

SCIENTIFIC

PHYSICAL THERAPY

Human Resting Muscle Tone - early concepts

By

Sam Betts, PT, MOMT, FAAOMPT

Introduction

Tone is defined as the resistance of muscles to passive elongation or stretch². Muscular tone is defined as the state of slight contraction usually present in muscles that contributes to posture and coordination; the ability of a muscle to resist a force for a considerable period without change in length².

The existence of resting muscle tone has received considerable attention over the years. In the early 1920's Sir Charles Scott Sherrington provided decorticated and spinal-transected experimental models to explain reflex neurogenic mechanisms of muscle tone. His models interrupted inhibitory central pathways of muscle contraction and emphasized neurogenic mechanisms as the cause of all muscle tonus³. Since that point, the contribution of skeletal passive human resting muscle tonus has been largely over-shadowed by neurogenic mechanisms causing resistance to passive stretch, despite the body of experimental research documenting the existence of resting muscle tension. Since then most text books consider muscle tone as entirely reflex in origin, maintained as a myotatic reflex in the muscle spindles.

One of the main objections to the generally accepted concept that resting muscle tone is reflexively mediated is the fact that there is no EMG activity even at very high amplitudes in a relaxed non-contracting muscle^{3, 4}. Since relaxed muscles do not exhibit EMG activity it seems likely that there exists basic resting tone as well as neurologically mediated muscle tone³. Basmajian stated that this results "from the natural elasticity of muscular and fibrous tissue⁴. Also the concept is further supported by the fact that resting muscles are not flaccid. Furthermore lower lumbar muscles have been found to be EMG silent in equilibrium postures (at ease) with no measurable action potentials. However, neuromotor activation occurs during movements, perturbations, and work. Human resting muscle tone (HRMT) may prove to influence the biomechanics of equilibrium postures and provide some form of mechanical and active control over imposed loading stresses during motion also. There is minimal qualitative research in the

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literature regarding passive/resting muscle tone, and even less in the way of quantitative studies describing passive muscle tension variations in normal subjects or patients with various back disorders, despite anecdotal communications amongst physical therapists and physicians.

Early theoretical concepts have proposed continuous tissue tension as a significant contributing factor assisting man to assume an upright bipedal posture against gravity⁵. Pre-tensing of tissue has been described as playing a significant role in the integrity of living organisms (biotensegrity), providing energetic and metabolic efficiency for many bodily functions, including maintenance of equilibrium and upright postures in man, and man's adaptations to bipedal gait⁵. This tissue tension referred by Levin as "tensegrity" has been hypothesized as an omnidirectional, and gravity independent passive myofascial support system, generating and dissipating forces, such that "it may actually strengthen a structure much as pre-stressed concrete or a wire under tension⁵."

The emerging field of "mechanobiology" amply indicates signal transduction pathways between tensions on external cell membranes and microtubular and microfibrillar connections to the nucleus that can induce cellular responses. Thus, every cell is theoretically responsive to mechanical stimulation in the body, particularly if it is applied locally or regionally and transmitted by interstitial tissues.

Background

Indirect clinical and biomechanical relations created interest for this proposed hypothesis; that muscle tone may be a passive resting state, independent of the neurological system. The underlying assumption is that human bipedal gait in our gravity field requires intrinsic postural mechanisms of tensile support, i.e., pre-tension, analogous to biotensegrity¹. Efficient biomechanical and energy design may favor an intrinsic elastic stability (mechanical) contribution in equilibrium postures, combined with well known existing muscular contractile neurological mechanisms.

The early concept discussed here proposes that postural and movement controls may be a combination of neuromotor activation in addition to tissue tensioning due to resting muscle tone. It proposes that human resting muscle tone is innate, may be a polymorphic/variable trait across the age and sex distributions of human populations, and may be measured and quantified. Precise, central neuromotor control of movements and of non-equilibrium postures are well documented, and not a focus of this new concept.

