EFFECTS OF 14-WEEK STRENGTH TRAINING PERIOD ON PASSIVE MOVEMENT EVOKED FIELDS, STRETCH REFLEX FUNCTIONING, MUSCLE STRENGTH AND BALANCE

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Autumn 2018
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ABSTRACT

Seipäjärvi, S. 2018. Effects of 14-week strength training period on passive movement evoked fields, stretch reflex functioning, muscle strength and balance. Department of Biology of Physical Activity, University of Jyväskylä, Master's thesis in Biomechanics, 54 pp., 2 appendices.

Strength training is known to affect the nervous system in various ways most often leading to improvements in performance. Changes occurring due to training or aging can provide us more information about the nervous system controlling our movements. Reflexes are an essential part of our body's "toolbox" in movement control and force production. One of particular interest is the functioning of the stretch reflex that is highlighted in some of the motor control theories aswell. Stretch reflex is widely known muscle response to stretch but the question whether a transcortical loop exists and affects the muscle response is still under debate.

The purpose of this study was to find out whether the stretch reflex response measured with magnetoencephalography from the brain and with electromyography from the muscles are affected by strength training. Timing of the cortical and muscle responses were investigated to find out whether transcortical loop could exist. Functional changes due to training were measured with isometric MVCs and dynamic balance test.

A total of 21 subjects were measured from which a total of 12 subjects were included for further analysis and divided into groups based on age: young (n=7) and elderly (n=5).

Results support the timewise possibility of transcortical loop to exist but cannot unambiguously prove it. Responses measured from the muscle during movement initiation suggest changes in the motor control in the working muscles due to strength training. Different age groups showed similar trends in the adaptations highlighting the possibilities of strength training even with greater age.

Keywords: strength training, passive movement evoked fields, stretch reflex, balance

TIIVISTELMÄ

Seipäjärvi, S. 2018. Effects of 14-week strength training period on passive movement evoked fields, stretch reflex functioning, muscle strength and balance. Liikuntatieteellinen tiedekunta, Jyväskylän yliopisto, biomekaniikan pro gradu -tutkielma, 54 s, 2 liitettä.

Voimaharjoittelu vaikuttaa hermostoon monin tavoin. Useimmiten nämä muutokset johtavat myös parannuksiin suorituskyvyssä. Harjoittelun ja esimerkiksi ikääntymisen aiheuttamat muutokset voivat tarjota arvokasta tietoa hermostossa ja liikkeen kontrollissa tapahtuvista muutoksista. Refleksit ovat yksi olennainen osa kehomme keinoista liikkeen kontrolloimiseen ja voiman tuottamiseen. Venytysrefleksi on varsin hyvin tunnettu refleksivaste, joka ilmenee lihaksen aktivaationa lihasta venytettäessä. Venytysrefleksin roolia on korostettu osassa motorisen kontrollin teorioista ja sitä pidetään tärkeänä osana liikkeen kontrollointia. Vaikkakin venytysrefleksin toiminta selkäytimen tasolla tunnetaan hyvin, on edelleen epäselvää vaikuttavatko aivot nähtäviin venytysrefleksivasteisiin kortikaalisen loopin kautta.

Tämän tutkimuksen tarkoituksena oli selvittää vaikuttaako 14 viikon voimaharjoittelujakso venytysrefleksin magnetoenkefalografialla mitattaviin kortikaalisiin vasteisiin tai elektromyografialla mitattaviin lihaksessa esiintyviin vasteisiin. Tarkastelemalla kortikaalisten sekä lihasvasteiden ajoitusta tutkittiin onko kortikaalisen loopin olemassaolo ajallisesti mahdollista. Suorituskyvyn muutoksia mitattiin maksimaalisen isometrisen voimantuoton sekä dynaamisen tasapainotestin avulla.

Yhteensä 21 koehenkilöä osallistui mittauksiin, joista 12 hyväksyttiin tarkempaan analyysiin ja jaettiin ikäryhmiin: nuoret (n=7) ja ikääntyneet (n=5).

Tulokset tukevat kortikaalisen loopin olemassaolon mahdollisuutta ajallisesta näkökulmasta tarkasteltuna mutta eivät todista sen olemassaoloa. Pääsuorittajalihaksista voimantuoton alusta mitatut EMG-vasteet viittaavat siihen, että voimaharjoittelu on muuttanut työskentelevien lihasten motorista kontrollia. Eri ikäryhmien muutostrendit olivat samanlaisia korostaen voimaharjoittelun mahdollisuuksia myös vanhemmalla iällä.

Avainsanat: voimaharjoittelu, kortikaalinen vaste, venytysrefleksi, tasapaino

ABBREVIATIONS

CNS	Central nervous system
COP	Center of pressure
EEG	Electroencephalography
EMG	Electromyography
ERD	Event-related desynchronization
MEP	Motor evoked potential
MEG	Magnetoencephalography
pMEF	Passive movement evoked field
TMS	Transcranial magnetic stimulation

1 INTRODUCTION

Strength training is known to affect both the nervous system and the muscles in a way that can improve our ability to produce force and control movements. The outcomes of training (strength improvements etc.) have been widely studied but the adaptative mechanisms within the body especially in the central nervous system and the sensory receptors are still very much unknown. Possible sites of adaptation include the afferent feedback system meaning the receptors that sense our surroundings and the afferent connections that provide the information for our neuromuscular system to function efficiently. The information can either be "utilized" at the spinal cord level or in the brain, which are both possible sites of adaptation. Finally the top-down control defining the task related operating strategies, the motor commands sent down and the muscles used are a possible sites of adaptation.

