

The neuromotor effects of transverse friction massage

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THE NEUROMOTOR EFFECTS OF TRANSVERSE FRICTION MASSAGE

Transverse friction massage (TFM), described by James Cyriax in the 1940s [1], has often been used in chronic inflammatory conditions, promoting local hyperemia, analgesia and the reduction of adherent scar tissue to ligaments, tendons, and muscles. [2] Given its known clinical effectiveness, TFM has not been scrutinized enough in order to find out its effect on the neuromotor driving mechanism. [3] In healthy subjects, this mechanism has recently been redefined with newly established methodology measuring possible time required for excitation-contraction (EC) coupling and force transmission during voluntary muscle contraction.[4,5]

TFM reduces the excitability of the motoneuron pool when tested via Hoffman Reflex, carried out as an electromyographic response to a mild electrical shock to the nerve. [6] The petrissage massage (rhythmic grasping and releasing of the muscle tissue) reduces motor-neuron excitability via muscle spindles and golgi tendon organs as well. [7] The other effect reported is freeing of the mechanoreceptive nerve ending, resulting from the stretching of cutaneous and myofascial tissue, and consequently reducing pain immediately after massage. [8] Basically, a number of studies have agreed on this reduction of neuromotor excitability [6,8,9,10,11], as well as the reduction of the muscle stiffness quantified by ultrasonography [12], when massage was applied over the muscle. Albeit a limited number of studies investigated the effect of application over the tendon, a decrease in Hoffman Reflex was observed, showing the reduction of neuromotor excitability in normal [13] and hemiparetic patients [14] after applied pressure on the Achilles tendon. This reduction is believed to be conveyed by Ia afferents from muscle spindles and it is consequently responsible for reducing the excitability in alpha-motor neurons [7,8] suggesting the involvement of, most likely,

centrally mediated inhibition from higher motor centers. [10] This centrally mediated inhibition has also been suggested to be a possible cause for the observed force reduction at slower muscle contraction velocities after a series of massage applications to the iliotibial band. [15] A decline in the power reduction was also reported after the massage of the gastrocnemius muscle. [16] On the other hand, a comprehensive study has shown that massage reduces neuromotor excitability but without affecting twitch contractile properties when interpreted analysing a peak torque and the time to peak torque parameters. [17]

In order to get some information about the contractile properties of the muscle, EC coupling as the smallest contractile event, was determined by measuring a time delay between the onset of the surface EMG signal and MMG signal. [4,18] Fortunately, increasingly developing ultrasound technology and its reliability in the motion analysis, enabled measurement of the onset of the fiber motion from inside the muscle and possible force transmission through the passive elastic components during voluntary muscle contraction. [5] This success prompted an investigation which would unveil potential changes of the contractile properties and force transmission after TFM applied over the quadriceps femoris tendon. Hence the aim of our study was to find out what kind of effects are produced in the muscle-tendon complex as a result of TFM applied over mechanoreceptor-rich tendon and MTJ. The time delay between the onset of surface EMG and US signal (EC coupling), US and FORCE signal (force transmission along the passive elastic components) and surface EMG and FORCE signal (electromechanical delay, EMD) was computed during voluntary muscle contractions before and after TFM, in the investigation group and resting period in the age-gender matched control group. It was hypothesized that TFM may produce twitch contractile changes (changes in EC coupling) inside the muscle when detected by centrally mediated voluntary muscle contraction.

METHODS:

SUBJECTS

Fourteen healthy male subjects and fifteen age and gender matched control subjects were recruited for the study. [Table 1] They were without any history of previous injury, metabolic or neurologic disease. No one of them was involved in any vigorous exercise on daily basis. The human subject ethical approval was obtained from The Hong Kong Polytechnic University.

