Katarina Juhart
Vojko Strojnik*

MUSCLE VISCOELASTIC STIFFNESS DURING MUSCLE CONTRACTION AND INHIBITION

VISKOELASTIČNA TOGOST MED MIŠIČNO KONTRAKCIJO IN NJENO INHIBICIJO

ABSTRACT

The aim of the present study was to analyse changes in muscle viscoelastic stiffness during reciprocal inhibition and isometric contraction to determine whether it would further decrease during increased reciprocal inhibition following a behaviour observed during muscle activation. Fifteen young adult volunteers performed a dorsal (reciprocal inhibition of the soleus muscle) and plantar (contraction of the soleus muscle) flexion at different intensities (10 %, 20 % and 30 % of MVC). During the dorsal and plantar flexion as well as in the case of a relaxed muscle, the viscoelastic stiffness of the m. soleus (Myoton III device) was measured. In addition, the H-reflex during a relaxed muscle and a dorsal flexion was examined to test the presence of reciprocal inhibition. The viscoelastic stiffness of m. soleus increased with a stronger plantar flexion, which is well in line with the cross-bridge theory. During a stronger dorsal flexion, the H-reflex was increasingly reduced, showing the presence of reciprocal inhibition, although the viscoelastic stiffness of the m. soleus was increased. Our conclusion was that viscoelastic stiffness during inhibition of the soleus muscle did not follow the trend observed when the muscle was activated. The multiple mechanisms involved in viscoelastic stiffness control of the soleus muscle acting in opposite directions were discussed.

Key words: muscle stiffness, H-reflex, Myoton, reciprocal inhibition, dorsal and plantar flexion

University of Ljubljana, Faculty of Sport, Ljubljana, Slovenia

**Corresponding author:*
Vojko Strojnik, PhD
University of Ljubljana, Faculty of Sport
Gortanova 22
1000 Ljubljana
Tel.: +386 1 520 77 00
E-mail: vojko.strojnik@fsp.uni-lj.si

IZVLEČEK

Namen raziskave je bil analizirati spremembe viskoelastične mišične togosti med recipročno inhibicijo in izometrično kontrakcijo ter ugotoviti ali se s povečevanjem recipročne inhibicije togost zmanjšuje in sledi smeri sprememb opazovanih med mišično aktivacijo. Petnajst prostovoljcev je izvajalo dorzalno (recipročna inhibicija mišice soleus) in plantarno (kontrakcija mišice soleus) fleksijo različnih intenzivnosti (10 %, 20 % in 30 % največje izometrične kontrakcije). Med dorzalno in plantarno fleksijo, kot tudi v mirovanju, je bila izmerjena viskoelastična togost m. soleus z napravo Myoton III. Spremembe recipročne inhibicije smo spremljali med mirovanjem in dorzalno fleksijo s H refleksom. Viskoelastična togost m. soleus se je povečevala s povečevanjem plantarne fleksije, kar se sklada s teorijo o prečnih mostičih. Med stopnjevanjem dorzalne fleksije se je H refleks postopoma zmanjševal (recipročna inhibicija), medtem ko se je viskoelastična togost povečevala. Ugotavljamo, da viskoelastična togost med inhibicijo mišice soleus ne sledi trendu sprememb dobljenih med mišično aktivacijo. V razpravi so obravnavani različni mehanizmi vključeni v kontrolo viskoelastične togosti mišice soleus, ki delujejo v nasprotnih smereh.

