somewhat hypertonic. This locked contraction continues to produce lactic acid and to block circulation in the veins and venules, whose thin walls and so-little blood pressure make them susceptible to being squeezed shut by the contracting tissue.
There are no lymphatics inside the perimysium (there’s nothing in there but capillaries, thoroughfare channels, and nerve twigs terminating in motor end plates to the fibers); so in order to get out to the interfascicular spaces where the lymphatics are located, everything that collects on the inside of the perimysium must drive itself out of the perimysial tissue by concentration gradient. This is not an immediate process.

Meanwhile, the lone lymph duct is actually trying to remove the lactic acid; but when the surrounding muscle is so full of it, that one lymph duct, designed to draw fluid out of the spindle, ends up doing the opposite. That’s how some lactic acid generated by the extrafusal fibers will migrate its way into the spindle.

That is primarily why the spindle membrane acts as an accumulating repository for lactic acid. Another reason is that the shape of the spindle organ (fusiform) tends to trap the acid.

Because the NeuroSoma® treatment releases trapped lactic acid, treatments sometimes produce an almost immediate histamine reaction of redness, whealing, or welts on the skin. Histamine is an organic compound involved in local immune responses, and is the body’s first line of defense, the pawns on our physical chessboard. When sudden bursts of lactic acid are released, histamine pours forth to attack. How much skin irritation develops depends on two factors: the 1st is the level of spasticity in the subject’s suboccipital muscles, as very spastic suboccipitals cause the mast cells throughout the body to produce large amounts of histamine. The 2nd is how far away from the surface, how deep into the muscle belly, the release is occurring. The deeper it is, the less likely we are to see the reaction at the surface; therefore only in the early stages of treatment do we see a very rapid or large surface response. Then as more and deeper layers of muscle are softened, we see less welting. It’s still happening, but it’s not reaching the surface where we can see it.

F. FUNCTIONS OF THE MUSCLE SPINDLE
The muscle spindle acts as both a mechanical synapse and an electrical synapse. It receives electrical, efferent signals through the gamma efferent nerves, and converts those into mechanical muscle contraction. Then the annulospiral nerve end organs take that mechanical reaction, i.e. contraction of the spindle, summate it with the action of the main muscle fibers, and convert all of that back into an electrical signal that is fed out through the posterior route into the spinal cord, and in a reflex arc, stimulates alpha motor nerves back into the main muscle fibers, causing them to contract. The flowerspray nerve end organs also summate mechanical contraction, but they feed directly back through gamma afferents to the cerebellum with a report on the amount of contraction taking place in their adjoining skeletal muscle. Ordinarily, synapses only feed forward from the CNS, and only allow electrical information to be summed; summations are what synapses are about. However the muscle spindle synapse summates both mechanical and electrical signals and is the only organ in the body to do so.

Again, the muscle spindle’s 4 assignments are to 1] activate the dynamic stretch reflex mechanism (20% of spindle function) using the bag1 fibers (both ‘dynamic’ and ‘myotatic’ refer to a rapid, powerful contraction that shows up on an EMG as a sudden sharp spike of activity; 2] control fine motor movements using the bag2 fibers; 3] maintain the static stretch reflex using the bag 2 fiber, (automatic, steady, balanced, constant muscle contraction against the constant force of gravity, in equilibrium; it shows up on an electromyograph (EMG) as almost nothing, or as background noise, regardless of how many fibers are involved); and 4] control and maintain muscle tone (80% of spindle function) using bag2 and chain fibers. Let’s look at these 4 functions of the spindle cell in more detail:
Spindle Function 1 ~ Activate the Dynamic Stretch Reflex Mechanism

The following text is taken from Guyton’s Textbook of Medical Physiology:

"Whenever a muscle is stretched, excitation of the spindles causes reflex contraction of the large (extrafusal) skeletal muscle fibers that lie around the spindles. In the basic circuit of the muscle spindle stretch reflex, a type Ia fiber (annulospiral nerve) originates in the muscle spindle and enters the dorsal root of the spinal cord. Then, in contrast to most other nerve fibers entering the cord, one branch of it passes directly to the anterior horn of the cord gray matter and synapses directly with anterior motor neurons that send nerve fibers back to the same muscle from whence the muscle spindle fiber originated. Thus, this is a monosynaptic pathway that allows a reflex signal to return with the shortest possible delay back to the same muscle after excitation of the spindle.