Table 1. Physical and anthropometric characteristics of the participants of both massage and control group

	Massage group (n=14)	Control group (n=15)
Age (years)	28.2±3.25	29±4.69
Weight (kg)	71.8±10.16	73.6±11.1
Height (cm)	172.4±5.84	175.6±7.5
BMI	24.1±2.62	23.8±2.72
Mid-sagittal thickness of the RF (mm)	20.4±2.40	12.3±3.95

EXPERIMENTAL DESIGN

Before visiting the laboratory for experimental procedure, subjects participated in a familiarization session to ensure that they could perform properly each step of the experimental procedure without producing muscle fatigue. The dominant leg was chosen for being tested and it was defined as the leg with which the subject preferred to kick a ball. After the anthropometric measurements, subject was seated with a back inclination of 80° and knee was adjusted at flexion angle of 30° below the horizontal plane on a calibrated dynamometer (Humac/Norm Testing and Rehabilitation System, Computer SportsMedicine, Inc.,MA,USA).

90 The 30° of knee flexion was chosen to activate the muscle with minimum pre-stretching of
91 the muscle fibers because increased slack within the muscle-tendon unit (MTU) produced by
92 increasing flexion angles may affect the shortening velocity of the fastest muscle fibers and
93 consequently effect results. [19]

94 Muscle activity during voluntary isometric contractions was recorded simultaneously by
95 sEMG, Torque and ultrafast US while the subject was seated on the calibrated dynamometer
96 (Humac/Norm Testing and Rehabilitation System, Computer Sports Medicine, Inc., MA,
97 USA) with the knee flexion angle adjusted at 30°. [Fig 1A]The test procedure consisted of 4
98 repeated isometric contractions followed by massage/ rest and again 4 repeatedcontractions of
99 the Quadriceps Femoris (QF) muscle. Between each contraction a resting period of 2 minutes
100 was allowed to prevent muscle fatigue. During the test, the subject was asked to apply
101 maximum isometric contraction as quickly as possible in 1 second and to keep it
102 approximately for 3 seconds. Verbal order was given to the subject about the start and
103 termination of the muscle contraction. The order "start" was given immediately after starting
104 the collection of A-mode signals in the ultrafast US device. After the termination of each
105 contraction, the position of the US probe was checked to ensure that there was no
106 displacement of the probe caused by the movement artifact of the muscle during contraction.

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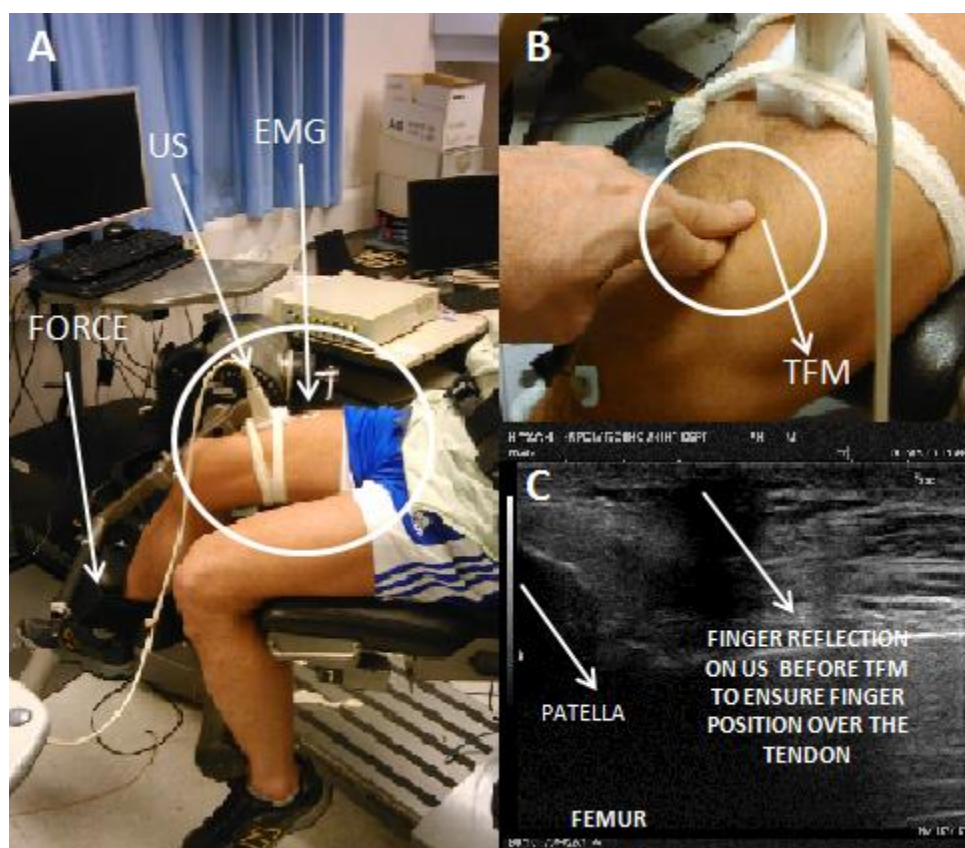


