INTRODUCTION TO SOMATIC DYSFUNCTIONS

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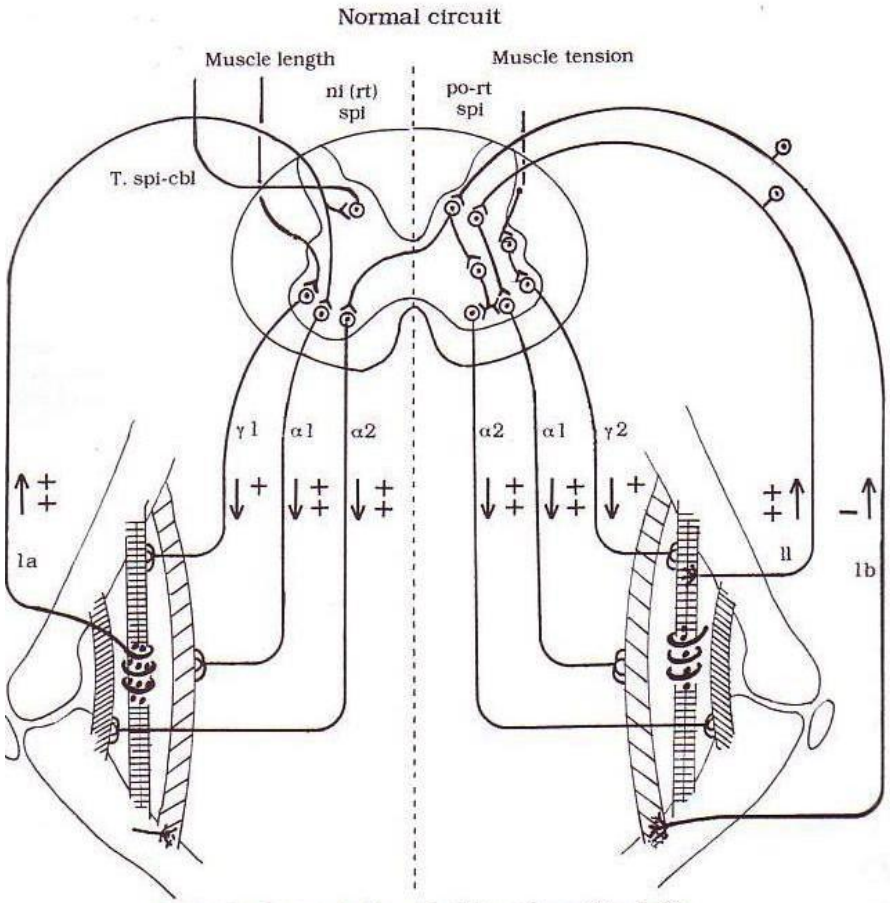

Control of muscle length (circuit on the left) and muscle tension (right) with the appropriate reflex arc (after Hassler)

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NEUROMUSCULAR SPINDLES (NMS):

Neuromuscular spindles are highly specialized structures, distributed within the muscle belly of skeletal muscles. They are considered by some authors as one of the most complex and sophisticated sensory organs in the human body, followed only from visual photoreceptors. About 43% of fibers in a peripheral nerve are sensory, with 20% of them myelinated, and 75% of these fibers originate from spindles.

Neuromuscular spindles are the primary cause of musculoskeletal dysfunctions. Understanding their dysfunctional dynamics is crucial, as it explains the effectiveness of certain techniques and the failure of others.

These structures were first described in 1893 by Ruffini and Kerschner and are part of the receptors for proprioceptive sensitivity. NMS (Neuromuscular Spindles) consist of small bundles of muscle fibers ranging from 3 to 10 mm in length, and width between 80 and 250 µm, wrapped in an expanded connective tissue capsule. This capsule contains a fluid similar to the vitreous humor of the eye, rich in polysaccharides, hyaluronic acid, water, and salts. Spindles are well-supplied with blood vessels and have a complex innervation, both motor and sensory.

The muscle fibers of the Spindles (referred to as intrafusal) are divided into two groups: bag fibers and chain fibers. Bag fibers can be further classified into static and dynamic, each serving different functions related to muscle elongation and speed.