To emphasize the importance of the gamma efferent system, one needs to recognize that 31 percent of all the motor nerve fibers to the muscle are gamma efferent fibers rather than large, type A alpha motor fibers. Whenever signals are transmitted from the motor neurons, almost always, the gamma motor neurons are stimulated simultaneously, an effect called co-activation of the alpha and gamma motor neurons. This causes both the extrafusal and the intrafusal muscle fibers to contract at the same time." (The last paragraph emphasizes the complete involvement of the muscle spindle in muscle physiology.)

So when the spindle cell senses that its muscle is being stretched, it reacts by causing the muscle to contract to a degree roughly proportional to the speed-rate of the applied stretch, in order to preserve its muscle’s dimensional integrity. In other words, the SPEED with which a stretch is applied to a muscle is automatically countered by a contraction at the same speed, in order to keep your joints from dislocating. About 20% of the muscle fibers in the spindle cell, the bag1s, are there to produce this reaction-to-action, the counter-balancing dynamic stretch reflex contraction.

The dynamic stretch reflex is generated as a protective mechanism when the spindle and its skeletal muscles are being stretched. During stretch, the annulospiral endings on the bag1 fibers transmit an action potential to the CNS at the spinal cord; the frequency of the action potential is proportional to the SPEED of stretching. As the spindle organ is stretched — becomes longer — the impulse frequency in the gamma afferent nerve increases, firing into the alpha motor nerves in the spinal cord, causing them to fire into the skeletal muscle to cause it to contract. As the skeletal muscles contract, the intrafusal chain fibers shorten, thereby relaxing the tension on the bag2 fibers, and reducing the discharge rate of the gamma afferents on its annulospiral endings.

Many books claim that spindles signal the length of the skeletal muscle to the brain by the frequency of their nerve pulse to the spinal cord. Griner says, however, that the cerebellum operates not just by information it receives from the spindle but also from information it receives from joint receptors, and it is these that report the angle of the joint and the rate-of-change of angle. And contrary to popular belief, the cerebellum uses that information to determine the length of the extrafusal muscle fibers, not what it hears from the spindle organs. It is important to understand that the flowerspray endings do not measure either the length, or the rate-of-change of length of their extrafusal fibers; they only measure the stress, or force their muscle endures when at rest. The amount of tension in a resting muscle = the amount of muscle tone.

The dynamic stretch reflex nullifies muscle tone. The stretch reflex will produce enough force to maintain the position of the joints as long as it is active, so there is no need for muscle tone. Muscle tone only operates in a resting muscle. However, once it becomes excessive, it never shuts off.

The ends of the Bag1 fibers controlling dynamic stretch reflex are attached to the perimysium of their fascicle at 2 different points; consequently, information about the length, and rate-of-change of length of the working muscle fiber is physically transferred into the spindle. When the Bag1 fibers (and the bundle of muscle fibers to which they are attached) begin to stretch, their annulospiral nerves traveling back to the spinal cord through the posterior route fire, activating alpha nerves that then drive forward from the spinal cord anterior route to
The extrafusal fibers (this is called a ‘reflex arc’), sending a compensating signal to the muscle to contract.

The dynamic stretch reflex is a response to an outside force, something happening to the muscle. It delivers a powerful, short-term contraction that will override and nullify the constant low-level signal of muscle tone, providing the skeletal muscles are relatively healthy and not in hypertonic spasm.

Spindle Function 2 ~ Control Fine Motor Movements

The alpha motor nervous system can’t handle very small, delicate movements in the hand; these are carried out by the real master movement-controller – the cerebellum, through the muscle spindle; and because these fine-movement muscles are fast-twitch, they fatigue readily. Additionally, fast-movement coordination isn’t as precise as slow-movement coordination, because the muscle spindle, with its little gamma nerves, can’t keep up if the movement is too fast. Griner read an article about an assembly plant where the employees carried out a particularly delicate assembly operation. The participants in the study actually had monitors placed on their gamma nerves to show when the gamma action was decreasing, indicating the fast-twitch, fine motor control spindle muscles were fatiguing. Then the employees would take a break.