The afferent feedback system includes many receptors one of especially interesting in regards of movement: the muscle spindles. Its functioning can be studied for example through the stretch reflex in which the muscle spindles play a vital role. The exact functioning of the stretch reflex in terms of whether it has a transcortical loop affecting the muscle responses remains unknown.

Aging is also major factor affecting the neuromuscular system as a whole for example by reducing the ability to produce force and power, changing the activation of agonist-antagonist muscle pairs and functioning of the motor units. These changes are thought to occur mainly because of sarcopenia and/or reduced physical activity that decrease the quality of muscle.

The purpose of this study was to determine how regular strength training affects the neuromuscular system, especially the afferent feedback to central nervous system and whether aging has effect on the outcomes. In addition we studied balance control to see how training and possible physiological adaptations affect this critical component of motor control that ultimately is one of the determinants of persons functional capacity in daily life.

2 PROPRIOCEPTORS

Proprioceptors account for multiple sensory organs including muscle spindles, Golgi tendon organs, joint capsule receptors and stretch sensitive free endings (Gardner & Johnsson 2013, 480). Proprioceptors provide the brain information about the state of the limbs and the trunk that is essential for control of movements (Latash 2012, 35).

In the current motor control theories (Feldmann 1966; Feldmann 1986; Feldmann et al. 2007; Latash 2010; Scholz & Schöner 1999) the role of the muscle spindles is often highlighted. But a much less studied sensory receptor, that is also crucial for giving information about the state of the whole muscle-tendon complex, is Golgi tendon organ. Kistemaker et al (2013) concluded after modeling the control of the muscle-tendon complex that muscle spindles alone result in poor control of movement. This is largely because of the fact that muscle spindles cannot sense changes in the tendon length and therefore fail to take into account for the whole muscle-tendon complex. (Kistemaker et al 2013.)

Golgi tendon organs are in series with the muscle fibers and located in the area where muscle and tendon/aponeurosis unite. The basic function of the Golgi tendon organ is to give information about the forces in the muscle-tendon complex. When the muscle and the connective tissues are stretched the sensory terminals activated causing excitation to the Ib-afferents. Ib afferents connect to motor neurons through one or two interneurons affecting the motor pool in a more widespread manner. Golgi tendon organs are mainly thought to function in a inhibitory manner reducing the amount of descending output to the homonymous muscles. (Enoka 2008, 254-255.)

Joint receptors form a group of varying sensors that provide more feedback for controlling the movement. Joint receptors vary in location and provide information through II, III and IV afferents of a smaller diameter. (Enoka 2008, 255-256.) Other mechanoreceptors in the cutaneous and subcutaneous area sense touch by sensing the physical deformation of the receptive surface and provide information for somatic sensation (Gardner & Johnsson 2013, 480).

As a whole sensory receptors provide us information about the state of our own body and its relation to the external world. These sensors transform the physical variables

into changes of the membrane potential so that our body can deal with them. (Latash 2012, 35.) This feedback to the brain has and will further be studied with techniques like magnetoencephalography (MEG) that can provide us information of the use of sensory input received by our body (Hari & Forrs 1999).

2.1 STRETCH REFLEX

The stretch reflex was originally reported by Sir Charles Sherrington in 1924 (Liddell & Charles 1924). He found that lengthening of the muscle caused it to contract. The stretch reflex is often considered as an involuntary reflex that is essential in posture control and movement (Shemmell et al. 2010). While others find the classification of voluntary actions and reflexes artificial since all the movements are associated with modulation of reflexes (Latash 2012, 42). The stretch reflex loop is shown in figure 1. In brief the functioning of the stretch reflex can be described as following: lengthening of the muscle causes the muscle spindles to send action potentials through afferent pathways to the spinal cord. In the spinal cord the sensory neurons form both mono- and polysynaptic connections to motor neurons of agonist and antagonist muscles. Muscle spindles form excitatory connections to motor neurons in the spinal cord that enhance the descending output to the agonist muscles and cause inhibition to antagonist muscles through inhibitory interneurons. (Enoka 2008, 251-255.)

Functioning of the stretch reflex loop and the state of the motoneuron pool is often studied with H-reflex which is a basically artificially produced stretch reflex -like muscle response. H-reflex is elicited with electrical stimulus to the afferent nerve. If the electrical stimulus given is strong enough it will cause an action potential in the motoneuron and a muscle response can be measured. (Enoka 2008, 257.) Next chapters describe the anatomy and functioning of the muscle spindles that cause stretch reflex.

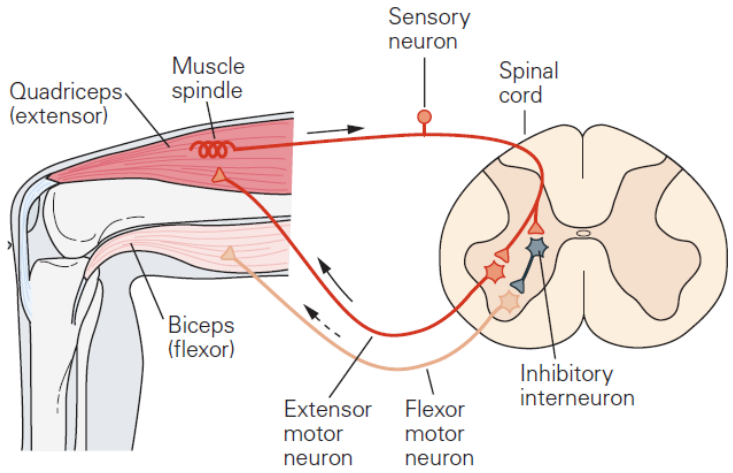

FIGURE 1. When activated through stretching of muscle the muscle spindles send excitatory potentials to motoneurons of the spinal cord through afferent pathways. Muscle spindles form excitatory monosynaptic connections to motoneurons of the homonymous muscle increasing the descending output to the agonist muscle while decreasing the descending output of the antagonist muscle through inhibitory interneurons. (Enoka 2008; Siegelbaum et al. 2013, 212.)