Figure 1. Experimental set-up. A: Simultaneous recording of the Torque, sEMG and Torque while subject is performing an isometric contraction of the Quadriceps Femoris muscle. B: Application of TFM by experienced physiotherapist. C: Position of the fingers above the QF tendon checked on US image before application of TFM.

EMG

Two surface EMG bipolar Ag-AgCl electrodes (Axon System, Inc., NY, USA) for differential EMG detection were attached on the rectus femoris muscle belly, approximately at the 50-60% of the distance between the spina iliaca anterior superior and superior patellar margin. To reduce the skin impedance, skin was cleaned with isopropyl alcohol and abraded with fine sandpaper. The ground electrode was placed over the tibial crest. Interelectrode distance between two surface EMG electrodes was 30 mm. The surface EMG signal was amplified by a custom designed amplifier with a gain of 2000, filtered by 10-1000 Hz bandpass analog filter within the amplifier, and digitized with a sampling rate of 4 KHz.

TORQUE and RATE OF FORCE DEVELOPMENT

A dynamometer (Humac/Norm Testing and Rehabilitation System, Computer Sports Medicine, Inc., MA, USA) with a back inclination of 80° and knee flexion angle of 30° below the horizontal plane was used to measure torque T_{t_1} . The torque signals were digitized with a sampling rate of 4 KHz, and stored on a personal computer. The rate of force development (RFD) was also estimated using the torque:

$$RFD = (T_{t_1} - T_{t_0}) / (t_1 - t_0)$$

where t_0 is the force onset time, and t_1 is the time of reaching 10% maximum force.

The average torque during the period of voluntary isometric contraction was manually identified from the torque wave.

ULTRASOUND

The US recording was made by a custom program installed in a programmable ultrasound scanner (Ultrasonix Touch, Analogic Corporation, Massachusetts, USA) with a 7.5MHz linear array ultrasound probe (Ultrasonix L14-5/35) to achieve a very high frame ultrasound scanning at a selected location. The US probe was placed as close as possible to the surface EMG electrodes, longitudinally to the muscle fibers of the RF muscle. [Fig 1.A,B] The A-mode US signal was collected at a frame rate of 4 k frames/s during 10 s. After the first frame of A-mode signal was collected, a signal was generated by the ultrasound scanner and outputted as an external trigger signal, which was inputted into the device for EMG/FORCE signal collection. The recorded US signal was processed to detect the root mean square (RMS) value of the selected region of interest (ROI). This RMS value obtained from each frame of US signal was then subtracted by the RMS value of the first frame and the result was used to form a new signal representing the US signal disturbance induced by the muscle contraction.

TRANSVERSE FRICTION MASSAGE (TFM)

TFM was applied transversely to the pull line of the quadriceps femoris (QF) muscle tendon by the top of the index and middle fingers. [Fig 1.C] Duration of TFM was about 15 min. The lower leg was detached from the dynamometer lever arm because of uncomfortable feeling of tightness produced by the straps over the lower part of the lower leg. The subject was advised not to place the knee into deep flexion angle during TFM so to avoid stretching effect of the QF muscle and tendon.

