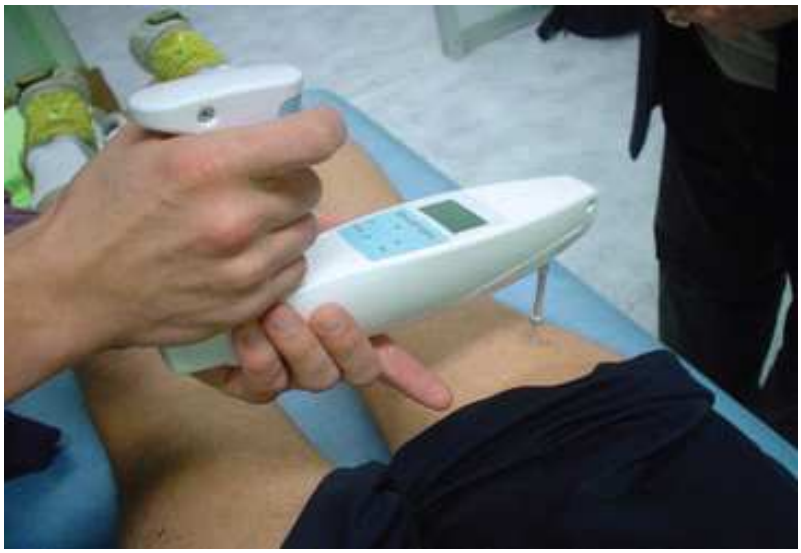
Kan massasje endre musklenes hviletonus? En pilotstudie utført på idrettsutøvere med en objektiv målemetode

Vedlegg (litteratursøk): Resting muscle tone: definition, measurement and physiological mechanisms



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Bakgrunn

Denne oppgaven består av en artikkel basert på et klinisk forsøk i relasjon til skjellettmuskulaturs hviletonus. Artikkelens tittel er:

"Kan massasje endre musklenes hviletonus? En pilotstudie utført på idrettsutøvere med en objektiv målemetode".

I tillegg presenterer vi et vedlegg - et litteratursøk med tittelen:

"Resting muscle tone: definition, measurement and physiological mechanisms".

Interessen for fagområdet kommer dels fra en idrettbakgrunn og dels som følge av fokus på muskel og skjellettsykdommer i den medisinske klinikk. Blant idrettsutøver kan muskeltonus være en subjektiv indikator for prestasjon og/eller treningsbelastning/overbelastning. I den generelle medisinske klinikken er muskeltonus et nøkkelbegrep innen livstils/jobbrelaterte sykdommer i bevegelsesapparatet.

I motsetning til forskning på muskeltonus basert på kontraktil aktivitet utløst fra nervesystemet så vi fort at det var gjort relativt lite forskning på muskeltonus basert på forhold i selve muskelen (hviletonus).

Hovedmålsettinger med denne studentoppgaven er derfor:

1. Måle hviletonus på en objektivmåte og se om den er påvirkbar av mekanisk stimulus

2. Presentere ulike mulige forklaringsmekanismer basert på et litteratursøk i PubMed.

Vi understreker at det er vår pilotstudie (artikkelen) som har vært hovedfokus i denne studentoppgaven. Vedlegget en ment som et supplement og for å bidra til å belyse mulig virkningsmekanismer og videre diskusjon.

Vi vil gjerne rette en stor takk til Per Brodal ved Avdeling for anatomi ved Medisinske Fakultet UIO for svært god og tett veiledning.

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"Kan massasje endre musklenes hviletonus?"

En pilotstudie utført på idrettsutøvere med en objektiv målemetode

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Sammendrag

Bakgrunn : Flere studier indikerer at økt muskeltonus kan gi vedvarende plager. Massasje er en vanlig brukt metode for behandling av dette. Effekt av massasje på muskeltonus er likevel ikke dokumentert med objektiv metodikk. Vi ønsker i denne pilot studien å undersøke om tverrmassasje kan endre hviletonus.

Materiale og metode : Fem mannlige idrettsutøvere på høyt nasjonalt nivå mellom 20 og 30 år ble inkludert i studien. Studien varte i 4 uker med målinger 1 dag i uken. Hviletonus ble målt i rectus femoris med et håndholdt myotonometer før og etter massasje. EMG ble brukt for å utelukke elektrisk aktivitet. Deltakerne ble massert på tvers av muskefiberretningning i 20 min.

Resultater : Av totalt 20 målinger viste 16 reduksjon i hviletonus etter massasje. Med utgangspunkt i en nullhypotese om tilfeldig variasjon gir 16 målinger med redusert tonus av 20 totalt en p-verdi på < 0,001. Gjennomsnittlig nedgang var på 3,3 % . Største nedgang var på 11, 5 %.

Fortolkning : Resultatene tyder på at en mekanisk stimulus, tverrmassasje, fører til reduksjon i hviletonus etter 20 minutters massasje. Størrelsen på studien er imidlertid for liten til å vise signifikant utslag på individnivå. Det bør gjøres flere studier for å undersøke om endringen som denne studien finner er stor nok til å kunne ha klinisk betydning. Det bør også undersøkes nærmere om det er en sammenheng mellom endring i hviletonus og langvarige muskelsmerter og i så fall hvor store endring i hviletonus det må være for å kunne gi smertesymptomer.

Introduksjon

Flere studier indikerer at økt muskeltonus kan gi vedvarende plager; enten som overtrening hos idrettsutøvere eller kroniske muskelsmerter i befolkningen generelt (1, 2)

Massasje er brukt som behandling av muskelplager og av idrettsutøvere for å oppnå raskere restitusjon. Vi oppfatter at det hersker en tro på dette som behandlingsmulighet i mange miljøer.

Et søk på PubMed frembrakte derimot ingen studier som ved bruk av objektivt pålitelige målemetoder undersøkte effekt av massasje på muskeltonus. I senere tid er det imidlertid blitt mulig å måle muskeltonus objektivt ved hjelp at et myotonometer.

Målinger med myotonometer gir data som har god korrelasjon med EMG, intracellulært trykk og standard klinisk undersøkelse (2, 9, 10). Tidligere målemetoder har ikke gitt konsistente resultater, og har derfor vært mye kritisert (11).

Hensikten med denne studien er derfor å benytte seg av denne objektive målemetoden for å se om en mekanisk stimulus, slik som tverrmassasje, faktisk kan redusere en muskels spenningstilstand i hvile – dvs. muskelens hviletonus. Svar på dette spørsmålet vil kunne bidra til bedre forståelse av hviletonus i

skjellettmuskulatur, og vil kanskje kunne bidra til å bedre behandlingen av tilstander med patologisk forhøyet muskeltonus.