Response of muscle to passive stretch

It has been long known that the rise in tension seen during slow stretch of skeletal muscle cannot be explained as a purely elastic behavior. The tension rise in muscle that is passively stretched is biphasic, with a steep rise in tension, followed by a subsequent decreased resistance, sometimes referred to as thixotropy. The magnitude of the discontinuity is dependent on whether or not a stretch had been given immediately preceding. The initial rise has been interpreted as being due to the presence of long term stable cross-bridges in resting muscle fibers⁶. However, this initial theory has been the source of recent controversy, due to the fact that initial stiffness reaches its peak value at muscle lengths beyond optimum for myofilament overlap in active contraction⁶. Other explanations such as "viscous resistance" to interfilament sliding, and mechanical properties of elastic elements (the gap filaments such as connectin) have all been proposed as mechanisms for tension changes observed when passive muscle is stretched.⁷

Short range elastic component (SREC:)

In response to slow stretch, resting skeletal muscle does not behave like a purely elastic structure. Liddell and Sherrington⁸ described the response to stretch of the denervated vastocruureus muscle of the decerebrate cat, and noted "a slightly steeper upgrade of tensile resistance at the commencement of the stretch". Following that study Denny-Brown in 1929 also studied the decerebrate cat noted similar findings when stretching denervated semitendinosus muscle. He also noted that initial stiffness was smaller in response to the second of two stretches, given 3 seconds apart.⁹

In 1968 D.K Hill performed initial quantitative research studying relations of force-velocity and force (tension)-length⁷. He developed a theory to explain the interaction between sliding (actomyosin) filaments. He noted that relaxed muscle was disproportionately stiff for very small movements, with an increased elastic force response occurring for only a very small length (0.2%) change⁷. Since this stiffness persisted for only a small length change he called it the short range elastic component (SREC.) Hill suggested that the elastic properties of muscle during this SREC were due to the mechanical stiffness of a small number of cross-bridges between actin and myosin in sarcomeres of resting muscles⁷. This study by Hill created some confusion as this rise in tension was accompanied by an initial stiffness phase reaching a peak value at muscle lengths beyond the optimum for active contractile myofilament overlap. As a side note, Hill also noted that elastic modulus was found to increase with temperature in the range of 6-24 degrees Celsius. The cellular mechanisms for this change with temperature are unknown, although an altered molecular interaction or catalyst influence could be involved, as possibly calcium flux.

Hufschmidt and Scwaller¹⁰ recorded tension responses of relaxed human muscle in vivo during the application of triangular stretch cycles at a range of constant angular velocities. These stretches were given at forces below the threshold for inducing stretch reflexes. They demonstrated a similar initial steep rise in tension that had been recorded by Hill (Hill) The tension recorded then leveled off forming a shoulder¹⁰. This phase was the equivalent of the “short range elastic component” or “SREC” observed by Hill⁷ when stretching resting frog muscle. They found that in live relaxed muscles the maximum tension sustained by the SREC depended on the preceding ‘history’ of the muscle, i.e., its thixotropic property. The mechanical events during the 10-20 s before the stretch influenced the initial steep rise in tension. When the muscle was stretched and released immediately, the SREC was barely visible during repeat testing. They found resting tension to be influenced by passive movements of very small amplitudes. This lead them to conclude that SREC and resting tension may be caused by different sources due to the history dependent behavior of muscle i.e., its thixotropic property (see below.)

Lannergren¹¹ also showed that the SREC did persist for about 0.2 % of the length change, becoming larger in hypertonic solution and smaller when measured a few seconds after a twitch, taking 3 minutes to recover fully¹¹. Lannergren then argued that if both the SREC and active

tension changes were both due to cross-bridges, then stiffness was expected to increase, not decrease during low level activation of the muscle. However the effect of the hypotonic solution on muscle mechanics was not accounted for during the experiment.