DATA ACQUISITION AND ANALYSIS

Collected signals were processed off-line using a program written in MatLab (version 2008a, USA). The EMG signal was rectified and condition of three standard deviations (SDs) from the mean baseline noise was observed to detect the onset of each signal. In order to define crossing time as the onset time, a condition for signal to stay 10 ms above the threshold level was set by the program and visually examined. The time delays between onsets (Δt EMG-US, Δt US-FORCE and Δt EMG-FORCE), TORQUE and RFD were calculated off-line for each contraction. The same experimental procedure was repeated immediately after an applied TFM in massage group and a resting period in control group. [Fig 2]

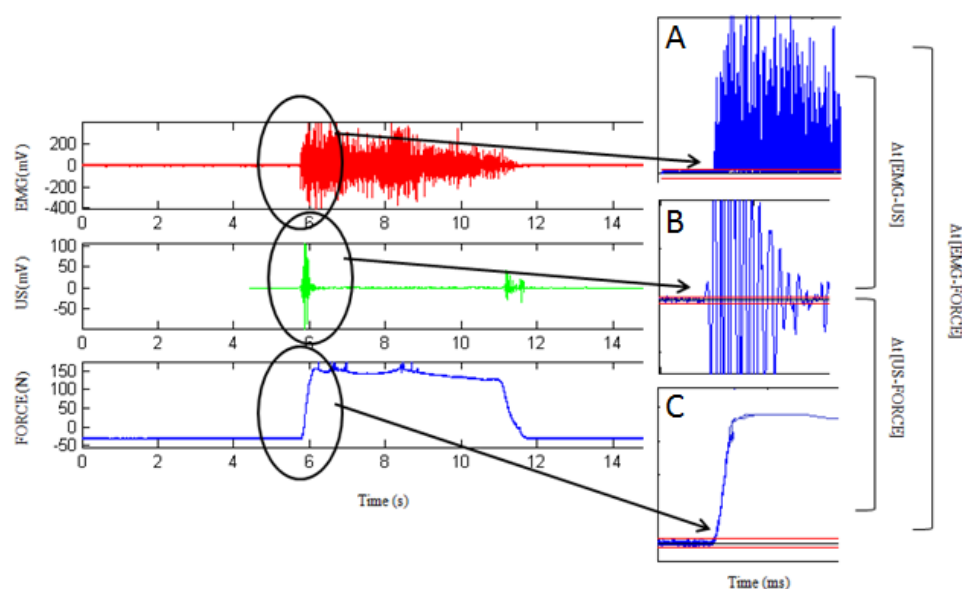


Figure 2. An example of signal processing off-line using a program written in MatLab. Simultaneous recording of sEMG, US and Torque was acquired during isometric contractions before and after TFM. As onsets of each signal were coming out in a sequential order, time delays [Δt] were calculated off-line and statistically analysed. A: automatically rectified sEMG signal in designed MatLab program. B: disturbance signal acquired by ultrafast ultrasound from the selected region of interest. C: torque signal acquired by active isometric contraction of QF muscle.

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177 STATISTICAL ANALYSIS

178 The data were analyzed with a software package SPSS V.19 (IBM SPSS Statistics for
 179 Windows, Version 19.0. Armonk, NY,USA). The normal distribution of the data was
 180 analyzed by the Kolmogorov-Smirnov test. The One-Way Analysis of Variance (ANOVA)
 181 for repeated measures was used to check whether there are any differences between repeated
 182 contractions within the pre- measurements of both TFM and control groups. An average and
 183 SD were calculated if there has not been found any difference between contractions and used
 184 for the comparison between pre-TFM and post-TFM in massage group and between pre-
 185 resting and post-resting in the control group. The paired sample T-test was used for
 186 comparison of the means within groups, whilst, 2-way ANOVA was used for comparisons
 187 between groups.