The afferent (sensory) nerve endings within the spindle originate from two types of nerve fibers: type Ia fibers (called annulospiral) and type II fibers (called flower-spray), each with specific characteristics of diameter and conduction velocity. The first, richly myelinated fibers, are mainly responsible for transmitting information about the speed of spindle stretching, relaying the speed of movement in space back to the spinal cord. The latter, partially myelinated but more specific than the former, convey not only information about speed but also about the extent of movement in space, including the distance traveled. This precise information about the position in space, the speed of movement, and the distance traveled reaches the supraspinal centers.

The efferent (motor) nerve endings towards the Spindles come from Gamma motor neurons, which are divided into two subgroups: Gamma I fibers innervate the dynamic subgroup of bag fibers, while Gamma II fibers innervate the static subgroup and chain fibers. This division appears to be related to the need for dynamic and static sensitivity in different movement situations. The activity of Gamma motor neurons is modulated by multiple factors, such as extrapyramidal descending pathways, the activity of Alpha motor neurons, and environmental conditions like temperature. Their activity is crucial for maintaining adequate muscle tension and the correct and coordinated control of movements. The Gamma motor neuron inserts at both ends of the spindle, and when the spindle is in a shortened condition, Gamma, by stretching the end, regulates its tension, allowing for continuous basal muscle tone and ongoing proprioceptive perception by supraspinal systems.

In summary: Neuromuscular spindles transmit sensory information to the spinal cord, which, in turn, communicates with supraspinal centers. These centers receive information about position, direction, velocity, and muscle force, modulating the activity of Gamma motor neurons to regulate muscle tension and ensure accurate spatial perception and movement control. Neuromuscular spindles are highly specialized sensory organs distributed in skeletal muscles, essential for perceiving position, regulating muscle tension, and controlling movements.

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NEUROPHYSIOLOGY OF (NMS):

It is important to understand the neurophysiology of somatic dysfunctions, with an emphasis on the role of neuromuscular spindles during their stretching and shortening.

Spindles are crucial for maintaining posture and for the stretch reflex (myotatic reflex), which is an automatic response to muscle contraction. This reflex can be modulated by the spinal cord and voluntary control. Myotatic reflexes can be divided into two types: the phasic myotatic reflex, responding to rapid muscle lengthening with involuntary contraction, and the tonic myotatic reflex, aiding in long-term posture maintenance.

The phasic stretch reflex is the response that occurs with an involuntary contraction of the skeletal muscle when it undergoes rapid stretching. It is monosynaptic and provides automatic regulation to muscle lengthening. The reflex is modulated at the spinal cord level but is inhibited or enhanced by voluntary action from supraspinal centers depending on the context. Therefore, it regulates muscle length, safeguarding muscle integrity in the event of sudden stretching through a quick and stereotyped response. The inappropriate term "osteotendinous reflex" refers to the response of a hammer on the tendon, causing a momentary stretching of the muscle with deformation and subsequent excitation of the spindle, leading to a contractile response of the muscle. This reflex, which can also be consciously regulated, is a phasic myotatic reflex.