Parallel Components

Close¹² proposed that the resting force between filaments was directly related to the amount of overlap of myofilaments, and inversely proportional to the lateral spacing of elements within the sarcomere. This indicated that elements in parallel such as connective tissue may create internal forces within the sarcomere if they exerted enough force to bring sarcomere components closer to each other.

Schleip et al introduced the hypothesis that intramuscular connective tissue, in particular the fascial layer known as perimysium may be capable of active contraction and consequently influence muscle stiffness¹³. Morphological and histological investigations demonstrated a high level of myofibroblasts, showing that in vitro contraction tests with fascia is able to actively contract and may be enough force to influence musculoskeletal dynamics They argue that the perimysium seems capable of response to mechanostimulation with a myofibroblast facilitated active tissue contraction, thereby adapting passive muscle tension to increased tensional demands. Due to the higher percentage of perimysium in tonic musculature, they proposed that tonic musculature has greater stiffness¹³.

Effect of Calcium

Moss et al¹⁴ studied tension responses of mechanically and chemically skinned frog muscle fibers. They found that the muscle fibers of the frogs did not exhibit SREC in normal relaxing solutions of low calcium concentrations. However, raising calcium concentrations slightly to levels just below those required for activation, re-established the initial stiffness responses to stretch. The lack of SREC generation in solutions with low calcium was taken as evidence that calcium release from sarcoplasmic reticulum was not involved in generating the SREC.

Another point of contention with a cross-bridge origin of SREC is that there is an increase SREC in a region of reduced optimal myofilament overlap, followed by a fall at very long lengths.^{7, 15} It has been shown for some time that the sensitivity of myofilaments to calcium changes with muscle length.¹⁵ However, it remains unclear how closely the length dependency

of SREC parallels that of changes in calcium sensitivity.

Moss et al¹⁶ studied resting frog skeletal muscle, showing that when stretched at a constant velocity muscle demonstrated a force response which, for length changes less than about 0.2%, was found to be elastic in nature. They proposed that the short range stiffness of muscle depended on the concentration of calcium in the fluid bathing the myofibrils and that short range stiffness was a property of calcium activated cross-bridges. They concluded that these calcium activated cross-bridges have either become attached to the thin filament sites, or at least changed, from a resting position to an activated position.

Thixotropy

Thixotropy, from the Greek *thixis* and *-tropy*, means “transformation by touch¹⁷.” A good example of this is stirring honey with a spoon in a honey pot. Initially it is hard to move the spoon, but with continued movement there is less resistance to the movement of the spoon, and it gets easier to stir the honey. Buchthal and Kaiser¹⁸ initially introduced the term thixotropy in connection with skeletal muscle physiology. They found that muscle fiber stiffness (or resistance to stretch) was temporarily reduced by one single stretch movement, with the stiffness slowly returning over a rest interval. In addition, other types of mechanical agitation such as that caused by electrical stimulation or voluntary muscle contraction were shown to cause thixotropic muscle responses. SREC response to passive stretch is shown to be history dependent, with prior motion creating a stretch point they describe as occurring at 1-2 % of resting length, versus 0.2% reported by Hill. The exact relation between experimentally demonstrated SREC (observed with stretches of 0.2 to 0.5% of resting length) and clinically demonstrated thixotropic decrease in muscle tension occurring after greater stretches (1-2% of resting length) is not clearly defined, but are suggested to be based upon similar mechanisms of actomyosin interaction.

Irrespective of underlying mechanisms, skeletal muscle thixotropy implies that muscle stiffness and passive resting tension of the muscles are critically dependent on immediately preceding movements and contractions¹⁷. Temperature has not been shown to influence the thixotropic effect (i.e., the rate of recovery of stiffness after movements¹⁷.) Also, a point of interest is that human resting muscle tone and thixotropy persist

during deep anesthesia further implicating non-neurogenic causes for passive resting muscle tension and thixotropic behaviors of muscle.