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192 RESULTS:

193 The results about demographic characteristics of the subjects are demonstrated in the Table 1.
 194 The Kolmogorov-Smirnov test revealed that all data were normally distributed, therefore,
 195 parametric tests were used for the comparisons. One-Way Anova for repeated measures
 196 revealed that there was no difference at all between 4 contractions in all data from the pre and
 197 post tests ($\Delta t_{\text{EMG-US}}$; $\Delta t_{\text{US-FORCE}}$; $\Delta t_{\text{EMG-FORCE}}$, TORQUE and RFD, $p > 0.05$), thus,
 198 averages were calculated and used for comparisons.

199 After TFM, a significant increase in time delays of $\Delta t_{\text{EMG-US}}$ (before: 19.2 ± 9.0 ms; after:
 200 28.5 ± 8.8 ms, $p < 0.01$) and $\Delta t_{\text{EMG-FORCE}}$ (before: 49.7 ± 9.9 ms; after: 54.1 ± 11.8 ms, $p < 0.05$)
 201 was found, whilst the time delay of $\Delta t_{\text{US-FORCE}}$ (before: 30.5 ± 11.9 ms; after: 25.5 ± 11.3 ms,
 202 $p < 0.01$) showed a decrease after TFM. (fig 1)

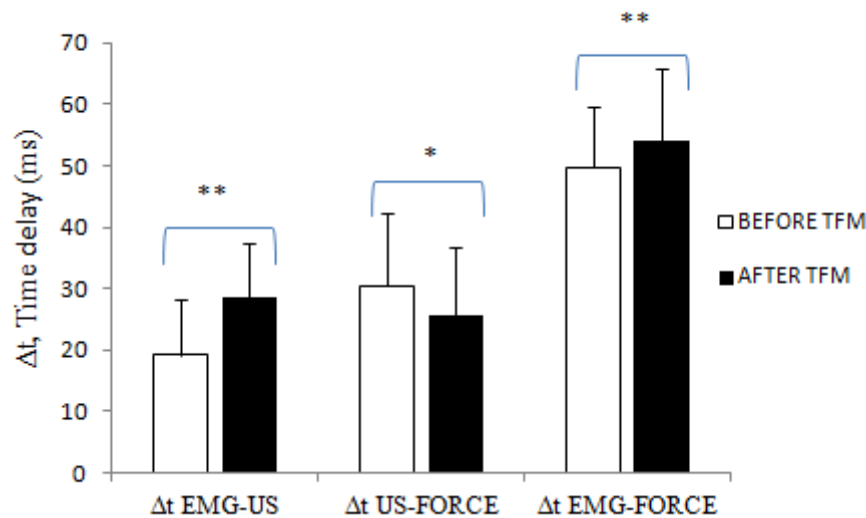


Figure 1 Comparison of the Mean (SD) of 4 isometric contractions before and after application of the transverse friction massage.

In control group, there was not found any significant difference between any average of time delays when compared before and after resting period of 15 min (Δt EMG-US, before: 16.8 ± 6.9 ms; after: 16.6 ± 8.7 ms, $p > 0.05$; Δt US-FORCE, before: 31.2 ± 8.5 ms; after: 30.7 ± 9.3 ms, $p > 0.05$; Δt EMG-FORCE, before: 48.0 ± 8.4 ms; after 47.5 ± 9.9 ms, $p > 0.05$). However, expectedly, there was also not found any difference between pre-measurements of massage and control groups (Δt EMG-US, before: 19.2 ± 9.0 ms; after: 16.8 ± 6.9 ms, $p > 0.05$; Δt US-FORCE, before: 30.5 ± 11.9 ms; after: 31.2 ± 8.5 ms, $p > 0.05$; Δt EMG-FORCE, before: 49.7 ± 9.9 ms; after 48.0 ± 8.4 ms, $p > 0.05$).

Since comparison before and after resting measurements in the control group did not reveal any significant difference, the pre-resting results were compared with after-TFM results and significant increase of time delay was found in Δt EMG-US (control: 16.8 ± 6.8 ms; TFM: 28.5 ± 8.9 ms, $p < 0.01$) and Δt EMG-FORCE (control: 48.0 ± 8.4 ms; TFM: 54.1 ± 11.8 ms, $p < 0.01$) and significant decrease was found in Δt US-FORCE (control: 31.2 ± 8.5 ms; 25.6 ± 11.4 ms, $p < 0.01$) (fig 2).