Tonus (eller spenningen) i en muskel kan enten skyldes kontraksjonsaktivitet utløst fra nervesystemet (nevrogent utløst) eller forhold i muskelen selv. I en hvilende, godt avslappet muskel viser det seg at nevrogent utløst aktivitet slik den kan registreres med EMG er tilnærmet lik null (3, 4, 5, 6, 7, 8). Hviletonus i en godt avslappet muskel må derfor skyldes forhold i muskelen selv. Hvis hviletonus endres ved massasje, må det derfor forventes at dette har virket inn på muskelens indre egenskaper.

Materiale og metode

Fem mannlige idrettsutøvere på høyt nasjonalt nivå mellom 20 og 30 år ble inkludert i studien. Studien er godkjent av regional komité for medisinsk og helsefaglig forskningsetikk og alle deltakerne ga informert skriftlig samtykke. Målingene ble utført hver tirsdag i en 4 ukers periode. Muskeltonus ble målt ved hjelp av et myotonometer rett før og rett etter 20 minutters massasje. For å utelukke nevrogen aktivitet ble det utført overflate-EMG registrering parallelt med tonusmålingene.

Myotonometeret som ble brukt var Myoton-2, utviklet ved Universitet i Tartu, Estland. Instrumentet er håndholdt og beregner tonus ved å registrere mekaniske oscillasjoner i vevet provosert ved en mekanisk impuls på ca 0,4 Newton i 15 millisekunder. Frekvensen (Hz) av oscillasjonene gir et mål på muskeltonus. Apparatet gir middelverdi av 20 målinger. Normalnivået for hviletonus hos friske mennesker ligger mellom 10 og 20 Hz (12).

EMG-registreringen ble foretatt ved hjelp av et bærbart apparat, Physiometer (PHY-400), software: versjon 3.02. Samplingraten var 1280 Hz. Root Mean Square (RMS) kalkuleres og overføres 40 ganger pr sekund til en datamaskin. Apparatet var satt opp med et såkalt EMG Band-Pass filter med ”cut off” frekvens på henholdsvis 20Hz og 800Hz. Amplifikasjonraten var10X ved lavinput, 1X ved stor input. Huden ved målepunktet ble spritvasket og det ble brukt engangselektroder med elektrodegel. Elektrodene ble ikke fjernet under massasjen for å hindre feilkilder ved bytting/gjenpåsetting av elektroden.

Samtlige målinger ble utført på rectus femoris med forsøkspersonen liggende i mest mulig avslappet stilling. Samme målepunkt ble brukt ved samtlige målinger. Målepunktet ble markert på huden og holdt ved like av deltaker til neste måletidspunkt.

Teknikken som ble benyttet var en såkalt tverrmassasje som innebærer at massasjen gjøres på tvers av muskelfiberretningen i skyvende og løftende bevegelser langs hele muskelbuken (13)

Resultater

EMG målingene utført samtidig med registreringen av muskeltonus viste typisk en elektrisk aktivitet i størrelsesorden +/- 10 mikrovolt. Så lave utslag er uttrykk for elektrisk bakgrunnstøy. Typisk vil en voluntær kontraksjon gi elektrisk aktivitet i størrelseorden 1500 til 2000 mikrovolt. Vi konkluderer derfor at muskeltonusen som ble målt stammer fra intramuskulære forhold. Resultatene var så konsistente at vi besluttet kun å registrere EMG under første målingsdag dvs. etter 10 EMG registreringer.

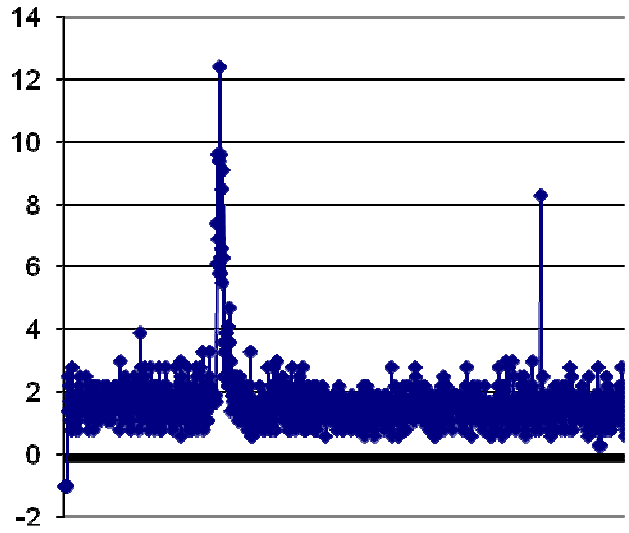

Diagram 1 Typisk EMG utslag i mikrovolt under muskeltonusmåling.

Av totalt 20 utførte målinger på de fem forsøkspersonene viser 16 nedgang i muskeltonus etter massasjen. 3 målinger viser økt tonus. Tre personer hadde nedgang i tonus i samtlige målinger. En deltaker hadde to målinger med økt tonus.

	Person 1		Person 2		Person 3		Person 4		Person 5	
	Før	Etter	Før	Etter	Før	Etter	Før	Etter	Før	Etter
Dag 1	17,3	16,7	13,9	15,1	16,7	15,7	14,7	13,6	12,1	11
Dag 2	16,1	15,5	13,3	14,2	16	15	13,6	13,2	10,2	10
Dag 3	17,3	17,2	14,2	13,6	16,5	14,6	14,1	14,1	13	12,5
Dag 4	14,4	14,8	13,6	12,7	15,2	14,3	14	13,5	12,3	11,5

Tabell 1 Hviletonus målt i Hz før og etter massasje

Gjennomsnittlig endring i tonus var 0,49 Hz. Dette tilsvarer en gjennomsnittlig reduksjon på 3,3%. Største enkeltvise nedgang var 11,5%. Med utgangspunkt i en nullhypotese om tilfeldig variasjon gir 16 målinger med redusert tonus av 20 totalt en p-verdi på $< 0{,}001$ hvilket skulle tilsi at massasje har signifikant effekt på hviletonus. Imidlertid kan det vurderes om det er riktigere å vurdere hver enkelt person for seg. Med kun 4 målinger pr deltaker i denne studien vil det ikke gitt signifikante verdier.

Diskusjon

Det er gjort svært lite forskning på om hviletonus kan påvirkes av massasje til tross for utbredt bruk Det er heller ingen av disse studiene som objektivt har forsøkt å måle effekten av massasje på hviletonus, men de har derimot vært basert på mer subjektive parametere som muskelpalpasjon (9)

Denne lille pilotstudien viser at en mekanisk stimulus, tverrmassasje, fører til en signifikant reduksjon i hviletonus etter 20 minutters massasje. Dette bekrefter at massasje har innvirkning på hviletonus målt ved objektive metoder. Studien er imidlertid for liten til å vise signifikant utslag på individnivå.