Role of the Giant Titin Molecule in Passive Muscle Tension and Elasticity

In the past few years it has been realized that a major contributor to the length/tension relation of muscle is provided by elastic filaments (gap filaments) spanning each half of the sarcomere. A high proportion of gap filaments are the giant macromolecule titin which is attached at one end to the Z-line and the other end to the thick filament. Titin is thought to play a major role in myofibril assembly, elasticity, and stability¹⁹. The titins (also known as connectins) are a family of giant proteins with polypeptides having large molecular masses. Titin is a main player in determining passive muscle mechanics, especially the physiology of cardiac muscle distension, as it develops passive tension in stretched non-activated muscle²⁰, shows high passive elastic recoil speed and helps to smooth out muscle contractions by contributing to the muscle's viscous/viscoelastic properties. It is not clear as to what degree titin is responsible for overall passive skeletal muscle tension levels, and even less clear the role they play (if any) in early phases of passive tension applied to skeletal muscle fibers.

DISCUSSION

The concepts discussed here prove that there is indeed resistance to passive stretch of resting skeletal muscle, during in-vitro experimentation. The emerging research provides new insights as to the origin of resting skeletal muscle tone, although the relative contributions and interactions of each of the sub-systems described in this article are unknown. As yet, passive muscle tone has not been shown to exist in humans although indirect clinical and biomechanical observations suggest its existence. Pain free and relaxed muscles “loosen up” following very light manual touch or massage, soften when exposed to warmth, and relax when warming up for exercise. These all represent examples of changes of human resting muscle tone due to thixotropy. What is it that creates variance between individuals during palpation of muscle tone and firmness? Are there mechanisms or factors unifying palpation variances between individuals? If there are polymorphic/variable traits across the age and sex distributions of human populations then finding this unifying factor or factors may have clinical implications for pathology and disease states.

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A Critical Review of Dr. Timothy Flynn's Guest Editorial In The April 2006 Issue of JOSPT

Dear reader;

In September of 2006 the following letter was forwarded to Guy Simoneau, PT, Ph.D, ATC, Editor-in-Chief, JOSPT:

Letter to the Editor Journal of Orthopaedic and Sports Physical Therapy

Dear Dr. Simoneau,

In his article entitled, "There's More Than One Way to Manipulate the Spine" (guest editorial in the April issue of JOSPT) Flynn states that "...an increased emphasis should be placed on evidence-based decision making and the development of hands-on skills that can be performed proficiently....". So, the question is: Has Flynn made the case for less precision in choosing spinal manipulation techniques based on evidence? To help us in answering this question, we can keep in mind Sackett's definition of evidence-based medicine: "...the integration of best research evidence with clinical expertise and patient values.", (quoted in the guest editorial of the June, 2006 issue of JOSPT).

Referencing Jull and Moore, Flynn says: "For example, decision making related to manual therapy interventions have often been based on theoretical biomechanical constructs." He goes on to imply that Jull and Moore address the identification of a segmental dysfunction based on intersegmental motion or alignment, identifying a very specific impairment that requires the application of a particular manual therapy procedure, a procedure that is reinforced by manual therapy educators who emphasize the negative consequences of failure to choose the "right" technique.

The Jull and Moore reference is actually an editorial, not a research report, and thus is not evidence. Further, the focus of the editorial was:

- Multidimensional approaches to assessing spinal pain;
- Multidimensional sources of spinal pain; and
- Comprehensive assessment is needed in order to identify the specific source(s) of pain in each individual patient.

Flynn's reference to Jull and Moore pertains to a single sentence in their editorial and not its primary focus.

Referencing Kulig, et al, Flynn says: "Kulig et al demonstrated that when a PA grade IV force was directed at any segment in the lumbar spine, rotation in the sagittal plane occurs at all lumbar vertebrae."