Spindle Function 3 ~ Maintain the Static Stretch Reflex

Bag2 fibers controlling static stretch reflex also travel to the spinal cord and activate alpha motor nerves back to the extrafusal muscle bundle in a reflex arc, but they are reacting to the DEGREE, or amount of stretching force occurring in the extrafusal bundle of fibers, rather than the speed of application. Although the static stretch reflex is predominately designed to provide support against the steady pull of gravity, any force great enough will activate this contraction, which will not rapidly dissipate, as does the dynamic stretch reflex contraction, but will remain until the activating force is removed, including passive stretch and applied pressure.

What exactly sets the tone level for a particular muscle? The tension on the static bag2 fiber. We’ve said several times in this paper that bag2 fibers have three functions: muscle tone control generation, static stretch reflex generation, and fine motor control generation ~ and that essentially, static stretch reflex is muscle tone. When we lie down with no gravity at work, muscle tone still exists because without it, we would disjoint. When we stand up, muscle tone increases; they call it ‘static stretch reflex’, but it’s still the same mechanism as muscle tone, except that it is driven through external forces, whereas muscle tone is driven by internal forces – the cerebellum causing gamma efferents to control tension of intrafusal muscles. So this is why bag fibers must be involved in muscle tone control – they are the actual generators of the signal for muscle tone. And remember, the bag2s are located next to/just under the bag1s, and are taking the stress off the bag1s, allowing the latter to sit waiting, quietly, comfortably, in case they are suddenly needed to fire their annulospirals to contract the extrafusal fibers.

When increased muscle tone is generated by the static bag fiber as opposed to the chain fiber, it is because the body does something that increases muscle tension against gravity, and then the cerebellum causes more tension to be applied to the static bag fiber. It can do this in 1 of 2 ways: 1] it can either increase the gamma efferent input to the static bag fiber end muscles, which contracts the ends and consequently stretches the static bag in the middle of the fiber; or 2] it can decrease the gamma efferent input to the chain fibers, which relaxes them. That transmits the force, or tension they were carrying to the static bag fiber. Remember, the chain fibers attach to and actually take the stress off the bag2 fibers.

The chain fibers are involved in activating the flowersprays, which are the feedback to the cerebellum; they are busy monitoring muscle tone, so it’s doubtful that they are also generating muscle tone. These flowerspray nerve endings are the very target NeuroSoma® aims for. We are actually trying to get them to fire, because they attach to afferent, sensory nerves going to the cerebellum in pathways we are attempting to clean up, or remove distortion from. But we need to stimulate them to fire without also stimulating the annulospirals to fire, which we’re able to because they are attached to fast-twitch chain fibers that adapt slowly, providing we stroke across the grain of the muscles, (stimulating each fiber for only a second) and use a pressure not great enough to activate the stretch reflex. The bag fibers will just sit
and watch these little stimulating pulses go by without firing their annulospirals. The faster our pulse is moving, the greater the rate of change will be, and the greater the rate-of-change in a slowly adapting system, the greater the neural output back to the cerebellum. That does NOT mean our stroke should be faster than we can perform perfectly, by the way.

Most books mention only the static stretch and the dynamic stretch, and most do not mention the main spindle function of muscle tone. Some books say annulospirals wrap both the chains and the bags. Some show the flowersprays as annulospirals, some show flowersprays on bag fibers. They talk about proprioception, about the spindle going silent, and its relationship with the Golgi tendon organ. Many times the text of a book doesn’t match up with its pictures. They say the annulospirals are afferent and never mention their efferent role in driving the skeletal muscles. They never mention exactly where the spindle is located or to what it is attached. A few books mention and explain fine motor control, and one correctly says that muscle tone comes out of the muscle spindle; thank you! Where else could it be coming from? It has to come from a place where there is an annulospiral nerve, because muscle tone has to be able to drive the extrafusal fibers. Muscle tone comes from the muscle spindle.

Because both the dynamic and static stretch reflex mechanisms instantly contract skeletal muscle tissue as a guarding reaction when the muscle is stretched past a certain set limit – whether that stretch is active or passive – the NeuroSoma® therapist applies a limited pressure to avoid turning on automatic contraction and irritation of the muscle.