2.1.1 Muscle spindle

Muscle spindles are sensory receptors located in a muscle signaling changes of the muscle length and informing the central nervous system (CNS) about the location of a certain body segment. Muscle spindles are located in parallel with muscle fibers or extrafusal muscle fibers. (Pearson & Gordon 2013, 794.) Muscle spindles (Fig.2) contain intrafusal muscle fibers of different kind that are able to contract and therefore adjust the muscle spindles sensitivity to give feedback (Pierrot-Deseilligny & Burke 2005).

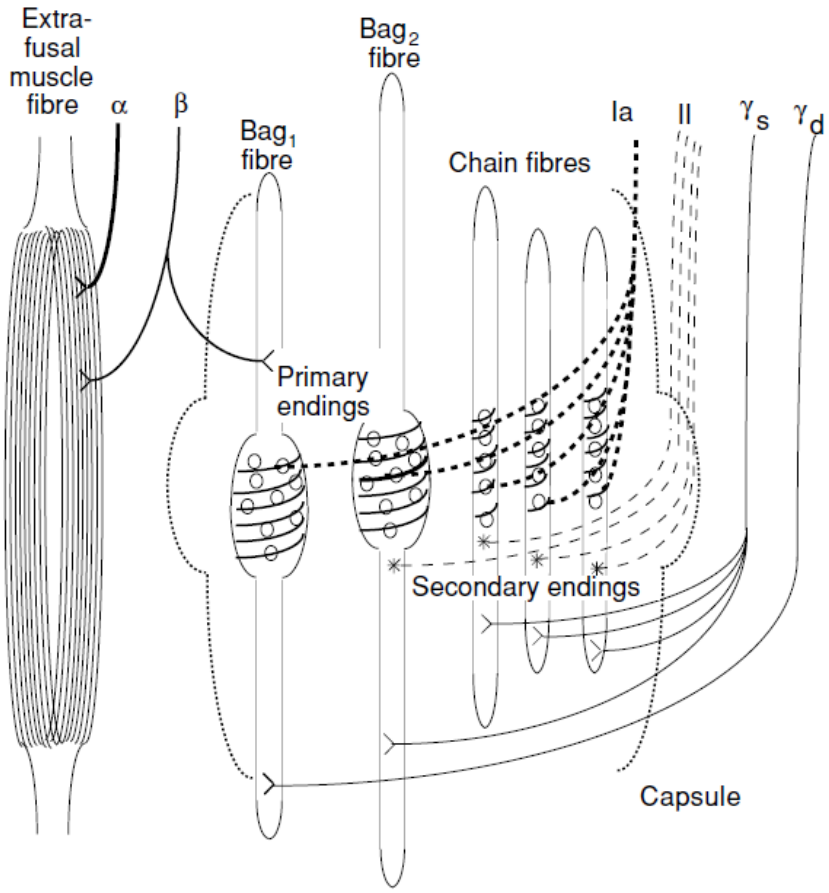

FIGURE 2. Muscle spindle and its afferent fibres Ia and II (Pierrot-Deseilligny & Burke 2005, 115).

Intrafusal muscle fibers. Muscle spindles contain nuclear chain fibers and nuclear bag fibres that have nuclei in the central part of the fiber. There are two types of nuclear bag fibers (bag_1 and bag_2). Primary sensory endings, that are sensitive to both dynamic and static stretch of the muscle, spiral on the central part of the fibers and connect to form a single Ia-afferent to the spinal cord. The bag_2-fibers and nuclear chain fibers also have secondary sensory endings that locate more distally on the fiber. Secondary endings are sensitive mostly on the static stretch and send type II-afferent connections to spinal cord. (Pierrot-Deseilligny & Burke 2005, 114–117.)

Efferent innervation controls sensitivity. Gamma motor neurons send two types of fusimotor efferents that innervate the muscle spindles (Pearson & Gordon 2013, 802). Static fusimotor axons (γ_s) innervate nuclear chain fibers and bag_2 nuclear fibers causing

the primary and secondary endings enhance sensitivity to static stretch. Dynamic fusimotor axons (γ_d) innervate only the bag_1 nuclear fibers and adjust the sensitivity of primary endings to dynamic stretches. (Pierrot-Deseilligny & Burke 2005, 114–117.)

2.1.2 Functioning of the stretch reflex

Ia-afferents arising from the primary endings in the muscle spindle form excitatory monosynaptic connections to the α-motoneurons innervating a homonymous muscle. Group II- afferents arising from secondary endings of the spindle also form weak excitatory monosynaptic connections to α-motoneurons of homonymous muscle and both excitatory and inhibitory connections homonymous and heteronymous muscles α-motoneurons through disynaptic pathways. (Pierrot-Deseilligny & Burke 2005, 114; 289.)

According to Magladery et al. (1951) & Burke et al. (1984) the monosynaptic pathway involving Ia-afferent and homonymous α-motoneuron is considered the main contributor for the short latency component of the stretch reflex (Shemmell et al. 2010).