Et sentralt spørsmål som bør belyses nærmere er om de endringene vi ser i denne studien er store nok til å ha biologisk-funksjonell betydning. Dette er usikkert og vil kun kunne besvares ved et mer langvarig forsøk. Et interessant poeng er allikevel at siden rectus femoris ble tverrmassert/målt på kun

et bein var det mulig for massør og sammenligne dette beinet med det ikke-masserte beinet. Dette ble også gjort og massør kunne palpatorisk kjenne forskjell på massert muskulatur og ikke-massert muskulatur.

Som nevnt er dette en pilotstudie. Antall deltakere er lite og utgjør en homogen gruppe. Dessuten er tidsintervallet også kort. Det bør også gjøres studier som undersøker om effekten av massasje holder seg lenger enn det vi har vist i denne studien. Vi har bare undersøkt friske personer, og tilsvarende målinger bør gjøres på personer med langvarig forhøyet muskeltonus.

Hvilke endringer i intramuskulære forhold er det som ligger til grunn for våre funn? Intramuskulære årsaker til hviletonus i skjellettmuskulatur er studert fra ulike innfallsvinkler. I hovedsak er det lansert fire ulike hypoteser, som riktignok ikke utelukker hverandre: 1. Kryssbindinger mellom kontraktile proteiner som myosin/aktin selv i en muskel uten nevrogen aktivitet (14, 15, 16) 2. Egenskaper ved andre intramuskulære proteiner enn myosin og aktin, som titin og enkelte andre (17, 18, 19, 20, 21) 3. Intracellulært/ekstracellulært væsketrykk (2, 22, 23, 24) 4. Kontraktile egenskaper i muskelfascien (25, 26).

Alle disse hypotesene har eksperimentell støtte, og enkelte nyere studer (27, 28) taler for at hviletonus avhenger av en kombinasjon av flere faktorer. Sannsynligvis kan den enkelte faktors bidrag variere mellom muskler – for eksempel er muskler svært ulike med hensyn til hvor sterkt utviklet fascien er.

En bedre forståelse av muskeltonus og påvirkningsmuligheter vil kunne endre tilnærmingen til muskelplager både med tanke på årsaksforhold og mulige nye angrepspunkter for behandling.

Til slutt vil vi takke veileder professor Per Brodal for hjelp under arbeidet, Magnat Corpus Fysioterapi for massasjeassistanse, Harald Vikne og Eva Bakke ved STAMI for lån av EMG apparat og Institutt for Biologisk Statistikk ved UIO.

Oppgir ingen interessekonflikter.

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English abstract :

"Can massage change the resting muscle tone in skeletal muscles ? A pilot study on athletes with an objective measurement method."

Several studies indicate that an increase in muscle tone may lead to sustainable muscular pain. Massage therapy is a commonly used method to deal with increased muscle tone, though the effect of this has not been thoroughly examined with objective measurements. In this study we examine the effect of massage on skeletal muscle tone.

Five athletes were given 20 minutes cross friction massage once weekly over 4 weeks. Resting muscle tone was measured with a Myotonometer (Myoton 2) and EMG was used to exclude neurogenic activity. Results after the massage showed a decrease in muscular resting tone in 16 out of 20 measurements (0,001) The average deduction was 3,3 % and the largest reduction 11,5 % This indicates that massage therapy may be a useful treatment to decrease resting muscular tone but to find out if this effect is sustainable needs further studies.

Vedlegg : Litteraturgjennomgang

"Resting Muscle Tone : Definitions, Measurement og Physiological Mechanisms"

Introduction

Traditionally, muscle tone has been explained by a continuous stream of nerve impulses to the muscles. It is well known, however, that a muscle under complete relaxation shows no signs of being activated from the nervous system, as measured by EMG. In addition, no afferent signals arise in the muscle spindles of a completely relaxed muscle. Thus in the resting condition the tone of the muscle cannot be explained by nerve activity, although it is obvious that the muscle shows a certain resistance when being palpated.

Muscle tone at rest must therefore depend on other factors than contractile activity caused by nerve impulses. Less research has been done focusing on supplemental causes of muscle tone increase/decrease as for example the intramuscular causes. What are these, to what extent can they explain changes and what clinical implication does this have ?

In this paper, we will look at these different characteristics of muscle tone under rest and exploit evidence of variation of this tone among individuals. In addition to this we will discuss possible ways of measuring these variations by non subjective measurement methods. Lastly we will discuss these factors and how they may be manipulated/altered from a clinical perspective.

Our motivation for writing this is partly based on experience with sports where muscle tone may have an important role in performance and recovery. Secondly, we are interested in altered muscle tone in a clinical context, as a source of pain and disability.

Defining resting muscle tone

In the literature, the definition of muscle tone has changed over time. It is further complicated by a variety of definitions explaining the same kind of phenomena.

For example, to illustrate the confusion, the definition indexed in Medline for muscle tone is solely based on tone evoked by efferent signals from the nervous system. However, most of the indexed results on the search "muscle tone" also include intrinsic properties of the muscle in addition to the efferent nervous signals.

In our article we will use the following definition of muscle tone in a relaxed state :

"The intrinsic viscous/elastic properties of the muscle in the absence of contractile activity"

Our definition is based on the overview article "Understanding and measurement of muscle tone as related to clinical muscle pain" (Simons and Mensa , 1998) where the authors make a few important distinctions and clarifications when defining muscle tone.

In the article by Simons and Mensa the authors conclude that muscle tension depends physiologically on two factors "the basic viscoelastic properties of the soft tissues associated with the muscle and/or the degree of activation of the contractile apparatus of the muscle".

This definition is similar to that of Almaki et.al. 2007 : "Muscle tone (stiffness, compliance, hardness) is defined as the interaction between muscle viscoelastic properties, structures and neural regulations"

Resting muscle tone is then by Simons and Mense defined as "the elastic and/or the viscoelastic stiffness in the absence of contractile activity"

Measurement of muscle tone

As stated in the introduction, we want to explore the possibilities of measuring muscle tone in an objective way. This possibility is essential to answers whether or not muscle tone varies from individual to individual and over time.

In clinical practice, the most common ways of measuring muscle tone is either by passive joint movements, as in the case of determining spasticity or rigidity, or by pressing the fingers manually/palpation against the muscle to register the force needed to displace it.

These kinds of measurements have obvious problems. They are highly subjective, depend on the experience of the therapist, and results from different individuals are difficult to compare. In addition, in the case of passive movements, the contribution of altered joint mobility due to changes of the joint itself cannot be excluded.

Since the 1980s, equipment has been under development to objectively evaluate muscle tone based on application of force/pressure to the muscle. Thereby independent of the dynamic testing activity/joint mobility.