As said previously, the important aspect of working across the muscle fiber is that it does not turn on the stretch reflex. Muscles can only take a static contact of 6 ounces before turning on the stretch reflex, therefore we must work across the fibers; if we work along the top and/or length of the muscle belly, we’re reduced to working with less than 6 ounces of pressure, but then can’t create enough stimulation to the nerve body to make any changes. By coming across the grain, we’re just pulsing on the muscle fibers, and can increase that pressure somewhat.

**Spindle Function 4 ~ Control & Maintain Muscle tone**

Muscle tone is a tricky subject. Gray’s Anatomy empirically denies that it even exists, claiming that contraction in a resting muscle cannot exist if it discharges no motor units that show up on an EMG. However an electromyograph can’t pick up low-level, steady-state action unless the motor units immediately adjacent to the contact are firing. It just reads as background noise.

Other authors postulate that rather than a system of contraction to maintain muscle tone, there is an inherent elasticity in the muscles; and still others talk about muscle tone but don’t have a clear picture of what it is. They confuse muscle tone with muscle contraction to resist the constant pull of gravity, which is the static stretch reflex. Now even though this isometric contraction against gravity (internal against external forces) is a much greater contraction than muscle tone (internal forces only), it doesn’t show up very well on an electromyograph either, because it is also static and balanced; but no one denies its existence.

Because muscle fibers twitch, they have to do so in synergetic, co-active, symbiotic motor units. In other words there are overlapping motor units so that when one relaxes, another one comes on. So the contractions of muscle tone are turning on and off, but overlapping so smoothly and steadily, with no spiking contraction, that the electromyograph doesn’t read it. All that twitching, yet nothing shows up. In regular skeletal muscle contraction of course, the electromyograph shows muscle contraction very clearly as spikes, and can measure how many pulses per second as well as the strength of each pulse.

True tonus is static and balanced, internal force against internal force, operated internally through the cerebellum and spindle. Muscle tone is a response to an inside pre-program in the cerebellum. The cerebellum is self-contained; it doesn’t want to know anything about outside sources, it already has all the information it wants, and is just waiting to distribute it. You probably know someone just like that.

Muscle tone is properly defined as ‘the amount of contraction in a resting muscle’, or even as ‘resistance to stretch’; a more detailed definition would be ‘a static, balanced, isometric contraction between agonist and antagonist (both internal forces) in every muscle in the body, for the purpose of maintaining joint integrity and posture’. Muscle tone is static because the job it has to do doesn’t change; it is a basal metabolic function. It never lets go completely, even in
sleep, when all dynamic muscle activity is gone. The static activity remains, just sitting there. It operates when one does nothing, and doesn’t change very much from that point. Muscle-building exercise does elevate tone, and can even slam it up considerably; but at whatever level the involvement of contraction, tone still remains static and constant at any given time.

And as we know, muscle tone can get totally out of whack and create unhealthy muscles to the extent of making the whole body ill. But it’s still static. So how is muscle tone increased? It’s done in two ways, which we will talk about when learning more about the individual spindle fibers and nerves.

For the purpose of muscle tone maintenance, the spindle only measures the amount of stress, or force endured by the extrafusal muscle fibers. It does not measure the length of the fibers, it doesn’t care how long they are. The spindle wants to keep the skeletal muscles at a certain level of contraction regardless of their length. Nor does it measure the rate-of-change of length of the fibers, because muscle tone is not about the stretch reflex, it’s about a constant contraction. And because the function of muscle tone is to maintain minimal tone – just enough to keep joint integrity – it measures extrafusal fiber stress only in a resting muscle.

When a healthy muscle is in motion, the alpha motor drive to the muscles is activated, and produces more than enough skeletal muscle contraction to maintain joint integrity, so the low-level contraction of muscle tone fades into the background. Providing the muscle is healthy. However, once muscle tone becomes excessive, the muscle is no longer healthy. For example a body builder is a mass of hypertonic spasm, and his muscle tone is more powerful than his stretch reflex response.