A review of the data presented in the Kulig, et al article reveals that:

- Rotation occurs PRIMARILY at the point of force;

Flynn's reference to Kulig, et al is over-simplified, not taking into account the actual characteristics of the specific data generated by their study. Further, Flynn fails to state that force without stabilization will always produce rotation.

Referencing Lee, et al, Flynn says: "Similarly Lee and colleagues reported that Grade III mobilization directed at the C 5 spinous process was not specific to the targeted segment; in fact, the greatest amount of rotation in the sagittal plane occurred at two to three segments above and below the targeted vertebra."

A review of the data presented in the Lee, et al article reveals that:

- their findings directly contradict those of Kulig, et al;
- the standard deviations are so large as to render the mean values irrelevant.

Flynn fails to point out these contradictions and anomalies and thus fails to take into account the fact that either or both data sets may be invalid.

Referencing Beffa and Matthews, Flynn says: "Beffa and Matthews found no correlation between a technique directed at L 5 and one directed at the sacroiliac joint. In fact, each of the techniques resulted in cavitations throughout the lumbosacral region."

A review of not only the data but also the imitations, and conclusions presented by Beffa and Matthews reveals enough serious flaws in their measurement approach (trying to correlate cavitation sounds with the focal points of manipulation) to invalidate their findings. Flynn fails to acknowledge the authors' depreciation of their own work.

Referencing Ross, et al, Flynn says: "Ross, et al reported that manipulation directed at the thoracic spine was accurate only 53% of the time, while lumbar spine manipulation was accurate only 46% of the time."

The Ross, et al study suffers from the same methodological flaws as Beffa and Mathews because it relies on the theoretical but undocumented relationship between the apparent location of the cavitation and the point of manipulation.

Even Ross, et al are equivocal on their measurement technique: "When the SMT is delivered, it is typically accompanied by an audible cracking sound. Although it has not been confirmed conclusively, this sound has been associated with the cavitation of spinal zygapophysical joints."

A review of the Ross, et al scatter diagram provides graphic evidence that any apparent relationship is coincidental.

Ironically, even Flynn acknowledges the absence of this relationship when he says: "Previously we have reported that the presence or absence of an audible popping sound during the manipulation is not related to outcomes in patients with low back complaints,"

Referencing Haas, et al, Flynn says: "Haas and colleagues reported on the response of patients with neck pain who received thrust manipulation to either a randomly assigned level or a level selected based on end-play assessment. The authors noted that both groups achieved equally significant reduction in neck pain and stiffness, with no apparent advantage in attempting to target the intervention to specifically identified 'impaired' segments."

A review of the Haas, et al article reveals that:

- it is a synopsis, not a research report, and, further, it is accompanied by a commentary that is highly cautionary in its focus! Clearly the Haas, et al article is not evidence;
- Flynn ignores the authors' caveats in addition to the caveats of the commentator, who says: "Importantly, the authors point out that this study does not evaluate long-term differences in clinical outcomes. It remains possible that segmental specificity is more important for some of the potential long term effects of manual treatment. Because of the lack of longer-term studies, and the relative paucity of research looking at joint-specific effects it would be prudent for clinicians to apply joint manipulation with a good deal of specificity. It's important to be aware that the effect(s) may not be as specific as we think."

Referencing the Cleland et al, Flynn says: “Cleland and colleagues provide further insight on the subject of specific technique selection. The authors report on a series of patients with low back pain who met criteria suggesting they would respond favorably to a particular thrust manipulation technique. But instead of using the technique that had previously been shown to be effective, the authors chose to perform an alternative technique that was **potentially different** [emphasis mine] in terms of the spinal region treated and the expected patient response. The results of this case series indicate that **perhaps** [emphasis mine] the particular technique selected is not nearly as important as determining the appropriate subgroup of patients in which spinal thrust manipulation is most likely to be beneficial.”