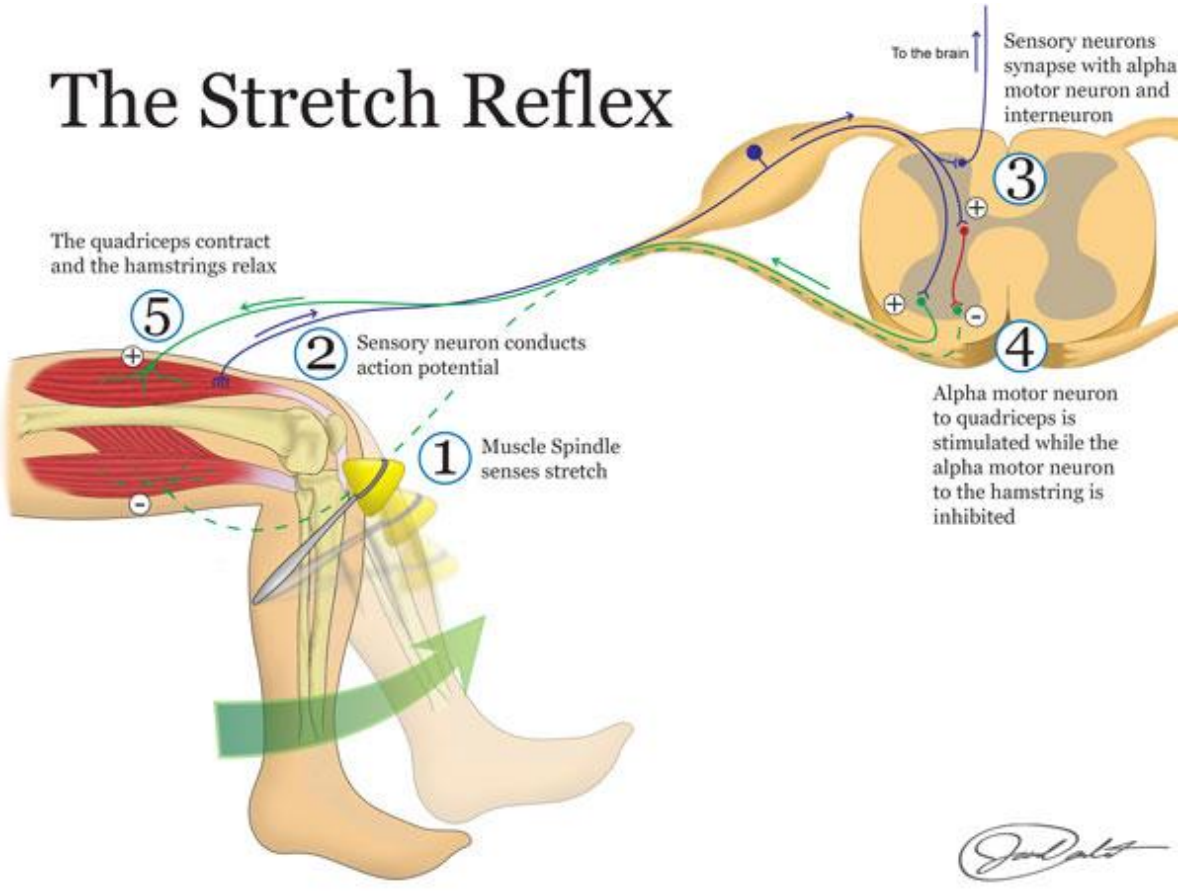

Image: Myotatic Reflex

Spindle tension serves as a reference for the muscle, ensuring that it is constantly measured and adjusted in its length through greater or lesser contraction. This occurs through minute forces of a few milligrams on intrafusal fibers to regulate forces of kilograms in extrafusal fibers. This mechanism is seen as a "servo-assisted" system.

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The tonic stretch reflex is characterized by the fact that even in a resting state, the spindle sends impulses through this basal tension activity, and the muscle at rest maintains a certain degree of tension, known as "muscle tone." This contraction of muscles against lengthening is particularly present in muscles opposing gravity. In postural muscles, the stretch reflex helps maintain postural balance, counteracting body oscillations, and is precisely referred to as the tonic myotatic reflex.

Shortening of neuromuscular spindles occurs in both active shortening situations, such as during voluntary action, and passive shortening situations, such as when muscles involuntarily shorten due to gravity, external position, or postural maintenance.

DYSFUNCTIONAL PATHOPHYSIOLOGY OF NEUROMUSCULAR SPINDLES:

In general, it must be clear how neuromuscular spindles and muscle reflexes are involved in regulating posture and muscle movement, and how dysfunctions in this system can lead to muscle and postural problems.

In the case of hyper-shortening, especially if done passively, neuromuscular spindles can remain silent. The central nervous system, no longer perceiving afferent impulses from the spindle, increases its activity towards the "Gamma" motor neuron, causing the hyper-shortened spindle to stretch (to perceive impulses from the spindle again). However, this creates a primary mismatch between the central nervous system's perception of the muscle position and the actual muscle position. This can lead to involuntary muscle contractions when the muscle is stretched again, especially if the stretching occurs rapidly or involuntarily. This can cause an overafferential discharge from the spindle, previously pre-excited by "Gamma" tension. This excessive overreaction leads to intense muscle contraction, restricting or limiting joint movement. This situation can result in persistent and painful postural problems and muscle tension, especially during muscle recruitment. Errors in the central nervous system's perception of body position can contribute not only to muscle but also to postural issues.