All muscle contraction begins in the core of that muscle. As more contraction is needed, fibers further out towards the surface contract. Even in flat muscles like latissimus dorsi, contraction starts in the central plane, and then works its way out. In very thin muscles such as the eyelid, even low-level contractions will involve the outer layers; but a quadriceps muscle standing against gravity (a soldier standing at Parade Rest for example), using only low levels of contraction, will show a lot of activity in the deep layers of the muscle but none in the superficial layers. The core of the quadriceps has enough power that it doesn’t need the superficial fibers for this job.

Hypertonic contraction also begins at the core of the muscle; and as the muscle gets sicker (hypertonic muscle isn’t actually ‘sick’, it is working just fine; but it is being overworked, like a horse that is never allowed to stop turning the stone in a gristmill), the ongoing process of over-contraction extends further out toward the surface of the muscle. But minimal, healthy muscle tone contraction is 5 pulses per second. Summation of the twitches of many fibers excited asynchronously at low frequencies ~ up to 5 per second ~ generate a total force that does not fluctuate. This is proper muscle tone.

Here are some cites from the varying texts regarding muscle tone so you will be prepared for the confusion you are bound to meet. As you will see, it gets better with each succeeding author; some are willing to butt heads with Gray’s. But the full picture is very hard to find.

Gray’s Anatomy: “At one extreme a muscle may be fully relaxed when much electromyograph evidence shows no activity, the muscle is electrically silent. This contrasts with earlier accounts by electro-physiologists who held that full relaxation was still accompanied by background activity rotating among a few units. Misconceptions concerning such muscle tonus were based on these views. In that the text is not titled “Gray’s Physiology”, it is hard to understand why he is even weighing in.

Anatomy of the Human Body by Lockhart, Hamilton and Fyfe: “Some writers discard the term ‘muscle tone’ or define it as the response of skeletal muscle to stretch. The electromyograph registers no activity in a resting intact muscle, but such a muscle is certainly different from one with its nerve supply cut. It may be that muscle tissue itself has an intrinsic elasticity.” (But if that were true, we’d have to be fighting against that elasticity every time we used a skeletal muscle.)

Guyton’s Physiology does at least accept that muscle tone is the residual contraction in the relaxed muscle. He says: “Even when muscles are at rest, a certain amount of tautness usually remains. This is called muscle tone. Since skeletal muscle fibers do not contract without an actual action potential to stimulate the fibers except in certain pathological
conditions, skeletal muscle tone results entirely from nerve impulses coming from the spinal cord. These in turn are controlled partly by impulses transmitted from the brain to the appropriate anterior motor-neurons and partly by impulses that originate in muscle spindles located in the muscle itself.”

**Structure & Function of the Human Body, Memmler & Wood:** “Muscle tone refers to a partially contracted state of the muscles which is normal even though the muscles may not be in use at the time. The maintenance of this tone or tonus is due to the action of the nervous system, and its effect is to keep the muscles in a constant state of readiness for action.”

And finally, **Fundamentals of Neurophysiology by Schmidt:** “Summation of the twitches of many fibers, excited asynchronously at low frequencies up to 5/s, generates a total force that does not fluctuate very much, with an amplitude that must be approximately proportional to the average frequency of excitation. The ‘background’ tension produced in this way by summation of the twitches of many fibers is called tone. All the muscles in a living organism possess such tone. Even in a relaxed limb, the motor nerves are activated at low frequency.”

As therapists, we need to be aware of a couple of things. The term ‘post isometric relaxation’ means that the muscles can be relaxed by first contracting them isometrically; but something else is actually happening: skeletal muscles have both slow-twitch fibers, which don’t fatigue easily, and fast-twitch fibers, which do. When one holds an isometric contraction, the fast-twitch fibers soon fatigue to zero. All contraction begins as a combination of fast and slow-twitch fibers, but when the fast-twitch drop out, and only that contraction from the slow-twitch fibers can be felt, the muscle feels softer than it did before. But the difference in muscle tension is fatigue, not relaxation. Be aware that the fast-twitch fibers rejuvenate in about 15 minutes and the original tension resumes. The mechanism that allows them to reset, and come back into contraction, is that circulation resumes in the tissue after the initiating force is removed.