In 1987 Fischer developed a device that calculated the relations between applied pressure and depth of penetration to the muscle. A number of other similar devices have since been developed.

This includes a so called hardness-meter by Horikawa (2001) and Bendtsen et.al. (1994). This method has been further developed by Leonard in the recent years and their device Myotonometer that has

shown very high correlation with EMG results and in comparison with resistance of passive stretch using the Ashworth scale (Leonard 2003,2004)

One of the main critiques of these devices is the fact that the results are influenced by a combination of skin, subcutaneous fat/tissue and the muscle compliance itself. The muscle thickness of the testing person also influences the results and must be taken into consideration of these type of measuring devices (Ylinen et.al 2006) These problems are less for the newer equipment, however (Leonard, 2004)

Another, slightly different approach to measuring muscle tone is based on damped oscillations by a mechanical stimuli. This can also be done manually, as described by Comeux (20003)

The principle for this kind of measurement technique is to have control over a given force applied to the muscle and next analyze the functional feedback from the muscle.

A strength of the devices based on the damped oscillations is that the influence of skin and subcutaneous fat/tissue may be less. In fact, a study by Gapeyeva in 2000 showed that for girls between age 15 to 17, subcutaneous tissue up to 12 mm did not interfere with the measurements.

One of these devices, the Myoton 2, has shown high correlation between muscle tone, EMG results and intramuscular pressure (Korhonen et.al 2005). It may therefore give a more objective view of muscle tone as compared to earlier instruments.

In the case of the Myoton 2, it is also possible to differentiate the different characteristics of the passive muscle. More precisely elasticity, tone and stiffness as separate entities (Gavronski 2007)

Elasticity is defined and measured as “the ability of a body to recover its shape” and is logarithmically calculated. It describes the loss of mechanical energy as the amplitude of the oscillations decline.

Stiffness is measured as the resistance tissue body offers to force applied to it.

Muscular tone is in this case functionally described as the frequency of these oscillations in Hz. (Korhonen, 2005)

When measuring different populations of people, these 3 characteristics vary. For example, when measured 3000 people in Estonia : older people shows increased tone, but at the same time lower elasticity. The same goes for people with higher BMI (Vain,2007)

Therefore, differentiating muscle tone, stiffness and elasticity may be interesting even though the characteristics to a large extent is overlapping.

The newer equipment based on oscillations, such as the Myoton 2, thus seems to be able to measure the characteristics of the skeletal muscle including muscle tone with accuracy. This fact is an important and necessary contribution for determining changes in muscle tone/clinical studies on how muscle tone may be manipulated.

Evidence for tone in resting skeletal muscle

As explained initially, muscle tension in resting skeletal muscle would depend on the viscoelastic properties of the muscle itself. (Simons and Mense, 1997). First of all it is necessary to establish the fact that resting tension is measurable in the absence of contractile activity. Some studies supporting this view will be presented below. We will later discuss the possible origin of resting tension and show that this could lie inside the muscle cell or in the surrounding connective tissue.

Studies by Clemmesen, 1951; Ralston and Libet 1953 and Basmajian, 1957 have shown that there is a measurable tension in resting skeletal muscle in the absence of electric activity measured by EMG. Over a period of six years, thousands of muscles in hundreds of persons were studied electromyographically. The completely relaxed muscles in these subjects showed no neuromuscular activity (Basmajian, 1957)

Furthermore resonant frequencies studies show no change in muscle tone before and after anesthesia (Lakie et al., 1979; Lakie et al., 1980) and finally when measuring the human hip (Walsh and Wright, 1987) muscle tone was recorded without any electrical activity (EMG).

Based on the above findings it seems that at least some of the resting tone found in skeletal muscle stems from intrinsic properties of the muscle itself and not contractile activity.

The characteristics of muscle tone in resting skeletal muscles – what has been recorded and how?

The general method used to investigate the tension of resting skeletal muscle is its ability to resist stretch. Both during one specific stretch or/and differences between repeated stretches. The studies have been performed either in vivo on animal/humans typically resistance through joint movement or in vitro of prepared muscle tissue suspended in different solutions and put on stretch. The latter more frequent in newer studies.

Results have been presented typically as time or stretch velocity vs. force diagrams. The characteristics of the stretch force response have proven important in explaining the causes for resting

muscle tone. Particularly in distinguishing pure viscous response from an elastic response (or a combination of the two). Typically a viscous response will be when tension is proportionate to the stretch velocity. An elastic response would be when tension is proportionate to stretch length.

Animal studies by Hill (1968) show an initial high resistance to stretch in a small part of the length change of skeletal muscle. He called this the short range elastic component (SREC). This observation has been reproduced by several others since but there has been much dispute on the underlying cause for this finding. We will present these different views later in this paper.

Early studies also show that the resistance to stretch in relaxed skeletal muscle has a history-dependent factor. It seems that the resistance decreases swiftly after the initial stretch also known as bi-phasic response. This has been compared to what physiologists term thixotropy.

In biomechanics the term thixotropy is used to describe the temporary reduction in stiffness that a muscle exhibits following an imposed movement (Cambell and Lakie, 2007). Another maybe more explicit definition is proposed by Walsh and Wright, 1988 : thixotropy is a property of long-chained polymeric molecules in solutions; weak physio-chemical bonds have disrupted by movement and take some time to reform.

As mentioned the thixotropic property of skeletal muscle has long been acknowledged. Early studies (Sherrington, 1925; Brown, 1929) noted a tensile resistance at beginning of stretch and also smaller response after two and three stretches (Mutungi and Ranatunga, 2000).

Studies on thixotropy in human skeletal muscle have been performed by others like Lakie and Robson (1987) and Walsh and Wright (1987). These studies show thixotropic properties both in the human hip and finger. Earlier studies (Lakie et al, 1984; MacKay et al, 1986) show the same results for wrist and elbow.

Although there is some controversy on the strength and time length of the thixotropic property of skeletal muscle it seems to be a well documented observation. We believe that discrepancies among studies could be a result of the different experimental designs – human/animal muscle, different muscles, vivo/vitro and lastly different measurement apparatus.

Furthermore the thixotropy of the muscle is not necessarily a constant stable phenomena. It could be that thixotropy is a dynamic property that will change according to the set of conditions that the musculoskeletal system has to respond to. We will discuss this later in this paper.

None indisputable evidence has yet been launched in order to explain the thixotropic behavior of skeletal muscle but several different possible theories have been presented. The two dominant theories are: 1. Cross Bridges between contractile proteins – Actin/Myosin 2. Properties of other muscle

proteins such as the titin or other filament molecules interactions. These theories will be presented and discussed in the following.