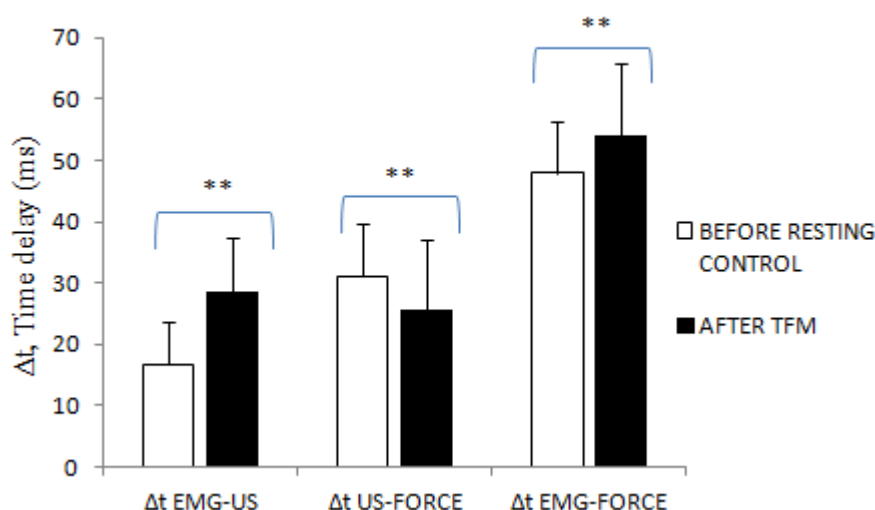


Figure 2 Comparison of the means (SD) between pre-resting measurements of the control and post-massage measurements of the massage group.

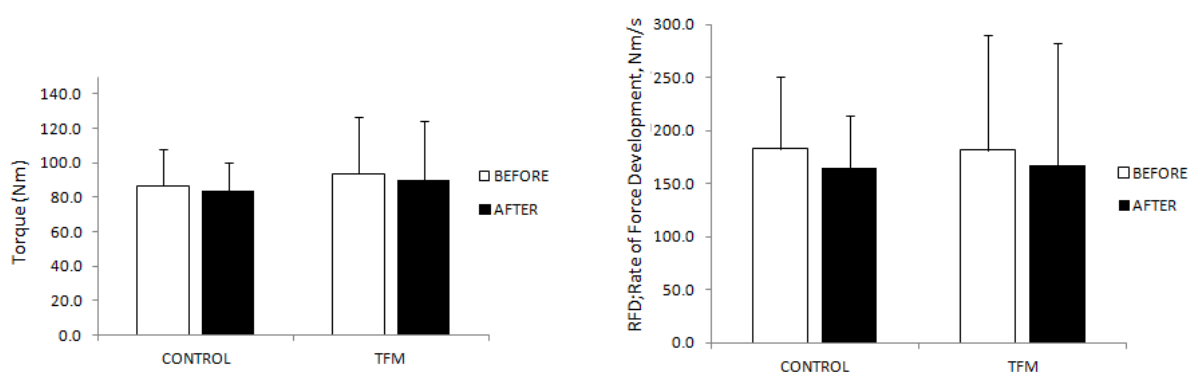


Figure 3 Comparison of the means (SD) torque and rate of force development results between PRE and POST in both control and massage groups.