Moreover, we must not fail to notice the background tension of muscle tone (such as resistance to passive bending of a limb), nor resistance in the superficial muscle if we’ve turned on the stretch reflex. Deep tissue therapists think they are reaching the deep muscle when they feel that resistance, so they go in deeper, push and hold deep into the muscle, until they gradually feel the muscle start to soften and melt. And what is that softening again? It’s the fast-twitch fibers fatiguing. And when it comes back, it’s back with vengeance because the stretch reflex mechanism has been activated, which powerfully contracts muscles, and eventually increases muscle tone.

Chiropractic adjustment typically does 3 things: it fatigues the fast-twitch fibers, it inhibits the superficial muscles by increasing the activity of the deep muscles, and in jerking the deep layers and activating the stretch reflex, it activates the Renshaw cells (interneurons in the spinal cord that inhibit motor neurons). The Renshaw cells are automatically coupled in, so that the more activity there is in the core of the muscle, the greater the inhibition to the superficial muscle. The feeling of softness is therefore even more exaggerated post-chiropractic adjustment than it is post-deep tissue massage.

So proper muscle tone is how many contractions per second? 5. And how many muscles have 5 pulses per second in the relaxed state? Not very many. Why? Because most of most of us are hypertonic. This excludes cardiac muscle, and also flat muscles, which do stay at about 5/s because they can’t poison themselves. Flat muscles resist becoming hypertonic because they can’t squeeze off their veins so they can’t encapsulate lactic acid. Cardiac muscle is non-spastic because it can burn lactic acid, and the number of pulses in cardiac muscle all depends on what the heart is doing. The heart gets excited to react to the conditions of the body, and is never in a relaxed state, except between beats. But neither are skeletal muscles ever truly relaxed; tone works 24 hours a day. We stand in awe of the heart muscle, but skeletal muscle deserves our appreciation and respect too.
The cerebellum is directed to maintain a constant, low level of automatic contraction in all skeletal muscle in order to maintain joint integrity and tension against gravity. For this purpose, the little brain relies on information it receives from tiny flowerspray feedback nerves issuing from chain fibers in muscle spindle nerve bodies, informing the cerebellum on the amount of automatic contraction, or ‘muscle tone’, the brain is producing in each muscle at any given time.

But lack of circulation in a muscle can allow lactic acid, a metabolic waste product, to reach a concentration level high enough to spill over and invade the spindles, where it sickens the flowerspray feedback nerves, weakening and distorting their signals to the brain. (The flowerspray ending is more exposed and provides a finer contact point than the annulospirals.) The cerebellum interprets that drop in signal to mean the tension in the extrafusal fibers has dropped, and believes it needs to ramp up the amount of contraction it is requiring from the muscle in order to maintain proper muscle tone (and remember, the cerebellum is disassociated from the extrafusal fibers; it determines the muscle’s feedback only through the spindle). The cerebellum has a set program, which it accesses, in response to what it thinks is this too-little tone situation; it relaxes the static chain fibers by decreasing their gamma input. This is turn creates more tension on the static bag2 fiber, which then fires its annulospirals in a reflex arc into the main muscle. The brain not only recruits more fibers within the muscle into automatic contraction in an attempt to reach its ‘set point’, but also ramps up the rate of contraction.

This increased contraction squeezes down on venules even more, further restricting circulation, and produces more metabolic waste; reduced circulation and more lactic acid, both in the muscle and then in the spindle, weakens nerve feedback even further, making the cerebellum call for more contraction. A vicious cycle sets in, where eventually the muscle becomes permanently hyper-contracted, or hypertonic; it has too much muscle tone. So that’s how the muscle becomes hypertonic. And in case you missed it, the little chain fibers and their flowerspray nerve end organs are the sickos here, and the target of NeuroSoma® muscle therapy!

Because of the brain’s ability to produce internal morphine, or endorphin, this overly contracted hypertonic muscle may or may not be in discernible pain; but inevitably, physical problems, ranging from premature aging to real illness, will result.