Ključne besede: viskoelastična togost, H reflex, Myoton, recipročna inhibicija, dorzalna in plantarna fleksija

INTRODUCTION

Muscle stiffness derives from intrinsic muscle viscoelastic properties (Masi & Hannon, 2008). It can be defined as the resistance of a muscle when being passively stretched (Schleip et al., 2006). Muscle resistance to a stretch relates to several active cross-bridges (Rack & Westbury, 1974) which is the main reason that muscle stiffness grows with increased muscle contraction. Alternatively, muscle stiffness can be assessed by analysing the muscle's response to a local vibration (referred to as viscoelastic stiffness), a method that has demonstrated sensitivity and high reliability during different contraction levels (Bizzini & Mannion, 2003). An important difference between the method of local vibration and muscle stretching is its acute effect on measured characteristics. Local muscle vibration is neutral and allows repeated measurements without the measurement itself affecting the muscle's mechanical properties (Leonard et al., 2003). This is not the case with muscle lengthening since every measurement affects the muscle's mechanical properties (Nordez, Cornu, & McNair, 2006; Guissard & Duchateau, 2004). Therefore, the local muscle vibration method seems to be of particular interest when studying acute changes and their time kinetics after treatment.

Changes in muscle stiffness during muscle contractions of different intensities are well explored. It has been consistently shown that muscle stiffness grows during increased voluntary muscle contraction (Rack & Westbury, 1974), which is related to a number of active cross-bridges during the contraction. However, changes in muscle stiffness during muscle relaxation have been studied less extensively. For instance, no studies have been found that analyse the muscle stiffness of a relaxed muscle with additional neural inhibition. Some attempts have been made in animal models where (Mannava et al. 2011) it was discovered that an injection of botulinum neurotoxin contributed significantly to the muscle-tendon unit's passive biomechanical properties by reducing its passive elastic properties. Similarly, static stretching reduced the H-reflex and passive muscle stiffness at the same time (Guissard & Duchateau, 2004). In the latter case, the reduced passive stiffness could be a consequence of repeated muscle stretches and not only neural inhibition.

One can assume that the viscoelastic stiffness of a relaxed muscle depends on a number of cross-bridges as well, albeit attached in a weak bond. Namely, the cross-bridges may be attached in two different states: a strong and a weak bond (Getz et al., 1998; Yu & Brenner, 1989) where a weak bond precedes a strong one. The weak bonds do not contribute to the muscle's active force generation but may develop substantial force during muscle stretching and therefore contribute to passive muscle stiffness (Cambell & Lakie, 1998). Since botulinum inhibits neuromuscular transmission and reduces the muscle stiffness in a relaxed muscle (Mannava et al., 2011), one can assume that neural inhibition might also result in reduced muscle viscoelastic stiffness.

Muscle stiffness measurements have an important practical value when it comes to assessing the effects of different interventions that can be observed via changes in viscoelastic stiffness. It may change after different treatments as potentiation (Sinkjaer, Gantchev, & Arendt-Nielsen, 1992), stretching (Reid & McNair, 2004; Bressel & McNair, 2002), resistance training (Ocarino, Fonseca, Silva, Mancini, & Gonçalves, 2008), fatigue (Ditroilo et al., 2011) and relaxation (Guissard & Duchateau, 2004). After treatment, muscle stiffness may increase or decrease and have a different time course. In specific cases, as with an abnormal muscle tone or spasticity which are partly associated with an impaired modulation of spinal inhibitory mechanisms (Mukherjee & Chakravarty, 2010), stretching may induce a strong reflex response making this method question-

able for such an assessment. Therefore, a simple non-invasive (with no effect on subsequent measurements) tool to assess changes in the viscoelastic stiffness of an activated as well as a relaxed (EMG silent) muscle would be very helpful. Since the stiffness during muscle contraction has already been well explored, the present study aims to test whether the viscoelastic stiffness of a relaxed muscle is sensitive to different levels of neural inhibition and compare it to the stiffness when the muscle is contracted.

METHOD

Subjects

The research team carried out the study on 15 young adult volunteers (11 females and 4 males; age: 26.5 ± 4.2 years, height: 173.1 ± 9.7 cm, body mass: 66.5 ± 13.7 kg). We informed the subjects about the procedures and obtained their written informed consent. We conducted the study in accordance with the Helsinki Declaration.