Intrinsic properties of the muscle as an explanation for resting muscle tone : possible explanations

As we have shown there is good evidence of the resting tension in relaxed skeletal muscles and that this tension seems to inherit several distinct characteristics. We will now try to present and explain the most important theories in the literature.

Several intrinsic properties of the muscle have been proposed in order to explain resting muscle tone. Among these are:

1. Cross-bridges between contractile proteins – Actin/Myosin
2. Properties of other muscle proteins than myosin and actin such as the titin and possible some other filamentous molecules.
3. Intracellular/extracellular water pressure and
4. Active fascial contractility.

1. Cross-bridges between actin and myosin

It is a well known fact that cross bridges formed between actin and myosin in the presence of calcium is responsible for the active contraction of skeletal muscles. It is now proposed that these bindings also could be at least partly responsible for the tone of relaxed muscle. This is based on the assumption that cross-bridges between actin and myosin can develop without neurological activity.

We find this an interesting theory as this would easily explain the initial rise in tension that is recorded in several studies. This tension will last for as long as the cross-bindings exist and drop off when these are lost when put at stretch or when the overlap between actin and myosin is less optimal. Secondly it could explain the thixotropic behavior of the resting tension by the fact that it will take some time for these cross-bindings to regenerate after one stretch reducing the initial tension in repeated stretches.

Below we will now present some of these findings as they have been modified and revised over a 30 years' time span. In short most studies partly try to modify the basic cross-bridge model and partly try to make the theory applicable to other observed phenomena in muscle. No definite conclusion has yet been approved but we believe that strong evidence of a cross-bridge exists.

Hill (1968) suggests that the elastic properties of resting skeletal muscle are due to a small number of cross bridges between actin and myosin. Hill presents the terms short range elastic component (SREC) and filament resting tension (FRT). SREC demonstrates the fact that the tension is high at the beginning of the stretch and rapidly declining and FRT is the equivalent of the memory dependent tone of relaxed skeletal muscle (thixotropy).

Hill's work demonstrates that fibers in hypertonic solutions show increased tension implying the possibility of formed cross-bridges between myosin/actin. This follows the fact that these bindings are Ca2+ dependent. This theory is further discussed by Lannergren (1971) replicating the results of Hill (1968) with regard to the initial muscle stiffness namely the SREC and to the increased stiffness in hypertonic solutions (sucrose-Ringer). Lannergren also found an actual decrease in SREC when elevated K+ and caffeine concentrations which he believed could question the actin/myosin cross-bridge theory.

In addition Lannergren found that the time of redevelopment of SREC and FRT after a stretch is clearly different proposing another underlying mechanism than cross-bridges. Contrarily Moss, Sollins and Julian (1976) showed that the size of the SREC in skinned skeletal and cardiac muscle fibers was dependent on the concentration of Ca2+ again supporting the cross-bridge theory.

Further discussion on the cross-bridge theory is presented by Campbell and Lakie (1998) who suggests that actin and myosin are linked by a small number of slowly cycling cross bridges, the so called "Cross-bridge Population Displacement Mechanism (CPDM). The CPDM aims to theoretically model the findings underlying the SREC and FRT under the assumption that these are due to the development of cross-bridges between actin and myosin. The main difference from earlier work is the fact that this model is based on the assumption of slow cycling of the cross-bridges compared to rapid cycling cross-bridges proposed in earlier work. Slow cycling of the cross-bridges enables the model to incorporate the initial stiffness in the stretch (SREC).

The model consists of three components: 1. Passive structures within the sarcomer (titin, sarcolemma, sarcoplasmatic reticulum and other passive structures), 2. Active structures within the sarcomer (cross-bridges actin/myosin).

By including passive structures inside the sarcomer to the model it also seems to make the model fit to the observed relationship between stretch velocity and tension (Hill, 1968 and Lannergren, 1971) which was put forward as a shortcoming of the pure cross-bridge theory.

A tension related phenomena in skeletal muscle is latency relaxation (LR) namely the drop in tension that occurs at the onset of a contraction (Hill 1968, Lannergren 1971). Caflin et al (1990) interprets findings in studies on muscle length dependence on LR that these are of cross-bridge origin.

Work by Proske and Morgan (1999) presents a cross-bridge model that is applicable to both latency relaxation and SREC. It is proposed that cross-bridges can exist in three states; one responsible for the resting stiffness (SREC), requires resting levels of calcium. When, during activation, calcium levels rise, cross-bridges enter a low-force, high-stiffness state, signaled by latency relaxation, before they move to the third, force generating state (Proske and Morgan 1999).

As one can see a substantial investigation has been conducted to improve and expand the cross-bridge theory. Some of the studies aim to replicate the measured characteristics of the stretch response solely by changing the presumed cycling of the cross-bridges. Others have incorporated other intrinsic characteristics of the muscle together with cross-bridges to account for the resting tone. And lastly some will make cross-bridges responsible for other muscle phenomena such as latency relaxation making the model likely more robust.

We think that there is a lot of evidence supporting the cross-bridge theory. The fact that the stretch response seems to be biphasic and that tension seems to be dependent on calcium concentration strongly supports the theory. However we think that the cross bridge theory more likely to be one out of many different contributors to tone in relaxed skeletal muscle like what is presented in the CPDM model put forward by Cambell and Lakie (1998).

2. Properties of other muscle proteins such as the titin and/or other filament molecules interactions

Skeletal muscle cells consist of several big/complex structural proteins in addition to actin and myosin. These give support to contractile proteins and enables forces of contraction to transfer on to ligaments and eventually joints. It is of our belief that these proteins in their nature (shape and form) necessarily also could give rise to some of the passive characteristics of the muscle.

It is likely that passive tension can stem from either sharing forces between filamentous structures or from the unfolding/unwinding of proteins in these structures. We will also show that there might be interaction between filaments similar to those between actin/myosin and that these could in fact be sensitive to calcium like actin/myosin.

Titin is one of the biggest proteins in the human body and is therefore of big interest in this manner. In the following we will present the development of theories that supports the view that interactions or inherent properties of this protein will contribute to tension of relaxed skeletal muscle. Research is both based on studies looking at the characteristics of the stretch response but also on investigating protein structures/architecture as well as their possible interactions.

In a study on passive force response to stretch of frog muscles Bagni et al (1995) found evidence of non-cross-bridge mechanisms for passive muscle tension. The tension responses were separated into three components. The initial rapid phase of tension rise called (P1) where it was found that the tension rose proportionate to the velocity of the stretch. They argue that this implies a viscous nature of the muscle in this phase. The P1 one was taken to represent the SREC and overall in accordance with earlier studies (Hill, 1968; Lannergren, 1971) which also show a multi phase tension response but the viscous nature of the response found was interpreted as non-bridge mechanism.