The goal of NeuroSoma®: We are stimulating flowerspray feedback nerves to increase their output, to in a sense lie to the cerebellum. We’re laying one lie on top of another, creating one lie to cancel out another. The original lie was the inadequate feedback from sickened flowersprays, telling the brain that not enough extrafusal fibers were being contracted. We give a corrective stimulation – not a proper 5 pulse per second stimulation; but it does correct for the fact that the signal is too low, and instructs the cerebellum to relax hypertonic muscle. It’s like a corrective lens, using one problem to counteract another problem within the eye. By stimulating the flowerspray nerve feedback, the cerebellum begins to believe the tone in the muscle is so high that it must re-tension the chain fibers, which takes the stress of the bag1s and stops the process of increasing contraction in the main muscle. NeuroSoma therapists obviously want to avoid stimulating bag1 fibers into firing the dynamic stretch reflex, so apply only sub-stretch reflex pressure; we also try to get in and out rapidly by stroking across the muscle fibers, and limiting the number of strokes in any one area. When working with a new client, we may do a couple of sessions where it seems like nothing is happening to the muscle tone. We have to bring the entire muscle tone system close enough back to the null point – having all elements equal to zero – for the system to start operating again. Then suddenly, on the 3rd treatment, there is a magnificent step change. The system just got close enough to the null point for it to start operating again, to know where it is and where to go.
H. THREE DEPICTIONS OF THE MUSCLE SPINDLE

From Gray's Anatomy demonstrating the confusion common in depicting the spindle; note the annulospiral nerves on both types of bag fibers as well as the chain fibers, and note the flowerspray endings on both bag 1 fibers but missing on the chain fibers. Gray's text material says the flowerspray endings are located on the chain fibers and the annulospirals are only located on the bag fibers. The drawing has no reference to the
location of the spindle nor does it mention the most important function the spindle performs – maintenance of the muscle tone system.

Drawing by Minette Minnaar, an engineer at the Koeberg Nuclear Power Plant near Cape Town, South Africa. When she took the NeuroSoma course she became obsessed with understanding the spindle, and drew this picture based on Griner’s drawing. Note that it does depict the annulospiral and flowerspray endings and nerves, whereas Griner’s does not.
Muscle Spindle Sensory Receptor by Thomas Griner

Rendering of the working spindle by Thomas Griner.
Footnotes

1\textit{RE location of the muscle spindle sensory receptors: Question:} Why aren’t spindles placed inside their fascicles, attached to individual extrafusal fibers’ endomysium, in order to offer first-person witness? \textit{Answer:} Because there’s no room for them in there; the space inside each fascicle is tightly packed already.

Think of a muscle – say the biceps brachii. You know the muscle belly is separated into fascicles, or bundles of fibers. Why do you think those separations exist? They exist because the corridors between those bundles are necessary: not for any neurological purpose\(^*\), but to provide avenues for everything that supplies the muscle itself. Along with arteries and small veins, nerve trunks run between the individual muscle bellies and send off nerve branches that penetrate into a muscle through the epimysium to run between the fascicles. Alongside the nerve branches in these interfascicular corridors lay arterioles, venules, lymph ductules, muscle spindles and blood maxi-shunts.

Then nerve branches divide into nerve twigs that pierce the perimysium; let’s follow them and go the next step down to penetrate the perimysium into the intermysial or interfibrillar space (between the muscle cells). Now we’re inside the fascicle; here we see that each cell, or muscle fiber, is wrapped in endomysium, which is pierced by those nerve twigs terminating on a motor end plate.

In addition to nerve twigs, in these interfibrillar quarters there are capillaries and thoroughfare channels (aka mini-shunts \textit{fan arteriolet plus a venulet equals a thoroughfare channel, which is a mini-shunt}) that are much larger than the capillaries. And that’s why we have these quarters, or separations: to present a pathway for all these structures to get in.

That is also why the idea of having fascial adhesions is preposterous, because all these structures would get in the way of the fascia adhering together. And pleurisy, which is fascial adhesion, is incredibly painful. Whenever there is fascial adhesion, you’ve got big trouble – but not what the bodyworkers are talking about. Griner claims it’s total nonsense.