Experimental design

The subjects were first prepared and checked for EMG signals, H-reflex and for a proper position in a knee-joint measuring device. They then started with a standardised warm-up consisting of 6 min stepping on a 20 cm high bench with a frequency of 30 steps per minute and with a change of leg every minute. After the warm-up, the subjects performed three isometric maximum voluntary contractions (MVC) for plantar flexion (PF) and dorsiflexion (DF) to obtain the MVC torques. According to those torques, the submaximal torque levels were set to: (1) relaxed (no torque); (2) DF 10% of MVC; (3) DF 20% of MVC; (4) DF 30% of MVC; (5) PF 10% of MVC; (6) PF 20% of MVC; and (7) PF 30% of MVC.

Two minutes after the MVC, measurements of the H-reflex on the m. soleus were performed; first in the relaxed muscle for a baseline and then during randomly chosen dorsiflexions of different intensities. After two minutes of rest, the viscoelastic muscle stiffness of the m. soleus was measured by a myometer (Myoton 3, AS Muomeetria, Tallinn, Estonia). First we started with a relaxed m. soleus and dorsiflexions in the same order as for the H-reflex followed by three randomly chosen plantar flexion conditions.

Position of the subject

During the measurements the subjects were in a supine position with their hip and knee joints flexed at 90°. They were instructed to maintain a relaxed position except for the contractions. The right leg was fastened into a custom-made isometric ankle-joint plantar- and dorsal-flexion measuring device. The ankle-joint axis was aligned to the apparatus' axis of rotation and the shank was fixed within a rigid frame to prevent any movement in the knee and ankle joints.

Torque control

The force transducer with a constant lever arm built into the measuring device allowed us to measure the isometric torque of the plantar and dorsal flexions. The signal was acquired by the PowerLab System (ML880/P; ADInstruments, Sydney, Australia) at 1 kHz and smoothed with a low-pass filter (cut-off 7 Hz). For the MVCs of the plantar and dorsal flexion, the subjects were

instructed to steadily increase the torque to the maximum (not explosive contraction) and then maintain the maximum contraction for 3–4 seconds. The maximal torques were established as the greatest signal during the steady part of the MVC. At submaximal torque levels, the subjects were instructed to slowly develop and maintain the set fraction of MVC torque. Torque feedback was provided in real time on a screen in front of the subjects consisting of two lines: one was the live torque of the subject and the other was the torque to be achieved. When the subject aligned both lines in a stable manner, the measurements of the H-reflex or viscoelastic muscle stiffness were performed, normally two seconds after the set condition was reached.

H-reflex measurements

Nerve electrical stimulation was performed with a cathode (10-mm diameter; Ag–AgCl, Type 0601000402; Contrôle Graphique Medical, Brie- Comte-Robert, France) placed over the tibial nerve in the popliteal fossa and the anode (10.2 cm x 5.2 cm; Compex, SA, Ecublens, Switzerland) placed over the patella. Electrical impulses (single, square wave, 1-ms duration) were delivered by a constant current electrical stimulator (DS7A; Digitimer, Hertfordshire, UK) and the time interval between percutaneous stimulations was at least 10 s. The stimulation site providing the greatest amplitude of the evoked potentials was first located by a handheld cathode electrode (0.5-cm diameter). Once determined, the stimulation electrode was firmly fixed to this site with straps. The surface EMG signal was recorded from the soleus muscle with two pairs of bipolar oval self-adhesive electrodes with an interelectrode distance of 2.5 cm (10 mm diameter; Ag–AgCl, Type 0601000402; Contrôle Graphique Medical, Brie-Comte-Robert, France). The positioning and skin preparation for the EMG electrodes were carried out according to the SENIAM recommendation (Freriks & Hermens, 1999). The EMG signals (Biovision, Wehrheim, Germany) were recorded at a sampling rate of 5 kHz.

The recruitment curves of the H-reflex and the M wave were first recorded for the relaxed condition. Stimulation followed every 10 seconds for 20 effective stimuli to define the H-M wave relationship (stress was given to provide points throughout the whole linear part of the H-M relationship). The EMG data were collected and plotted in real time by a custom-made program written in Matlab using the NI-DAQ 6024 A/D converter (National Instruments). The peak-to-peak amplitude of each H-reflex was plotted against the size of the associated M wave representing a polynomial function. The linear part of the polynomial function (regression line) describing the relationship between the H-reflex and the M wave was used as the reference excitability level (Dyhre-Poulsen, Simonsen, & Voigt, 1991) and represented our base line (100%).