Pathways contributing to the long latency components are not so clear (Shemmell et al. 2010). Long latency components were thought to be contributions of afferent loops of slower type or fast conducting Ia-afferents carrying information to supraspinal elements and therefore forming a cortical loop. (Hammond 1956 according to Shemmell et al. 2010)

2.2 STRETCH REFLEX MUSCLE RESPONSES

2.2.1 Stretch reflex components

The electrical response that can be measured from the muscle with EMG after an elicited stretch consists of excitatory components (Fig. 3) occurring at different latencies (Hammond 1955 according to Shemmell et al. 2010). The short latency response is thought to be mainly mediated by monosynaptic pathway while the pathways involved in the long latency reflexes involve multiple synapses. Short latency response, usually seen at a onset latency of around 40 ms for plantar flexors (Nardone et al. 1996; Sinkjaer et al. 1996), is also called M1 and the later responses M2 and M3 (Lee and Tatton 1975). M2 usually occurs at a onset latency of around 70 ms (Grey et al. 2001;

Uysal et al. 2009) and M3 around 100 ms (Sinkjaer et al. 1999). It is known that the type of stretch affects the reflex responses. This was shown when stretch reflexes applied in different ways including a tap like, ramp-and-hold and half-sine resulted in different type of activity bursts. (Yavuz et al. 2014.) Also the velocity of the stretch has been shown to affect the reflex amplitude in a way that a faster stretch induces a greater electrical M1 and M2 responses (Spitzer & Claus 1992).

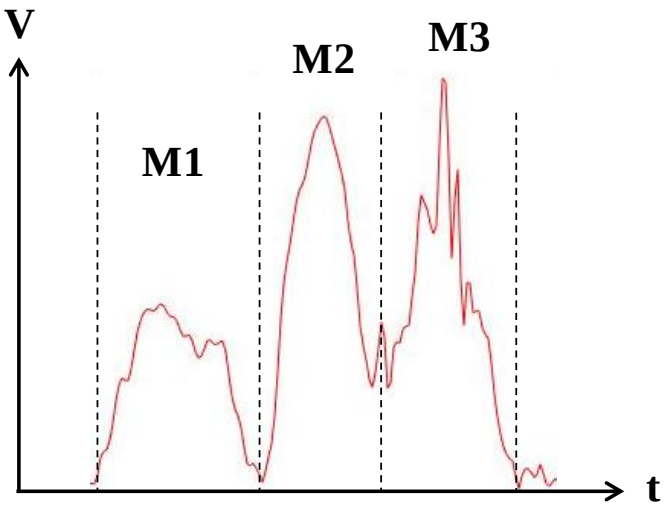

FIGURE 3. Although highly variable and individual the different stretch reflex components or atleast some of them are usually recognizable in the measured EMG signal.

2.2.2 What is known about the M1 response?

The Ia-pathway is thought to be the main contributor in M1 response observed in human studies. It has been shown that M1 response can be altered voluntary and it is no longer thought to behave only in a stereotypical manner. (Shemmell et al. 2010.) This was shown by Cronin et al. (2015) who showed that fascicle stretch is poorly correlated with short latency response. They studied responses from m. triceps surae using different stretch amplitudes and preactivation levels. (Cronin et al. 2015.)

Modulation of the reflex responses occurs presumably through supraspinal control of gamma motor neurons that control the sensitivity of the muscle spindles (Shemmell et al. 2010) or through presynaptic inhibition of the Ia-afferent (Morita et al. 1998). Modulation of the M1 response has been shown to behave task-dependently. Mutha et

al. (2008) showed that changes in a visual task also changed the reflex sensitivity of a performing arm. Reflex responses were measured from muscles with EMG 100 ms after a change in visual task. (Mutha et al. 2008.) Taken together it should be noted that even the functioning of the simplest loop including only one synapse can be modified from task to task.

2.2.3 What is known about the M2 and M3 responses?

Hammond (1956) argued that long latency component of the stretch reflex was due to activation of slow type II-afferent fibres or Ia afferents carrying information through longer reflex pathway involving supraspinal structures (Hammond 1956 according to Shemmell et al. 2010).

Corna et al. (1995) used tizanidine to selectively block group II afferents and found it to cause decrease in M2 responses (Corna et al. 1995). Similar results were found by Grey et al. (2001) when they studied soleus stretch responses in different conditions. Conditions included different stretch velocities and conditions like ischemia that affect the different afferent fibres. They concluded that M2 component was largely contributed by slower group II afferents of the muscle spindle. (Grey et al. 2001.) Gotlieb et al. (1983) used ischemia to block the transmission of Ia-afferent, which also had a decreasing effect of the long-latency response (Gottlieb et al. 1983). Conflicting evidence has been presented by Matthews (1989) who used cooling of the arm to study the afferent pathways and concluded that group II afferents have no major role in long-latency reflex (Matthews 1989).

It has also been hypothesized that afferent feedback resulting from a stretch might synchronize the motoneuron firing. This would occur because multiple motoneurons would fire and hyperpolarize quite simultaneously because of the stretch evoked input and with persistent afferent feedback there could be multiple bursts or components of activity seen. (Schuurmans et al. 2009.)

M2 and M3 responses have been shown to be modulated in a task-dependent way (Lee & Tatton 1975). Mackinnon et al. (2000) results suggested that instruction-dependent modulation of M2 response occurs downstream from inputs to the motor cortex. This conclusion was made after different tasks elicited different M2 responses but no

changes in evoked potentials in the cortex measured with EEG. (Mackinnon et al. 2000.) However, it has also been suggested by Rothwell et al. (1980) that long-latency responses are merely an interaction between longer reflex pathways and fast voluntary responses and modulation is not caused through central pre-setting of nervous systems excitability (Rothwell et al. 1980). This assumption was also supported by Pruszynski et al. (2011) who concluded that long-latency response is a net output of at least two functionally independent processes with overlapping time windows. These processes have similar attributes to short latency reflexes and voluntary control. Manning et al. (2012) also showed that occurance of M2 modulation was highly related to reaction time of voluntary responses in measured muscles. These results strongly suggest the contribution of superimposed voluntary responses to M2 response. (Manning et al. 2012.)