\*\textit{RE the neurology of muscle fascicles ~ Just to be certain you understand this:} I just said above in the previous footnote that separation of muscle into bundles, or fascicles, is required in order to provide corridors, and not for any neurological purposes; interfascicular corridors are strictly for logistics as explained above. Don’t be confused in thinking that fascicles contract as a motor unit; motor units are not confined to one fascicle. Innervation passes from one fascicle to another, and as it gets further out into the muscle, that motor unit is spread out over many fascicles. Neurological function is controlled by the \textit{distribution of the nerves}, and one nerve goes to several different fascicles. For example, motor units on a round muscle would be distributed in an arc around the muscle. On flat muscles, the nerves are distributed in a flat plane, but still across different fascicles. And on top of the motor units are the synergistic motor units; once there is a certain circle of motor units, muscles twitch on and off. When one twitches off, another twitches on seamlessly, smoothly, so they are coordinated with each other unnoticeably. That’s what ‘synergistic coupling’ means. Separate muscles are also neurologically coupled together, but not by a single nerve at the level of the muscles; they are synergistically coupled back at the spinal cord, because that’s where the time-loop system is located that causes the asynchronous firing of the motor units to take place.

2\textit{RE Golgi tendon organs:} We often read that the muscle spindle is “in parallel” with the fibers, and the Golgi tendon apparatus is “in series” with the skeletal muscles. The muscle spindles are adjacent to – attached along the outside of the fascicle wall of – the muscles, and so are in parallel with the extrafusal muscle fibers. Be aware also that ‘in parallel’ and ‘in series’ are just anatomical designations, rather than neurological.

The Golgi tendon organs are attached to the side of and at the end of the tendon fibers, so are not ‘in series with the fibers’, but ‘in parallel with the tendons’. The tendon itself is in series with the muscle belly, so the Golgi organs are actually \textit{in parallel with the tendons that are in series with the muscle}. In order for them to be in series, you would have to break all the tendons and put the Golgi’s in series with the tendons, i.e. tendon-Golgi-tendon-Golgi etc.
Golgi tendon organs attach between the fascia and tendon; they monitor tendon stretch at the end of the tendons. (For a long time it was thought that tendons and the perimysium were all one continuous piece, because they’re both tendinous material; that at a muscle’s end, the fascia surrounding the muscle layers was continuous with the tendons, since each fascicle in the muscle belly is identical to the fascicles found in the tendon. But after the advent of the electron microscope, a separation between the 2 kinds of tissue was discovered.)

If tendons are overstretched, the Golgi organs shut down (inhibit) that stretch; and of course they don’t operate at low levels because if they did, they would be in the way all the time. Golgi’s only operates at very high levels of stretch, and you can’t activate the Golgi without first activating the muscle spindle.

It is sometimes erroneously thought that ‘tendon reflexes’ refers to Golgi tendon organ activation. Even knowing that Golgi organs inhibit muscle reaction, the connection with inhibition on the one hand, and the knee jerk from a tap to the reflex on the other, isn’t made.

It is actually the muscle spindle that is being activated in a knee jerk reaction; it’s confusing because it is called a ‘tendon reflex’, but that’s only because you strike the tendon; striking just under the patella is hitting the tendon, not the Golgi organ above the knee at the end of the muscle belly. Striking up where the Golgi organ is located would be striking in the deflective portion of the muscle belly, where only a local action from the deflection of the giving fibers of the muscle could be measured. Striking the less deflective tendon below the knee activates the entire muscle.

So remember that the Golgi organs actually inhibit muscle contraction, and that all tendon reflexes ~ knee, elbow, ankle etc. ~ are measured by hitting a tendon, not a Golgi organ, and that the resulting muscle contraction is caused by the muscle spindles through the myotatic stretch reflex.

Incidentally, because Golgi’s do inhibit muscle contraction, some people believe that mashing down on the Golgi will shut off a spastic muscle. You know now that smashing the Golgi activates the spindle-driven stretch reflex. In any case, trying to get in deep enough to work on a Golgi organ would activate the stretch reflex, and the spindles would turn the muscle on so tight, the deeper layers where Golgi’s are could never be reached.

Finally, there is no neurology to the Golgi organ other than collateral nerve branches coming off nerves to the muscle that run to the tendon to activate it to stiffen up when its muscle contracts. If you remember from the section of Lecture 2 covering ligaments and tendons, tendons are piezoelectric crystal; they stiffen when neurologically excited. Without these collaterals, they would stretch when the muscle contracted, thus counteracting the muscle’s contraction.