The rise in force beyond P1 was described by a viscoelastic component called P2 were velocity is constant and force proportionate to stretch length. The final tension designated P3 was assigned to a non linear elastic element.

The relative myofilament overlap and sarcomere length were also measured during stretch showing that the tension increased even at sarcomere length beyond that of maximum overlap in which one might not suspect if cross-bridges was the underlying source of the tension (Bagni et al, 1995).

Discussing other underlying mechanics than cross-bridges Bagni et al (1992) propose that P1 could stem from the relative sliding motion of thick and thin filaments. This would be in accordance with work by Huxley (1980). A possible explanation for P2 could be related to the presence of the titin elastic fibers.

Matungi and Ranatanga (1996,1998) extend observations to mammalian muscle. Their findings were in accordance with those of Bagni et al (1995) with regard to finding distinct discontinuities in the tension response (P1 and P2). However they execute their measurements at much slower stretch velocities. P1 having same tension-stretch velocity dependence as of Bagni et al (1995) supports the view that it has its origin in a purely viscous resistance in other words non-cross-bridge origin.

The results of Matungi and Ranatanga (1996) also show differences in the measured tension related to stretch velocity and fiber type. The breakpoint tension between P1 and p2 is estimated 2 times bigger for fast twitch muscle fibers. It is also emphasized that the skinning of the fibers reduces the viscous tension of P1. We emphasize that these findings underline the many possible sources of error in comparing the different studies.

The possible sources for P1 and P2 presented by Matungi and Ranatanga (1996) are in line with Bagni et al (1995). The difference between fast and slow fibers is thought to be due to differences in the myoglobic structures, mitochondrial volume, lipid droplets and/or different Z-membrane width. With reference to works by Hu at al (1986), Horwits (1992) and Hill and Weber (1986) suggesting the presence of different titin isoforms Mutangi and Ranatanga imply that the complexity of the titin molecule makes it more likely to inherit viscoelastic properties than merely simply elastic.

A vast number of studies have been conducted on the nature of the giant molecule titin. Labeit and Kolmerer (1995) produced results on the architecture of titin showing that different isoforms correlated with passive muscle tension. Titin is likely to be the most important elastic stabilizer of the relative position of actin and myosin filaments through its position in the I bond in striated muscles (Tskhovrebova and Trinick, 2003). Additional support for the proposal that titin develops passive force in response to passive stretch has been obtained by experiments with low dose of ionizing radiation to degrade titin and that showed that this greatly reduced the ability of relaxed skeletal muscle fibers to generate passive force (Horowits, 1986)

Though outside the scope of this paper to fully reproduce the full insight into titin and all its molecular properties nevertheless new and improved techniques like optical tweezers and atomic force spectroscopy have proven to produce new and interesting results in the field. Titin presents in a remarkable variety of sizes and configurations ranging from 600kDalton to 3,7MDaltons in weight and 0,2 – 1,4 um. It is likely that the contour length and the intrinsic extensible properties of the I band are main determinants of muscle tension. Smaller isoforms of titin (shorter I bands) has been found in typically stiff muscles such as cardiac muscle and contrarily longer isoforms is found in typically extensible muscle as the soleus (Tskhovrebova and Trinick, 2003).

Several interesting domains of the titin molecule have been identified. The PEVK domain has won some attention as it is believed to inherit properties that may interact with alpha-actin (Kulke et al, 2001; Like et al, 2002; Bianco et al, 2007). It is hypothesized that an interaction of such kind could function as a viscous element in the change of muscle length (Bianco et al, 2007) In addition the PEVK domain can unfold to ten times it folded length (Tatsumi et al, 2001) and is therefore of great interest with regard to its contribution to the elasticity of the muscle.

Another property of the PEVK domain is its affinity to Ca2+ first reported by Wang, 1985. Tatsumi et al, 2001 reports Ca2+ binding sites to a 400kDa fragment on the N-terminal based on fluorescence techniques. It is believed that Ca2+ not only alters configuration of the extensible regions thereby modulating the possible elastic properties but also regulates binding to actin (Granzier and Labeit, 2006) discussed above. A reduction in the passive tension in cardiac myocytes has been reported after phosphorilation of certain domains of the titin molecule (Yamasaki et al 2002)

(Joumaa et al, 2008) investigates further the relationship of Ca 2+ and resting tone in relaxed skeletal muscle. Their findings indicate that the Ca 2+ dependent tension is partly due to cross-bridges between actin-myosin 75% and cross-bridges between titin filaments.

We believe that there is strong evidence that structural proteins such as titin play a substantial role in contributing to tone of relaxed skeletal muscle. It seems that evidence lies not only in the way that

these proteins is arranged (parallel sharing forces), but also in the way that these will unfold/unwind under stretching as well as possible interact with another.

Further discussion on cross-bridges vs. properties of other intra muscular structures.

As presented above there are different views and findings related to the passive characteristics of skeletal muscle. Solid evidence based data is obtained and presented for a cross-bridge mechanism as well as for a non-cross-bridge mechanism. A short recap is presented below.

Our review of the literature has shown that the research community has been divided; researchers have tried to partly present strong evidence for a view and partly discourage the opinions of others. However some newer papers have managed to present models that incorporate both theories which are in our view a more intuitively correct description of the tension in a relaxed muscle.

The increased tension response (SREC/FRT) to Ca2+ (and 2,3-butanedione 2-monoxide (BDM)) concentrations could be a strong advocate of the cross-bridge theory in that these substances are modulators of actin/myosin interactions directly and indirectly (Campbell and Lakie, 1998; Hill, 1968).

Bagni et al (1995) and Rantatunga and Matungi (1998) present findings that the initial stretch response of a relaxed skeletal muscle is of a pure viscous nature. These findings suggest other mechanisms underlying the SREC and FRT than those of the cross-bridge theory.

Both Hill (1968) and Lannergren (1971) prove that the tension is more than proportional to the velocity of the stretch. This tension-velocity dependence is clearly difficult to explain with regard to the cross-bridge theory as cross-bridge mechanism is more likely to be of a merely elastic nature in which tension is proportional to stretch length not to stretch velocity (Bagni et al, 1995).

Tension increase has been reported even at sarcomere length beyond that of maximal overlap (Bagni et al, 1995) not consistent with what one might suspect from a cross-bridge view.

Theoretical modeling (Proske and Morgan, 1999; Campbell and Lakie, 1998) implying at least a cross-bridge component seem to agree reasonable well with experimental data.

Could other mechanical properties give rice to the thixotropic behavior (SREC/FRT) of skeletal muscle? The complex protein titin has been presented as possible contender. When inducing radiation damage to titin in rabbit muscle (Horwits et al 1986) reduction in resting tension was reported. Campbell and Lakie (1998) announced that titin has to inherit properties of a “softening spring”

should it account for thixotropy. Work by Tskhovrebova (1997) shows that titin behaves as a non linear stiffening elastic element.