Due to these conflincting views of the pathways involved to the stretch reflex responses it seems likely that the observed response is a combination of multiple perturbation-sensitive pathways and voluntary supraspinal control contributing to the outcome as was suggested by Shemmell et al. (2010).

4 TRANSCORTICAL CONTRIBUTION TO STRETCH REFLEX

According to Hammond (1956), there is a possibility of a transcortical contribution to the long latency component of the stretch reflex. He thought that longer latency of the response might be due to a longer reflex pathway that carried the information with Ia-afferents to supraspinal structures. The long latency response is still a wide term describing the activity occurring after the monosynaptic short latency response. (Hammond 1956 according to Shemmell et al. 2010.)

Cortical responses to stretch reflexes have been studied by Xiang et al. (1997) who measured somatosensory evoked magnetic fields followed by passive movement of the toe. Five main components were identified in the responses recorded from the foot area of the primary sensory cortex. The measured responses are thought to mainly reflect the feedback from muscle and joint afferents. (Xiang et al. 1997.) Similar findings showed that muscle spindle induced neural activity in the brain was related to better balance highlighting the probability of cortical contribution in the stretch reflex (Goble et al. 2011).

The possibility of the cortical loop has been studied by terms of delays of the loop. Petersen et al. (1998) measured stretch-evoked responses from ankle dorsiflexors with needle electrodes and reported cortical potentials at a mean onset latency of 47 ms. MEPs measured from the m. tibialis anterior were evoked at a mean latency of 32 ms. Taken together these results it is possible that the reflex travelling through transcortical pathway affects the later part of M2 and all of the M3 reflex responses. (Petersen et al. 1998) Similar latencies were found by Kurusu & Kitamura (1999) reported a mean delay of 35.3 ± 1.8 ms when they measured somatosensory evoked potentials (SEP) evoked with electrical stimulation of the tibial nerve at the ankle. They also measured latency of efferent pathway to short toe flexor muscles with TMS. They reported mean latency of 42.2 ± 2.0 ms. (Kurusu & Kitamura 1999.) Major component that also affects the latency of the loop is the central processing time in the brain. Central processing time for m. soleus was shown by Kumpulainen et al. (2012) who studied the optimal interstimulus interval for paired associative stimulation and reported the greatest MEP responses to occur when TMS was applied 18 ms after the somatosensory evoked potential occurred (Kumpulainen et al. 2012).

There are multiple studies supporting the existence of a "cortical loop". Firstly, subjects are able to affect on the M2 response of a stretch when given different kind of instructions. This was demonstrated by Crago et al. (1976) when they studied stretch reflex responses of elbow flexors during perturbations in "compensate" or "do not intervene" conditions. Similar results was obtained by Mackinnon et al (2010) who showed that M2 responses were instruction-dependent and EEG showed synaptic activity in the contralateral sensorimotor cortex. Secondly Capaday et al. (1991) observed stretch reflex responses in the thumb muscles of the contralateral limb of the stretched thumb. This observation was made in subjects who had congenital mirror movements. (Capaday et al. 1991)

The existence of a transcortical loop has been studied with TMS. Perenboom et al. (2015) found that subthreshold TMS, when timed to arrive to the muscle 60-90 ms after the stretch, was able to augment EMG responses while no effect on the background EMG was observed. Similar results were shown by Van Doornik et al. (2004) who used subthreshold TMS-stimulation in an active situation to cause inhibition in the motor cortex. They showed a decreased M3 response in the m. tibialis anterior muscle when a stretch reflex was coupled with a TMS-stimulation causing activation of intracortical inhibitory circuits. The suppressing effect was significantly greater in the M3 response occurring at a latency of 98 ± 8 ms than in the background EMG. (Van Doornik et al. 2003.) Several studies have used timed TMS stimulations in order to study the possible contribution of a transcortical loop to the stretch reflex. Palmer & Ashby (1992) and Lewis et al. (2004) found increased corticospinal excitability when MEP was timed to match the latency of a long-latency muscle response of hand muscles. Measured muscle responses were bigger than the sum of a single TMS stimulus and stretch reflex implying a presence of a transcortical loop. (Lewis et al. 2004) Similar results have been obtained by Mrachacz-Kersting et al. (2006) who found that correctly timed TMS stimulus increased the measured muscle’s responses. However, the effect was only seen in M3 response and only in m. rectus femoris and not the other synergist muscles of the thigh. (Mrachacz-Kersting et al. 2006).

Taken together there is evidence suggesting that a transcortical loop could exist but none of those can prove it unambiguously. It has also been proposed that transcortical loop might not exist in all of the muscles (Thilmann et al. 1991).

5 HOW SOMATOSENSORY AFFERENCE AFFECTS THE CORTEX

Movement evoked cortical potentials and fields have been studied over the years to clarify the role of proprioceptive feedback to the brain. Passive movements evoke cortical activity in areas around the vertex that include the primary motor and somatosensory cortex and this activity reflects the proprioceptive feedback received. Since afferent feedback is crucial for producing movement and motor skill acquisition, changes seen in the cortical feedback can tell us something about the adaptations occurring due to training. (Alary et al. 1998; Rothwell et al. 1982; Smeds et al. 2017). Intramuscular receptors, involving muscle afferents and especially the muscle spindles, are known to contribute in the proprioceptive feedback from the periphery to the cortex but the responses are also affected by cutaneous receptors. (Heath et al. 1976; Gandevia et al. 1984: Kristeva-Feige et al. 1996)

Studies have shown cortical rhythms, e.g. mu rhythm, exists in human cortex. Rhytmic oscillation around 10 and 20 Hz can be observed in the rolandic area of relaxed subjects (Salenius et al. 1997). The 10 Hz rhythm is mainly thought to be generated in the somatosensory cortex and 20 Hz rhythm is predominantly generated in the motor cortex (Chen et al. 1999). Aforementioned proprioceptive feedback also affects the cortical rhythms of the cortex which are seen in the transient changes in the amplitude in both of the main frequencies can be seen after voluntary movements and peripheral stimulation (Salenius et al. 1997).