Relationship with other muscles: These dysfunctions can occur following trauma or abrupt movements, affecting muscles and causing tension in various parts of the body. For example, if the biceps brachii muscle experiences a severe dysfunction, it may become much shorter than normal, creating tension in surrounding muscles such as the brachialis, the pectoralis major, and the anterior deltoid. This can also lead to weakness in the triceps brachii and other antagonistic muscles. These dysfunctions can affect the opposite limb too, inhibiting muscle tone and limiting movements. Over time, such dysfunctions can cause pain, especially in the inhibited muscles of the opposing limbs. These pains are often perceived as muscle fatigue and weakness, but they are actually the result of neurological inhibition.

Such abnormal tensions cannot be easily resolved spontaneously because the system perceives a false tension message and often responds to it in an exaggerated manner. The body tries to cope by compensating for these dysfunctions through abnormal muscle tensions, which can lead to changes in posture and mobility. Tensions caused by these neurological errors can persist throughout life and be greatly limiting if not addressed appropriately, posing challenges for resolution.

So the key element is that: *"The greater the discharge from the pre-excited spindle, due to a higher initial Gamma activity, and the more forceful the muscle's contraction and the greater its resistance to being lengthened"*. The greater the displacement and misalignment of the joint heads pulled by those tense muscles; and the greater the joint's resistance to being moved in the opposite direction to the traction. *"During high Gamma activity, the spindle may, in effect, be calling for contraction when the muscle is already shorter than its resting length."* (Irvin M. Korr). Attempting to bring the joint

back to the correct position by shortening the hyper-elongated muscle (antagonist) will be difficult and painful. Instead, moving towards greater postural deformation, i.e., towards the shorter side (agonist), will be more manageable.

There are then several possible causes of muscle pain, which can result from sudden traumas or a gradual breakdown of the system, or as seen above, from neuromuscular dysfunctions that lead to a perception of pain, also given the body's attempt to compensate for these neurological errors through abnormal muscle tensions and postural changes.

The clinician can assess the neurological error inherent in the myofascial system, as it leads to the palpable objective observation of at least one of the following aspects:

- Decreased mobility
- Rapid increase in joint resistance in one or more planes of movement
- Muscle tension
- Pain during stretching within physiological ranges

The study of neural reflex mechanisms is at the core of the clinical significance of osteopathic dysfunctions. Erroneously, many tend to think that the problem often originates within the joint itself. However, in light of what has been explained above, it is more reasonable, as stated by Prof. Irvin Korr, to understand that *"the problem arises within the various muscles that cross and move a joint rather than always and only within the joint itself."* Korr's pioneering experiments and hypotheses were validated only 30 years later in 2006 through a clinical study conducted by the American Osteopathic Association (AOA) and published in the Journal of the American Osteopathic Association (JAOA), giving scientific value to Korr's postulates.

These abnormal receptor tensions in the peripheral system can over time convey anomalous information through somato-visceral reflex arcs, affecting deeper structures such as viscera and organs, blood vessels and lymphatics, and connective tissue through myofascial tensions. Thus, over time, genuine dysfunctions and pathologies of other systems beyond the musculoskeletal one can develop. Several studies on this topic were conducted as early as the 1940s by various researchers (Denslow, DO Burns, DO). It is also true that viscerogenic issues reflect through viscero-somatic reflex arcs on the muscle spindles, causing over-reactivity and abnormal tension, forming what is referred to as a "nocifensive mechano-tensive" mechanism.

The most effective way to reprogram these neurological errors, which are much more common than we might think, is to use a technique that passively returns the spindle to a discharge silence situation, preventing the Gamma motor neuron from exerting excessive traction or stretching on the spindle ends. After an initial attempt to recruit the hypercontracted spindle, the central nervous system will gradually decrease its discharge towards "Gamma." At this point, a slow and gentle return to the neutral direction will allow the correct restoration of the physiological length of the spindle and its afferent discharge. With this, muscle tension will also decrease, resulting in a reduction in pain and contraction. To date, the most suitable technique for peripheral receptor reprogramming and neuro-modulation is undoubtedly Counterstrain. In its execution, Counterstrain precisely mirrors the exact process of extinguishing and reprogramming the neuro-dysfunctional process.