Comparison of the TORQUE and RFD between pre and post measurements did not reveal any significant difference in both control (Torque, before 86.6 ± 21 Nm; after 83.8 ± 16 Nm; RFD, before 182.6 ± 68.1 Nm/s; after 164.9 ± 49.0 Nm/s) and TFM groups (Torque, before 93.7 ± 33 Nm; after 90.2 ± 34 Nm; RFD, before 181.3 ± 108.8 Nm/s; after 167.7 ± 114.5 Nm/s). (fig 3)

DISCUSSION

The TFM has produced an increase of both the time required for the excitation-contraction coupling [Δt_{EMG-US}] and an overall time delay named as the electromechanical delay [$\Delta t_{EMG-FORCE}$] in both TFM and control groups. A force transmission through non-

contractile elements, when measured as a time delay between the onset of the fiber motion and the onset of the force output [$\Delta t_{US-FORCE}$], has displayed a significant decrease after TFM within TFM group and also when compared with control group.

To justify the effect of manual therapy, such as TFM, prior studies had often been focused on the measurement of a resting EMG activity, muscle inhibition signs and associated it with the effect of manual therapy. This effect has been theorized to occur due to stimulation of the mechanoreceptors or proprioceptors producing a spinal cord mediated effect. The neurophysiological mechanism involved in manual therapy is likely to originate from a peripheral mechanism, spinal cord mechanism and/or supraspinal mechanism but an advanced modification of research methodology was needed to find out peripheral changes within contractile and non-contractile elements. [3,20]

It is likely that TFM, applied predominantly over the tendon and MTJ, increases the EMD between the moment of the action potential generation measured by surface EMG and force generation measured by dynamometer. This time course was named as an EMD and has been used as a reliable measure under different experimental circumstances, when muscle dynamics were matter of investigation. [4,5,21,22,23,24] The EMD has been attributed to the inside changes emerging from the musculotendinous compliance and stiffness [16], where the key role players are contractile (active) and non-contractile (passive) components of the musculotendinous complex. [4,5,23,24] It was reported that subjects who showed the greatest increase in EMD also showed the greatest decrease in musculotendinous stiffness, and vice versa [22] but without detailed investigation of the stiffness changes in contractile and non-contractile elements.

EC coupling is one of the initial events inside the measurement of EMD, where presented time is thought to be the time between action potential generation and cross-bridge formation between contractile components, actin and myosin. [4,23,24] When a muscle is stretched or massaged as a whole anatomic structure without focused application, either over the muscle or tendon, detachment of the cross-bridges happens [4,25] and the time required for the excitation-contraction coupling is expected to be increased. In our study, some measures of precaution have been taken not to massage the muscle tissue itself, relying on a sharp and popping feeling caused by the tendon motion beneath the fingers, and ensuring that massage is applied over the tendon. In this case, without massaging the muscle itself, detected increase of time delay belonging to the EC coupling suggests that TFM applied over the tendon may

indirectly effect the twitch contractile properties, via some afferent pathways. Since EC coupling has been detected using an US signal from inside the muscle, detecting the onset of the muscle tissue motion, we could suggest that increase in both EC coupling and EMD could be the consequence of the reduction in the excitation of α -motor neurons. It is likely that this inhibition is being initiated by stimulated Golgi-tendon organs, which are predominantly located in the MTJ, and lesser amount in the tendon itself [26], causing presynaptic inhibition of the α -motor neurons. It was also established that deep pressure may produce synaptic changes in the brain, so that leads to the reduction of the reflex activity. [8] This may also likely be a mechanism for the increased period of EC coupling because of the involvement of the cortical areas during voluntarily produced muscle contraction in our study.

With indirect evidence, it has been demonstrated that pressure-sensitive and stretch sensitive free nerve ending in muscle tissue, connect to inhibitory neurons, and therefore play a role in reducing motor-neuron pool excitability. [6,27] On the other hand, it has been established that some group III afferent fibers, responsible for mediating mechanoreception, terminate in intramuscular connective tissue and within muscle spindles, which implies that the mechanoreceptors within the muscle might respond to tendon stretch or pressure applied over tendon. [28] Relying on our results detected during centrally mediated voluntary contraction the question is whether increased EC coupling is caused by central mediation involving cortical area or some reflex mechanism inside a muscle.