At each submaximal dorsal flexion, 5 to 8 electrical stimuli were delivered with 10 seconds of rest between them. The stimulation intensities corresponded to those of the first third of the linear part of the regression line measured during the rest (the left part of the regression line). For each contraction one stimulus was delivered. The relative position of each H-wave according to the base line at an amplitude of the corresponding M-wave was calculated and presented as an average value for statistical analysis. No traces of co-contraction of the m. soleus during dorsal flexion were observed in the background of the raw signal during the H-reflex measurements.

Muscle stiffness measurements

Measurements of the viscoelastic stiffness were performed with a myometer (Myoton III, AS Muomeetria, Tallinn, Estonia). Nontoxic marks were drawn on the skin on the lateral side of

the soleus muscle, close to the two self-adhesive electrodes used to record the EMG signal. The impact measuring head was placed on the marked point in a vertical position, crosswise to the tissue to be measured. Care was taken not to tilt the Myoton from the vertical position during the measurement by more than a few degrees in any direction. The average value of 15 measurements (5 contractions, each with 3 successive measurements) performed at each submaximal torque level of PF and DF and relaxed muscle was used for the further analysis.

Data analysis

The SPSS (18th release, SPSS Inc., Chicago, IL) was used for the statistical tests. The Kolmogorov-Smirnov test was performed to evaluate conformity to a normal distribution, and ANOVA with repeated measures to test differences among the mean values of conditions measured by the Myoton III and the H-reflex. A post-hoc evaluation of differences between single pairs was performed with a Bonferroni adjustment. If the homogeneity of covariance (sphericity) was violated, we used the Greenhouse-Geisser correction. We determined the level of statistical significance at $P<0.05$ (two-sided).

RESULTS

The mean values for the m. soleus viscoelastic stiffness measured by the Myoton III device increased as the contractions became stronger (Fig. 1). Interestingly, this happened for the plantar as well as the dorsal flexions. Significant differences ($P < 0.05$) were obtained in the plantar