It has been demonstrated that mu rhythms are desynchronized during movement or somatosensory stimuli. This event-related desynchronization (ERD) can be seen in motor and sensory areas related to the task. It is believed that desynchronization is accompanied with cortical activation and increased excitability. The synchronized rhythms are thought to present unactivated, areas that are not processing stimuli etc. The synchronized rhythms or ”idling state” are thought to be related with lower excitability. (Pfurtscheller et al. 1996.) Peripheral stimulation is known to cause periods of increased and decreased cortical excitability in the first 100–200 ms after the stimulation (Chen et al. 1999). Petersen et al. (1998) studied stretch-evoked potentials during tonic contractions and suggested responses to cause increased cortical excitability (Petersen et al. 1998). The responses observed after fixed stimuli can therefore offer us further

information about the functioning of the brain and the afferent system. This was also shown by Goble et al. (2011) who showed that the sensory feedback from periphery seen as cortical activity was linked to better balance.

6 AGING AFFECTS THE NEUROMUSCULAR SYSTEM

Aging is known to affect the neuromuscular systems ability to function in various ways resulting in motor performance impairment. Sarcopenia is a common syndrome with aging people that includes progressive loss of muscle mass and strength (Nascimento et al. 2018). The decrease in muscle mass is often accentuated with the loss of fast-type muscle fibers (Merletti et al. 1992) that also causes the quality of the muscle, meaning the force producing capacity in relation to cross-sectional area, to decrease (Moore et al. 2014). Part of the decrease in ability to produce force might also be due to failure of central activation to voluntarily activate the muscle (Stackhouse et al. 2001).

Aging is thought to affect the activation of agonist-antagonist muscle pairs. This hypothesis is supported by the findings that show the elderly to have a greater leg stiffness (Hortobágyi & DeVita 1999) and Häkkinen et al. (1998) who showed strength training for the elderly to decrease co-activation to same level as younger partipants (Häkkinen et al. 1998). Also changes in the functioning of the motor units in terms of firing rates have been shown occur due to aging including a decrease in average firing rates (Erim et al. 1999) and increased firing rates to produce a certain force (Ling et al. 2017).

Anatomy and functioning of the brain is known to change with age (Kaasinen et al. 2000; Taniwaki et al. 2007) leading to impairments in motor control (Hollman et al. 2007) with the emphasis on the fine motor control (Smith et al. 1999). In daily life impairment in motor control is seen for example in ability to maintain balance (Lauhgton et al. 2003). Postural sway is one way of measuring balance control that has been shown to increase due to aging leading to increased risk of falls (Maki et al. 1994). Impaired balance and motor control might result from changes occurring in the stretch reflex pathways that seem to differ between muscles (Obata et al. 2010; Obata et al. 2012). Kawashima et al. (2004) showed soleus muscle M1 reflex response to occur with shorter latency with elderly than the young in the resting conditions which might have occurred because of reduced elasticity or “slack” of the muscle. Another finding was that the amplitude of the stretch reflex did not change between passive and active state in the muscle with the elderly like it did with younger group. Because no differences were seen in the measured H-reflexes between groups authors suggested that the differences were probably not in the motoneuronal level but the in the level of the

muscle and the muscle spindle. (Kawashima et al. 2004.) In addition to muscle spindles the proprioception as a whole is known to impair with aging (Kaplan et al. 1985).

Even though aging impairs the functioning of the neuromuscular system the changes can be slowed or even reversed with training (Korhonen et al. 2006). Therefore studying the neuromuscular systems of the elderly and the young can provide us further knowledge about the functioning of the whole sensorimotor system.

7 HOW DOES TRAINING AFFECT THE NEUROMUSCULAR SYSTEM AND THE STRETCH REFLEX?

Proprioceptive training has been shown to induce improvements in the functioning of the sensorimotor system. Even though this term includes a variety of methods including the use of vibration, balance training, electrical stimulations etc. that aim to improve motor functioning, the actual mechanisms in the body causing these improvements are not that clear as was concluded by Aman et al. (2015.) Balance is a critical component of motor functioning in our daily lives that has been shown to depend on some extent from muscular strength especially in the elderly (Ferrucci et al. 1997) and it can be improved by training the proprioceptive system (Gauchard et al. 1999) or rate of force development (Piirainen et al. 2014). Whole body vibration training has been shown to improve both the ability to produce strength and proprioception (Trans et al. 2009) and is thought to affect at least partly through stretch reflex modulation (Ritzmann et al. 2013).

Ogawa et al. (2012) studied the differences in stretch reflex response between highly trained endurance runners and control group consisting of regularly active males. Using mechanically evoked stretches to the plantar flexors during rest and tonic contractions of different intensity they measured stretch reflex amplitudes from m. soleus and normalized those to maximal M-wave. Results showed enhanced responses between rest and tonic conditions for both groups but the extent of enhancement was higher in the trained endurance group. Results therefore show training to affect the amplitude of the stretch reflex. (Ogawa et al. 2012.)

Gruber et al. (2007) studied the effects of a 4-week ballistic strength training period on m. soleus stretch reflex responses and found no significant changes while sensorimotor training decreased the stretch reflex amplitude. Sensorimotor training included different kind of balance exercises. They concluded that the two types of training involved different kind of neural adaptations in the spinal level. (Gruber et al. 2007.)