Since TFM was applied over the tendon, it is expected that mechanical properties (viscoelasticity) of the tendon tissue get affected [29] which consequently leads to a decreased viscoelastic response after TFM. It is well known that tendons are capable of sustaining great tensile loads [30], and they are responsible for the force transmission. As contraction intensity increases, the time needed for the force transmission decreases, as well as an increase in the intramuscular pressure was expected. [31,32] Therefore, given the expectation to find out an increased time belonging to the force transmission, and therefore affected viscoelasticity, we have found a significant decrease, which is contradictory to the known effect of TFM. This decrease also becomes statistically significant when compared with a control group suggesting that TFM applied over a tendon actually may stiffen the tendon material. Therefore, faster force transmission (an increased loading of the tendon) may happen during voluntary muscle contraction. This time was calculated between the onset of the fiber motion detected by US and the onset of the force output. During testing procedure subjects were asked to produce an isometric contraction within the first second which means that tendon

underwent very fast loading. It is known that tendon stiffness increases curvilinearly as the force acting on the tendon increases, resulting in a decreased tendon lengthening for a given force at high force levels.[32] If this is the case, a decreased time required for the force transmission would mean that tendon structures were stiffened by a given high force exertion during voluntary muscle contraction. Eventually, this decrease was found statistically as significant in both TFM and when compared to control group and it could be observed that tendon tissue was not possibly stiffened by TFM. When compared with the control group, this time decrease was significantly decreased even though same testing procedure was carried out and no torque differences between groups were observed.

Very recently, a similar finding was the reason to raise a question, whether the application of transverse force components applied over the muscle could be the cause of an increase of the longitudinal loading of the MTU. [33] Even though in that study results were obtained at the same time while massage was being performed, it is difficult to make an argument because our results were detected several minutes after TFM. This matter should be examined thoroughly through further investigations. A redesigning of the methodology, considering a more selective measurement of the force transmission under different circumstances is essential to highlight the effects of manipulative techniques aiming to reduce the loading of the tendon. Furthermore, as long as resilience of the tendon at the first moment of different contraction intensities is important, the resilience during contraction which is slow and lasts several minutes up to the isometric plateau, would carry very significant clinical implication.

In the present study, contraction intensity was standardized in such a way that subjects were asked to apply maximal voluntary contraction within the first second after the onset of the synchronized recording. The investigation of the relationship of loading (contraction) intensity and muscle-tendon unit behavior showed that increasing intensity caused a decrease in tendon lengthening in humans. [32] The lesser tendon lengthening during high contraction intensities means lesser tendon compliance, faster force transmission, increased rate of force development, and therefore increased loading of the tendon. [34] With increasing rate of loading, the muscle gets stiffer, so shorter display of the EMD is expected. [34] On the other hand, it has also been reported that during a sustained isometric contraction, the intramuscular fluid and acid accumulate, and therefore, intramuscular pressure and muscle volume increase. [35] This finding might be seen as disadvantage when investigating the effect of massage because of prevailing high contraction intensity that may attenuate

effect produced by the massage since these effects are reportedly better observed at lower contraction intensities.[15] In our study, fast contraction within one second should be discussed as a possible methodological limitation. If that is the case, then TFM should be applied carefully before applying it on some sportsman who performs plyometric exercises.

In the present study, there has not been observed any significant change, neither in Torque nor in RFD after TFM. This non-significant change should be reinvestigated, with redesigned methodology, in order to find out the answer whether or not a contraction intensity is important when investigating a massage effect. On the other hand, our results could be quite convincing since TFM, even at high muscle contraction intensities, shows the persistent influence on the neuro-motor driving mechanism via afferent pathways and eventually increases the time within the EC coupling. Since this time has been detected during voluntary muscle contraction, it could be suggested that dynamic stiffness of the muscle is most likely modified by the effected activation patterns of the muscle. According to our results, the decreased muscle stiffness after TFM, mainly preceded by affected EC coupling (increased time delay between EMG and US), could be convincingly supported by our finding of an increased EMD, and eventually decreased active muscle stiffness, detected during active voluntary muscle contractions.