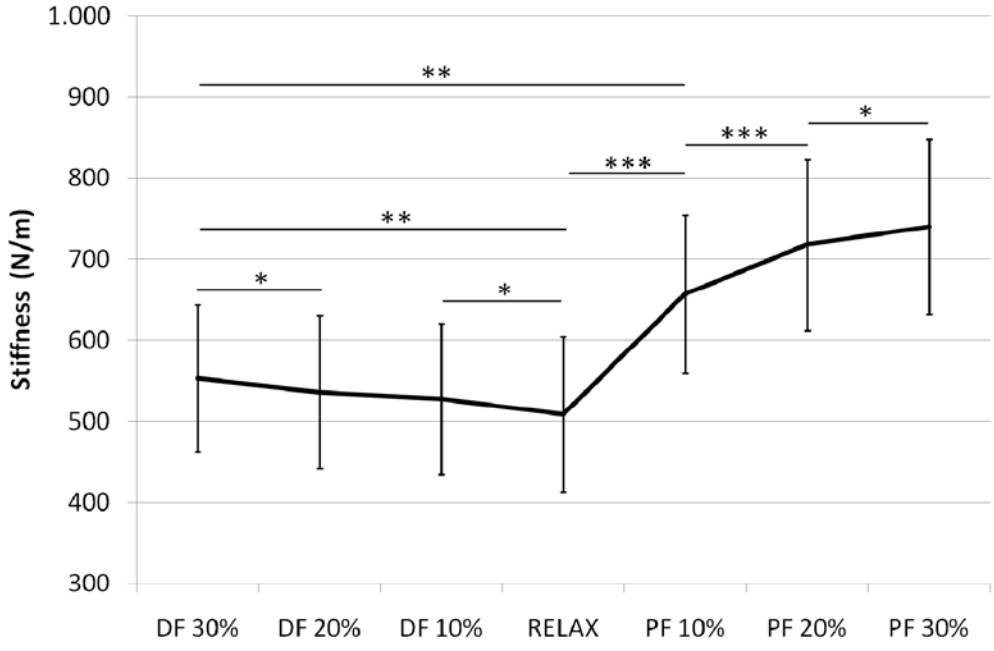

Figure 1: Mean and standard deviation of m. soleus stiffness for the analysed conditions. DF – dorsal flexion, PF – plantar flexion, 30% – 10 % of MVC, RELAX – relaxed muscle. * - p<0.05, ** - p<0.01, *** - p<0.001

flexions between all pairs of plantar flexions with a relaxed condition. The viscoelastic stiffness in the relaxed condition was the lowest. The plantar flexion had a considerably greater impact on the viscoelastic stiffness of the m. soleus than the dorsal flexion as the viscoelastic stiffness during DF 30% was significantly lower than during PF 10%.

Changes in the H-wave amplitude during dorsal flexion were in line with the strength of the PF where a stronger PF induced a greater H-wave reduction (Fig. 2). Significant differences were found in all pairs of conditions ($P < 0.05$) except between a relaxed muscle and DF 10% ($P = 0.57$).

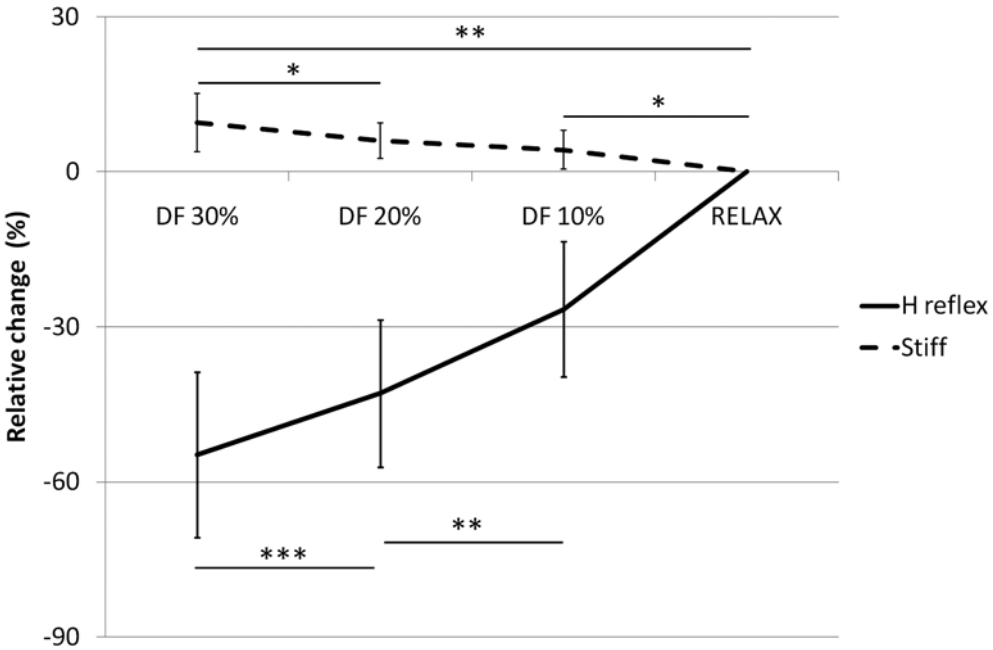

Figure 2: Mean and standard deviation of the H-wave and stiffness change of m. soleus during dorsal flexion of different intensities. Values are normalised to the RELAX condition. DF – dorsal flexion, 30% – 10 % of MVC, RELAX – relaxed muscle. * - p<0.05, ** - p<0.01, *** - p<0.001.