Durbaba et al. (2013) studied the effects of a 4-week high intensity isometric strength training period on stretch reflex induced tremor. Physiological force tremor occurring during isometric contraction is argued to be largely affected by Ia-loop (Christakos et al. 2006; Lippold 1970). Durbaba et al. measured force fluctuations in anisometric

conditions between training and the control group. They found that tremor was equal between groups when using relatively same loads but the amplitude of oscillation was decreased in the trained group when the loads were absolutely the same. Thus suggesting neuronal changes in the stretch reflex loop. (Durbaba et al. 2013.)

Salles et al. 2015 found improved shoulder joint position sense after an 8-week strength training period. Authors suggested that better neuromuscular control of the joint was due to enhanced performance of muscle spindles. (Salles et al. 2015)

Though it is clear that training has positive effects on the functioning of the motor system the underlying mechanisms are unclear. Since proprioception and and the ability to produce force seem to share common elements, studying strength training and proprioception simultaneously could be useful.

8 PURPOSE OF THE STUDY

The purpose of the study is to evaluate the effects of regular strength training on the stretch reflex and its cortical components, and whether there is any difference in the training adaptations between the young and the elderly. Research questions were as the following:

Research question 1. Does training affect the latency of the stretch reflex components? Does the latency of a cortical activity fit the other stretch reflex components to support the existence of a cortical loop?

Hypothesis: Passive movement evoked fields will occur in a time frame possible for cortical loop to exist.

Background theory for hypothesis: Earlier studies have shown strength training to affect neural changes within the stretch reflex arc in the lower levels of neuromuscular system. (Ogawa et al. 2012; Durbaba et al. 2013). Earlier studies done with TMS support the possibility of a cortical loop (Petersen et al. 1998; Perenboom et al. 2015)

Research question 2. Does training affect the afferent feedback to cortex?

Hypothesis: Training will induce changes either in the signal transmission latency or the amplitude of the afferent feedback to the cortex.

Background theory for hypothesis: Earlier studies have shown strength training to affect neural changes within the stretch reflex arc in the spinal level (Ogawa et al. 2012; Durbaba et al. 2013) and also within the cortex (Falvo et al. 2010). Taube et al. (2007) also showed changes in the cortical excitability following a 4-week balance training and concluded those to be related to supraspinal changes. Therefore it seems reasonable to suggest adaptations in the afferent pathways or cortex to occur.

Research question 3. Are there differences in the training adaptations measured from different age groups?

Hypothesis: The adaptations occurring differ in terms of magnitude between the young and the elderly.

Background theory for hypothesis: Aging is known to affect the functioning of the motor system (Mattay et al. 2002) and sensory feedback processing (Goodin et al. 1978; Zumsteg & Wieser 2002). Sarcopenia is common syndrome in the elderly that impairs the functioning of the neuromuscular system (Nascimento et al. 2018) and causes changes in the motor system leading to decreased muscle quality (Moore et al. 2014). Strength training is known to cancel these effects or even to induce improvements as was shown by Korhonen et al. (2006). Aging might also cause differences in the efficiency of training adaptation mechanisms or functioning as was suggested by Lambert & Evans (2002).

9 METHODS

9.1 Subjects

A total of 21 subjects were measured from which a total of 12 subjects were used for the analysis because of the changes in the ankle kinematics from PRE to POST. The elderly group consisted of five subjects (mean age 68.2 years, height 170 ± 7 cm, body mass 73 ± 14 kg, 3 males) and the young group consisted of seven subjects (mean age 24.9 years, height 170 ± 9 cm, body mass 66 ± 5 kg, 2 males). Further analysis was done with the combined group which had all the subjects pooled together. All subjects reported right leg as their preferred kicking leg.

Subjects were pre-screened for any neurological diseases, movement disorders, metal objects in the body and for the level of physical activity. Subjects included in the study were at the age between 18 and 30 years in the young and 65 to 75 years in the elderly group and had a body mass index under 37. Subjects had no prior background of strength training and reported less than three hours of weekly aerobic exercise and no smoking. Subjects reported no serious cardiovascular diseases, use of medication that affected neuromuscular or endocrine system or previous use of testosterone altering treatment. Subjects did not use walking aids and reported no lower limb injuries that may have lead to complications or limitations with training. Written informed consent was obtained from each subject beforehand. The study was done according to the declaration of Helsinki recommendations and it was approved by the ethics committee of the University of Jyväskylä.

9.2 Experimental protocol

Data was collected on two separate occasions PRE and POST (Fig. 4). Stretch reflex measurements were conducted at the University of Jyväskylä's Centre for Interdisciplinary Brain Research (CIBR). Stretch reflex measurements included measures of cortical activity with MEG, foot kinematics with accelerometer and muscle activity with EMG.

Performance measurements were conducted in laboratories at the University of Jyväskylä's Faculty of Sport and Health Sciences. Performance measurements included

measures of maximal bilateral plantar flexor force with EMG and measures of dynamic balance.

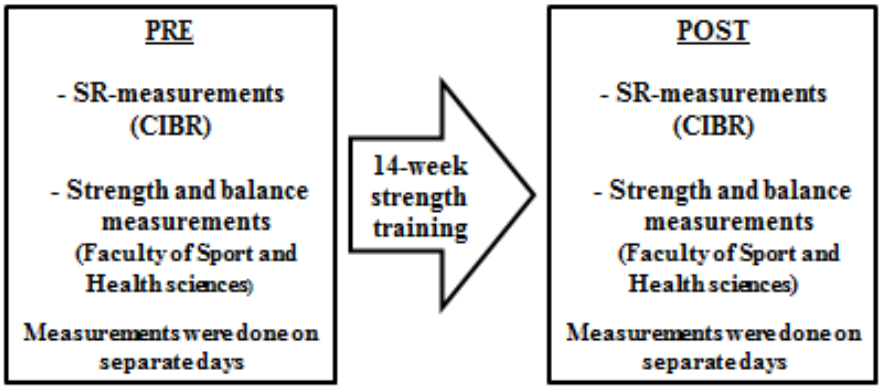

FIGURE 4. Experimental protocol.