A review of the Cleland et al article reveals that:

- The bolded words indicate that Flynn is being very cautious in his claims here;
- The CPR criteria are very general, and may not have differentiated between subjects to the extent necessary to warrant a more specific treatment approach;
- With two exceptions, all pre-treatment scores on the ODI were below 50%, indicating mild to moderate disability to begin with;
- No record was provided on the actual number of manipulations each patient received.

Conclusion: Flynn fails to make the case for less precision in choosing spinal manipulation techniques based on evidence:

- Articles are cited that are not evidence-based (i.e., Jull and Moore; Haas, et al)
- The focus of authors’ articles are misrepresented (i.e., Jull and Moore)
- Contradictory findings between cited authors are ignored (i., Kulig, et al and Lee, et al)
- Inclusive data are ignored (Ross, et al)
- Authors own acknowledgements about the limitations of their studies are ignored, along with limitations cited by commentators (i.e., Haas, et al)
- Cites findings of studies, the measurement techniques of which Flynn himself has discounted (i.e., Beffa and Matthews; Ross, et al)
- Fails to account for alternative explanations (i.e, Cleland, et al)

Flynn is the current president of the AAOMPT. As such he is representing the highest authority in the field of Orthopedic Manual Therapy in the United States. His editorial interpretation of research and clinical facts are misleading and unreliable. It is an attempt to underplay the importance of clinical skill and specificity in the field of manipulative therapy. Numerous references he is not quoting confirms that pain inhibition may occur as a result of non-segmental and unspecific manipulation of the spine. However, diagnostic procedures, protection of contra indicated influence from manipulation, as well as biomechanical , segmental dysfunction require a highly specific approach as to segment, direction, and force.

In misrepresenting the need for specificity in the field of spinal manipulation, Flynn is endangering the patients and the future of this intervention among physical therapists. He is well advised to add significant clinical experience and education to his interpretation of scientific material.

Respectfully Submitted,

Ola Grimsby,
Chair of the Board,
The Ola Grimsby Institute

Eddy Miller, Ph. D.
Director of Research
The Ola Grimsby Institute



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October 8th we received a letter from the editor, correcting our letter as to “not dated” that we referred to Dr. Flynn’s guest editorial as “an article,” that he (Dr. Simoneau) did not believe that our interpretation of the guest editorial was correct, and the format of our letter was not very conducive for publication.

Dr. Miller and myself responded with the following comment in a letter dated November 2nd .:

Dear Dr. Simoneau:

We are in receipt of your October 8 letter in which you review the reasons for not accepting our previous letter to you regarding Dr. Flynn’s guest editorial. Needless to say, we are both disappointed, not only in your unwillingness publish our letter, but in your seeming disregard for the important points we raised in it.

The fact that our letter may have been undated, that it may have arrived later than you wished, that its length is not conducive to publication, and that the focus of our concern was a ‘‘Guest editorial’’ and not an article, are all peripheral issues; they have nothing to do with the substantive challenges we raised regarding Dr. Flynn’s hypocritical position on evidence-based practice, as demonstrated by his consistent misrepresentation of the references he cited.

In order to insure that Dr. Flynn’s abuses of the literature do not go unchallenged, we look forward to your suggestions on how his lack of scholarship might be exposed. Since Dr. Flynn’s presentation is in the form of a ‘‘Guest editorial’’ please invite us to submit a ‘‘Guest rebuttal’’, based on our letter to you.

We look forward expectantly to your encouraging reply, knowing that you are committed, as we are, to building the knowledge supporting clinical practice.

Eddy Miller

We have not yet received a response from the Editor-in Chief. By rejecting our letter concerning Dr. Flynn’s abuses of the literature concerning specificity of manipulation, Dr. Simoneau has chosen to share responsibilities with his Associate Editor in endangering the patients treated with manipulation. Due to the severity of this attitude, we find it necessary to distribute our correspondence concerning this matter to clinicians offering manipulative intervention.

Respectfully

Ola Grimsby,
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