DISCUSSION

The study sought to explain the change of muscle stiffness measured in m. soleus according to the intensity of the contraction, regardless of whether m. soleus was activated as an agonist or reciprocally inhibited with a dorsal flexion. Muscle stiffness is related to the intensity of contraction (Rack & Westbury, 1974), with the novelty of the present study being the presentation of its behaviour during muscle inhibition. It was expected that greater inhibition would result in less stiffness. In this case, the viscoelastic stiffness would continuously decrease throughout the whole range of activation-inhibition states, enabling its relatively straightforward determination and making it very applicable to studies assessing muscle tone changes. However, stronger contraction regardless of whether it was PF or DF always resulted in greater viscoelastic stiffness of the m.

soleus. This effect was significantly more pronounced in PF when the m. soleus was activated than during DF when it was inhibited. As DF became stronger the H-reflex was more depressed. Therefore, we observed a situation where stronger DF viscoelastic stiffness increased while the H-reflex decreased.

During PF contractions, the stiffness of the m. soleus grew with an increase in the PF torque. the results are comparable to those observed by Bizzini and Mannion (2003) who showed the consistent behaviour of stiffness regarding changed muscle tension and length in a day-to-day repeatability. Since muscle stiffness (Rack & Westbury, 1974) as well as muscle force are related to the number of activated cross-bridges, it was therefore expected that increased muscle force would result in increased muscle stiffness.

The opposite was expected for reciprocal inhibition, namely to find a consistent decrease of viscoelastic stiffness that would be related to an increased inhibition level. The obtained changes in the H-reflex of the m. soleus during dorsal flexion in the present study were consistent with previous studies (Crone & Nielsen, 1989) showing that a stronger dorsal flexion depressed the H-wave to a greater extent, therefore pointing to an increased reciprocal inhibition of the m. soleus.

Since the expected decrease of viscoelastic stiffness was not observed, some other possible mechanisms involved in viscoelastic stiffness control were considered to explain such behaviour. The length change of the muscle-tendon complex might affect muscle stiffness during dorsal flexion (Bizzini & Mannion, 2003; Kovaleski, Norrell, Heitman, Hollis, & Pearsall, 2008). During DF, lengthening of the m. soleus could be expected and would cause increased viscoelastic stiffness. Greater DF torque would logically produce greater stretching of the m. soleus and therefore increased stiffness. However, we believe that in the present study this was not very likely since the leg was mounted in a measurement device that prevented ankle and knee joint position changes. Another possible explanation of the increased viscoelastic stiffness of the m. soleus during DF might be related to epimuscular myofascial force transmission between antagonistic muscles (Huijing, van de Langenberg, Meesters, & Baan, 2007). Crural fascia is attached in the upper and anterior part of the leg to the tibialis anterior and the extensor digitorum longus muscle, while posteriorly it covers the gastrocnemius and soleus muscles. As the fascia may transmit mechanical tension which is generated by muscle activity or external forces (Schleip, Klinger, & Lehmann-Horn, 2005) it could have an influence on m. soleus stiffness during m. tibialis anterior contraction by increasing the strain of ligaments and thus increasing the stiffness of the m. soleus.

If myofascial force transmission was effective, then two opposite effects of DF on viscoelastic stiffness occurred simultaneously whereby the myofascial force transmission seemed to have a stronger effect on the viscoelastic stiffness of the m. soleus than mechanisms related to the number of weak cross-bridge bonds in a relaxed muscle inhibited by reciprocal inhibition. It has been estimated that in a rat the myofascial force transmission between antagonist muscles (from DF to PF) may reduce the maximal triceps surae muscle force by up to 16% (Rijkelijkhuizen, Meijer, Baan, & Huijing, 2007). This shows that substantial extramuscular myofascial force transmission occurs between antagonistic muscles. Unfortunately, there are no quantitative data regarding humans, although we know that in humans this effect is also present between the PF and DF muscles (Huijing et al., 2007). For this reason, it would be pure speculation to quantitatively estimate the effect of myofascial force transmission from the DF muscles on the

m. soleus and consequently to estimate the effect of reciprocal inhibition on the viscoelastic stiffness of the m. soleus.