However newer research has revealed new and interesting properties of titin and its different domains many of which could be proven to explain at least some of the complex nature of the resting tension of skeletal muscles.

The Cross-Bridge Population Displacement Mechanism model presented by Campbell and Lakie (1998) consists of elements from both cross-bridge theory and other filament properties theories. This is in line with recent studies by Joumaa et al (2007) where data suggest that the Ca2+ dependent tension of passive skeletal muscle is based on the properties of titin (25%) and the rest on possible cross-bridge formation.

The data reviewed above strongly suggest that more than one biochemical property contributes to resting muscle tone. Both as a result of the molecular structure of myofibrillar proteinsl and/or by interaction between them as with the cross-bridge theory. Their relative magnitude of impact on resting tension is yet to be discovered. More studies need to be conducted.

3. The importance of water content in the muscle

It is well known that the water content in the muscle varies during exercise. Saltin and coworkers (1984) showed that 3x3 minutes of intense bicycle exercise increased that total water load in the vastus lateralis from 313 to 359 ml/100 g dry weight water and an extracellular increase of H_20 from 34 to 60 ml/100 g.

This finding is recently supported by the work of Raja et.al (2006). Using multiple-frequency bioimpedance analysis, they measured a 12.6 % increase in intracellular water content during exercise.

Another paper from 2006, presents a simple model where it is concluded that “the relationship between water activity, viscosity and stiffness is discussed. It is concluded that the three parameters vary cyclically and that when water activity decreases (sarcomere shortening, cross bridge attachment) viscosity and stiffness increases” (Grazi, Bona 2006) In this article, it is emphasized that viscosity has shown as related to water activity. As this decrease or the osmotic pressure by proteins increase, viscosity and stiffness increases.

Intramuscular pressure is influenced by water content, compartmental volume as well as properties of the surrounding muscle fascia (Bourne and Rorabeck 1989 from Korhonon et.al 2005)

Karhonen et.al (2005) shows a linear relationship between muscle tone, EMG results and intramuscular pressure ; and conclude that both surface EMG and myotonometry are indicative of intramuscular pressure.

Based on this, water content may have an important role in muscle tone variations as related to intramuscular pressure and adds to the theories of cross bridges/titin molecule to explain muscle tone under resting conditions. What remains to be explored experimentally is whether or not there is a direct correlation between the water content in the muscles related to the muscle tone and how this may vary under resting conditions.

4. Contractile properties of muscle fascia

In addition to the above, could it be that characteristics of the connective tissue in the muscle are contributing to resting tension? A more recent discovery has shown that connective tissue surrounding the muscle cell might inherit contractile properties. Intuitively one might expect that increased tension in connective tissue necessarily would increase tension of the whole muscle since connective tissue surrounds each muscle fascicle (perimysium) and the whole muscle belly (epimysium). It is also interesting because it is a well known fact that the distribution of connective tissue differs widely across different muscle groups. This could therefore partly explain the difference in resting tone measured in different muscle groups (Gavronski G, 2007)

Connective tissue in musculoskeletal system consists of the endomysium, perimysium and epimysium and it is part of the structures and mechanisms that contribute to passive properties of the muscle (Gajdosik 2000). We have decided to present some recent insight to the function of the perimysium as it is believed that the perimysium inherits some contractile properties in which resembles that of the muscle itself although this is a property that lies outside the muscle cell .

Examination of the perimysium with light microscopy and scanning electron microscopy has revealed that the orientation of the crimped collagen changes as the length of the muscle changes. The perimysium undergoes a mechanical deformation and realignment that should contribute to the exponential, or curvilinear increased resistance when a muscle is stretched (Gajdosik, 2000).

Of the total connective tissue in skeletal muscle perimysium is by far the most abundant (Light et al, 1985). Findings also seem to conclude that it is the amount of perimysium differ substantially between muscles (Mc Mahon, 1984; Borg and Caulfield,1980)). These observations need to be investigated in relation to elasticity.

An interesting property of some connective tissue cells is their ability to express the gene for smooth muscle actin and thereby possible inherit contractile properties. Actually it has for some time been

acknowledged that several cells in the musculoskeletal systems has the ability to express muscle actin , so called alpha-actin (Spector, 2002). These findings are based on immunohistochemistry. Some examples are: Chondrocytes, meniscal cells, ligament cells, invertebral disc cells and osteoblasts. This property is believed to relate to wound and tissue healing and the expression of alpha-actin is typically seen in relations to tissue damage (Spector, 2001). The fibroblast that express alpha-actin are called myofibroblasts.

The presence of myofibroblast in fascia has been demonstrated (Schleip et al, 2004; Masood et al, 1996; Pipelzadeh and Naylor, 1998). Work on the human anterior cruciate ligament by Spector and Murray (1999) show that the density of myofibroblasts correlates to the level of vascularization and is greater in the more crimped areas. Being highly vasculated and crimped areas Schleip et al (2005) postulates that perimysiumet could be rich in myofibroblasts.

We believe that there are convincing facts supporting the contracting fascia theory. Although more research need to be done to fully prove that this contractile property is of great enough magnitude to impact the resting tone of skeletal muscle. In addition further investigations needs to be conducted in order to reveal the mechanism that regulates the expression of contractile proteins in connective tissue cells. Lastly it would be of interest to know what type of stimuli these cells would response to in order to contract.

Individual variations in muscle tone

We have now presented the main theories/factors behind muscle tone in skeletal muscles. But do these vary and thereby explain individual variations in muscle tone over time?

As stated in the introduction, one of our aims with this article is to look at individual variations in muscle tone ; between individuals and over time for the particular individual.

Individual variations in muscle tone is interesting for many reasons. It is interesting both in terms of subclinical implications, how to treat an pathological increase in muscle tone, and to understand the theoretical framework behind muscle tone.

Research shows that individual differences exists “if the pooled data from the muscles are compared between subjects…..in the relaxed state, these parameters are specific to the muscles and to the individuals” (Gavronski G, 2007)

Variations also exists between ethnic groups, as shown by Fakashiro et al (2002)

Unfortunately, there is little specific information about how individuals vary when it comes to cross bridge formation, thixotropy, fascia contraction. More information on this would have made it easier to pinpoint what the more exact mechanisms for these variations.