The results show that the measurement of viscoelastic stiffness may be applicable in patients after a stroke and other conditions with an elevated muscle tone, where subjective measurements of spasticity for assessing therapeutic interventions are still used in clinical practice because the muscle relaxation is not complete and therefore sensitive to viscoelastic stiffness changes. This agrees well with Leonard, Stephens and Stroppel (2001) who showed a significant relationship between the MAS (Motor Assessment Scale), a qualitative test of muscle tone, and myotonometer measurements, which were also able to detect smaller changes in muscle tone. Further, Rydahl and Brouwer (2004) reported that the changes evaluated by a myotonometer were related to total ankle stiffness and primarily reflect the intrinsic properties of the muscle tested in stroke survivors. However, it seems that in the case of the m. soleus it is impossible to assess the effect of neural inhibition by means of viscoelastic stiffness as measured with the Myoton III. The main reason is that it would not be possible to distinguish between the myofascial force transmission and relaxation (inhibition) and to therefore relate the data solely to inhibition. This also opens up the question of the relationship between viscoelastic stiffness and inhibition of (EMG silent) a muscle that is already relaxed.

CONCLUSION

In summary, in this study we assessed how affective the Myoton is for recording changes in the muscle viscoelastic stiffness of the m. soleus of healthy subjects during plantar and dorsal flexion in isometric conditions. Although the alpha motor neuron pool of the m. soleus exhibited activation and decreased excitability, this could not be seen uniformly in muscle viscoelastic stiffness. The stiffness changes during the activation part were consistent with other studies. M. soleus viscoelastic stiffness during neural inhibition, a unique contribution of the present study, did not decrease as expected. In this regard, we concluded that the ability to analyse intervention effects in such conditions performed on healthy subjects is questionable.

REFERENCES

Campbell, K. S., & Lakie, M. (1998). A cross-bridge mechanism can explain the thixotropic short-range elastic component of relaxed frog skeletal muscle. *Journal of Physiology, 1*;510 (Pt 3), 941–62.

Crone, C., & Nielsen, J. (1989). Spinal mechanisms in man contributing to reciprocal inhibition during voluntary dorsalflexion of the foot. *Journal of Physiology, 416*, 255–272.

Bizzini, M., & Mannion, A. F. (2003). Reliability of a new, hand-held device for assessing skeletal muscle stiffness. *Clinical Biomechanics (Bristol, Avon), 18*(5), 459–461.

Bressel, E., & McNair, P. J. (2002). The effect of prolonged static and cyclic stretching on ankle joint stiffness, torque relaxation, and gait in people with stroke. *Physical Therapy, 82*(9), 880–887.

Ditroilo, M., Watsford, M., Fernández-Peña, E., D'Amen, G., Lucertini, F., & De Vito, G. (2011). Effects of fatigue on muscle stiffness and intermittent sprinting during cycling. *Medicine & Science in Sports & Exercise, 43*(5), 837–45.

Dyhre-Poulsen, P., Simonsen, E., & Voigt, M. (1991). Dynamic control of the muscle stiffness and H reflex modulation during hooping and jumping in man. *Journal of Physiology, 437*, 287–304.

Guissard, N., & Duchateau, J. (2004). Effect of static stretch training on neural and mechanical properties of the human plantar-flexor muscles. *Muscle Nerve, 29*(2), 248–55.

Freriks, B., & Hermens, H. J. (1999). European Recommendations for Surface ElectroMyoGraphy, results of the SENIAM project. In B. a. H. R. P. EU (Ed.), SENIAM - Surface-Electro-MyoGraphy for the Non-Invasive Assessment of Muscles Roessingh Research and Development b.v., (CD-Rom).

Huijing, P. A., van de Langenberg, R. W., Meesters, J. J., & Baan, G. C. (2007) Extramuscular myofascial force transmission also occurs between synergistic muscles and antagonistic muscles. *Journal of Electromyography and Kinesiology, 17*(6), 680–689.

Kovaleski, J. E., Norrell, P. M., Heitman, R. J., Hollis, J. M., & Pearsall, A. W. (2008). Knee and ankle position, anterior drawer laxity, and stiffness of the ankle complex. *Journal of Athletic Training, 43*(3), 242–248. doi:10.4085/1062-6050-43.3.242

Leonard, C. T., Deshner, W. P., Romo, J. W., Suoja, E. S., Fehrer, S. C., & Mikhailenok, E. L. (2003). Myotonometer Intra- and Interrater Reliabilities. *Archives of Physical Medicine and Rehabilitation, 84*(6), 928–932. doi:10.1016/S0003-9993(03)00006-6

Leonard, C. T., Stephens, J. U., & Stroppel, S. L. (2001). Assessing the spastic condition of individuals with upper motoneuron involvement: validity of the myotonometer. *Archives of Physical Medicine and Rehabilitation, 82*(10), 1416–1420. doi:10.1053/apmr.2001.26070

Mannava, S., Wiggins, W. F., Saul, K. R., Stitzel, J. D., Smith, B. P., Koman, L. A., Smith, T. L., & Tuohy, C. J. (2011). Contributions of neural tone to in vivo passive muscle-tendon unit biomechanical properties in a rat rotator cuff animal model. *Annals of Biomedical Engineering, 39*(7), 1914–24.

Masi, A. T., & Hannon, J. C. (2008). Human resting muscle tone (HRMT): Narrative introduction and modern concepts. *Journal of Bodywork and Movement Therapies, 12,* 320–332.

Mukherjee, A., & Chakravarty, A. (2010) Spasticity mechanisms - for the clinician. *Frontiers of Neurology and Neuroscience, 17*(149), 1–10.

Nordez, A., Cornu, C., & McNair, P. (2006). Acute effects of static stretching on passive stiffness of the hamstring muscles calculated using different mathematical models. *Clinical Biomechanics (Bristol, Avon), 21*(7), 755–60.

Ocarino, J. M., Fonseca, S. T., Silva, P. L., Mancini, M. C., & Gonçalves, G. G. (2008). Alterations of stiffness and resting position of the elbow joint following flexors resistance training. *Manual Therapy, 13*(5), 411–8.

Rack, P. M. H., & Westbury, D. R. (1974). The short range elastic stiffness of active mammalian muscle and its effect on mechanical properties. *Journal of Physiology, 240,* 331–350.

Reid, D. A., & McNair, P. J. (2004). Passive force, angle, and stiffness changes after stretching of hamstring muscles. *Medicine & Science in Sports & Exercise, 36*(11), 1944–8.

Rijkelijkhuizen, J. M., Meijer, H. J., Baan, G. C., & Huijing, P. A. (2007). Myofascial force transmission also occurs between antagonistic muscles located within opposite compartments of the rat lower hind limb. *Journal of Electromyography and Kinesiology, 17*(6), 690–7.

Rydahl, S., & Brouwer, B. (2004). Ankle stiffness and tissue compliance in stroke survivors: A validation of Myotonometer measurements. *Archives of Physical Medicine and Rehabilitation, 85*(10), 1631–1637.

Schleip, R., Klinger, W., & Lehmann-Horn, F. (2005). Active fascial contractility: Fascia may be able to contract in a smooth muscle-like manner and thereby influence musculoskeletal dynamics. *Medical Hypotheses, 65*(2), 273–7.

Schleip, R., Naylor, I. L., Ursu, D., Melzer, W., Zorn, A., Wilke, H., Lehmann-Horn, F., & Klinger, W. (2006). Passive muscle stiffness may be influenced by active contractility of intramuscular connective tissue. *Medical Hypotheses, 66*(1), 66–71. doi:10.1016/j.mehy.2005.08.025

Sinkjaer, T., Gantchev, N., & Arendt-Nielsen, L. (1992). Mechanical properties of human ankle extensors after muscle potentiation. *Electroencephalography and Clinical Neurophysiology, 85*(6), 412–8.

Yu, L. C., & Brenner, B. (1989). Structures of actomyosin crossbridges in relaxed and rigor muscle fibers. *Biophysical Journal, 55*(3), 441–53.