In terms of water content, there is some information in the data compiled from the studies about water content during exercise. In the example of the vastus lateralis, the male subjects at rest varied from the total H_20 content from 308 to 326 (in ml/100 g dry weight muscle tissue) and extracellular H_20 from 22 to 34. After exercise the values showed variations of 320 to 380 ml/100 g and 47 to 93 ml/100g for the respective total and extracellular H20 (Sjogaard and Saltin, 1982)

However, what is interesting is that Gavronski et.al (2007) in the article "Evaluation of viscoelastic parameters of the skeletal muscles in junior athletes", evaluates eight muscles and compare them using a myotonometer (Myoton 2) . These muscle are the biceps brachii (caput longum), triceps brachii, biceps femoris (caput longum) rectus femoris, tibialis anterior, gastrocnemius, latissimus dorsi and pectoralis major (pars sternocostalis).

The data shows that the tibialis anterior is under constant tension, as compared to many of the other muscles. It is the most tense, elastic and stiff muscle – and interestingly : in the contracted state the elasticity did not change, although the stiffness in the muscle near doubled. The gastrocnemius showed similar but less pronounced characteristics.

The importance of postural stability and the role of the leg muscles may be part of the explanation to this.

There is also evidence that an chronic increase of muscle tone in subjects complaining about overtraining/lack of athletic performance (Vain A.2002) and with patient that chronic conditions such as compartment syndrome (Karhonen RK et al 2005) or chronic muscle pain in the trapezius muscle (Vain, 2007)

Discussion

There are several important and interesting aspects to look at and discuss based on the above.

In our view there has been a lack of a proper definition of skeletal muscle tone and resting skeletal muscle tone in particular. Most research has been focused on muscle tone related to contractile activity. We emphasize the importance on muscle tone based on intrinsic factors of the skeletal muscle as this could be important in understanding and treatment of muscle diseases or in relation to optimize sports performance.

We have chosen to use the definition of tone in relaxed skeletal muscle that is presented by Simon and Mense (1998) that is: "the elastic and/or the viscoelastic stiffness in the absence of contractile activity". This implies factors inside muscle namely muscle cell or surrounding connective tissue.

Earlier papers discuss the challenge of objectively measuring muscle tone. This is obviously essential for studying the phenomena. There is good evidence in the research papers that the newer measurement equipments such as the Myoton 2 can reproduce objective results of muscle tone levels. To measure only the resting tension of the muscle, EMG is used to rule out any contractile activity.

We believe that there is strong evidence for a measurable tone in relaxed skeletal muscle based on the articles presented. In the literature several interesting intrinsic causes of this tone have been presented: cross-bridges theory, titin or other filamentous proteins, water content and lastly contractile fascia. Any of these factors are in our view possible causes but it still rests unsure the magnitude of impact each factor inhibits.

What is the relative importance of each respective factor in relations to the "total sum" of muscle tone in relaxed skeletal muscle? Which of the factors titin molecules, cross bridges, fascia contractility and water content are the most important?

This is unclear, and one problem is that most of the models we have looked at aims to explain muscle tone (variations) mainly based on one factor exclusively ; either titin molecules, cross bridges, fascia contractility or water content respectively. Only two integrated models have been presented. One by Youmaa et al (2008) suggests that 25 % titin contribution an 75% actin – myosin contribution. Another by Cambell and Lakie (1998) combines cross-bridge theory and other filament theory. But still in these models, the possible role of water content and fascia contractility is not taken into account. What seems missing is a comparison between the factors and how influencing one of them may change the resting muscle tone compared to influencing the others.

In the introduction we stated a need to look at variations of resting muscle tone over time. It seems that there is some individual variation. A good question in extension to this is: are some of the intrinsic factors more sensitive to changes over time – long term and short term and does this differ from one muscle to the other ? We will try to discuss this below.

What may be the case is that factors such as water content has a more dominant influence short term while more "structural" factors such titin molecules are a more dominant in the total sum – but does not vary as much over short periods of time.

In fact, changes in water content of muscle in resting muscles after exercise compared to before exercise show that water content in the muscles has the ability to shift in the course of short period of time. We have shown that there is a connection in resting tension to water content. It also a fact that

level of intracellular/extracellular water is easily manipulated for example through electrolytes or possible different nutrition.

Discussions with elite athletes, especially in power sports, reveal the use of protein supplements and creatine for more “pump”/subjective feeling of power in the muscles. In the course of relatively short time, 1-2 weeks after the start of these supplements, these athletes report a gain of several kg of bodyweight . This increase can only be a result of mostly increase of water content in the body taking into consideration the short time span. At the same time, there is a reported increase in “pump”/power output. This reported increase in the muscle “pump”/power feeling may in fact be caused by changes in water content leading to alterations in muscle tone based on our finding above. If this is the case, looking at manipulating water content by nutrition may be an interesting way to go in terms of “optimal” muscle tension for athletes.

As in the case of titin molecules, there are no data concerning short term changes, but there is a finding in aerobic trained mice that shows a significant increase in titin molecule expression after a 45 days exercise regime (Ballafiore M et.al 2007). This suggests that the titin molecule is in fact possible to alter, at least in a long term perspective. The most likely course is through change expression of titin genes but it would be of interest to see if change to intracellular proteins such as titin also could occur after mechanical influence or altered intracellular environment (pH for example).

Furthermore, in the section about individual variations of muscle tone, we referred to the different muscles/muscle groups as having different muscle tone. For example, postural muscles such as tibialis anterior has a constant high tone even at rest compared to most other muscles. The reason for this may be different concentrations/distribution of the different intrinsic factors influencing muscle tone. With water content these differences between different muscles has been show with comparisons between the vastus lateralis and soleus: the vastus lateralis having the lowest extracellular H20 while the soleus the lowest total H20 (Sjoogaard 1982 et.al) In the case of titin molecules, cross bridges, fascia contractility there is unfortunately no clear date on individual differences from one muscle to the other. But it is a well known fact that the amount of connective tissue in a muscle differs greatly across muscle. In light of the discovery of contractile fascia it would have been interesting to investigate a possible connection between degree of connective tissue in skeletal muscle and resting tone.

More insight into how these different factors vary and can be manipulated would be valuable info that may help optimize “muscle tension” for athletes and treat high muscle tone in patients with chronic muscle pain as stated in the introduction. One common treatment method athletes use for muscle recovery between hard trainings is massage. Clinical work (Høgseth 2005 et.al) on overtrained athletes suggests that massage done in the opposite direction of muscle fiber direction can be successfully used to increase recovery time from an overtraining period. It may be possible that the massage being done

in the opposite direction of the muscle fibers can mechanically influence intrinsic factors such as cross bridge formations and thereby lower the increased resting muscle tone in the overused muscle.

The possibility of manipulating muscle tone is interesting in terms of practical work to relieve muscle stress/a pathological increase in tone. Little research has been done in this field. With the ability to measure changes through the current measurement equipment this seems like an interesting aspect to explore.

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