9.3 Strength training intervention

Measures for neuromuscular functioning were recorded before and after the 14-week strength training period. The strength training program is presented in appendix 1 and 2. In brief, progressive 12-week heavy resistance training was performed in supervised conditions twice per week. Strength training period was preceded with a 2-week familiarization period involving same exercise movements. Loads used in the primary movements were 40–60% of maximum weights in the familiarization period and 70–95% of maximum weights in the strength training period. Sets with maximal rate of force development during the concentric phase were also included in weeks 10–14.

Calf-exercises were performed in every session during the 12-week strength training period with exercises including sitting calf raises, standing calf raises (Hack-machine, Smith-machine & leg press) and calf jumps. Other than calf jumps the loads used for calf-exercises during the intervention were 60–90% of maximum weights.

9.4 Stretch reflex measurements in MEG

Stretch reflex responses were evoked to ankle plantar flexors with a custom built pneumatic ankle movement actuator (Aalto NeuroImaging and Department of

Neuroscience and biomedical engineering, Aalto University, Espoo, Finland) capable of producing fast movements of approximately 200 deg/s (Fig. 5). The principle of the device is explained in Piitulainen et al. (2015). In brief, ankle actuator used pneumatic artificial muscles that shorten with increasing air pressure. Movement range was around 6.6 degrees. Stretch evoked potentials occurring in the brain were measured with magnetoencephalography (MEG) and with electromyography (EMG).

FIGURE 5. Adjustable pneumatic ankle movement actuator used in the study.

Subjects sat in the MEG device while one leg was being tested at a time (Fig. 6). 60 stretch reflex stimuli with the ankle actuator were evoked approximately in intervals of six seconds with slight variation to reduce the possibility of stimuli anticipation. Visibility of the leg was blocked with paper and earplugs were used with constant brownian noise to block extra auditory stimuli. Subjects were instructed to stay relaxed and keep their vision focused on a round black point. Point (4 cm diameter) was located on a wall in front of them on the opposite side of the leg being stimulated. Stimulated

leg was supported with pillows in order to avoid any extra movement and hands were placed on the table to minimize any additional stimuli.

FIGURE 6. Measuring setup used in the MEG-experiment.

9.4.1 Magnetoencephalography, MEG

Cerebral activity was measured using 306-channel, whole scalp neuromagnetometer (Elekta Neuromag® TRIUX™, Elekta Oy, Helsinki, Finland) in a magnetically shielded room (Magnetical Shielding Cabin, VACOSHIELD, Vacuumschmelze GmbH & Co. KG, Hanau, Germany). The recording passband was 0.1-330 Hz and the signals were sampled at 1 kHz. Position of the subject's head was constantly monitored using four constant head position indicator (cHPI) coils placed on the scalp. The location of the coils in relation to the heads anatomical landmarks was determined with electromagnetic tracker (Fastrak, Polhemus, Colchester, VT, USA). Eye movements were measured with electrooculography (EOG).

9.4.2 Foot kinematics

Foot kinematics were were measured during the stretch reflex measurements done in the MEG. Peak acceleration of the ankle during the movements was measured in order to make sure that the movement stimuli was similar in PRE and POST situations. Acceleration was measured using a MEG-compatible 3-axis accelerometer (sensor: ADXL335 iMEMS Accelerometer, Analog Devices Inc., Norwood, MA, USA.) customized by Helge Kainulainen, Aalto NeuroImaging, Aalto University, Espoo Finland. The accelerometer was attached on the skin above the first metatarsophalangeal joint.

9.4.3 EMG

The electrical activity of the muscles during the stretch reflex protocol was measured with bipolar configuration using Ambu Neuroline electrodes. EMG activity was measured from m. tibialis anterior, m. gastrocnemius and m. soleus. A ground electrode was placed on the clavicle. EMG-activity was measured with simultaneously with MEG (Elekta Neuromag® TRIUX™, Elekta Oy, Helsinki, Finland) using a sample rate of 1KHz.

9.5 Strength and balance measurements

Strength and balance measurements were done in the Faculty of Sport and Health Sciences.

9.5.1 Maximal plantar flexor force

Maximal bilateral isometric plantar flexion strength was measured PRE and POST. Subjects were measured in a seated position by a custom-built plantar flexion device with knees flexed to approximately 90°. The balls of the feet were positioned on a shelf connected to the strain gauge (90° ankle joint-angle) and the knees were held in-place by a cushioned board. Participants performed 3–4 isometric maximal plantar flexion actions.

9.5.2 Dynamic balance test

Dynamic balance was measured using a same device as in the Piirainen et al. (2013) study. Device had a force plate (BT4 balance platform; HUR Labs, Tampere, Finland) placed over a custom-built perturbation machine (University of Jyväskylä). The device in which the the force plate could move backward, forward and sideways to produce perturbations with certain acceleration, amplitude and velocity. The dynamic balance tests were conducted by suddenly moving the force plate, in which the subjects were standing, backwards to produce a stretch for plantar flexors. The peak-to-peak amplitude of displacement of the center-of-pressure (COP) during 500 ms after the plate had stopped was used as a measure of efficiency of the balance performance. Three different velocities were used with a total of 16 perturbations. These included 5 low velocity, 5 medium velocity and 6 high velocity perturbations from which an average displacement of COP was calculated and reported. Table 1 presents the velocitities, accelerations and displacements used in the tests.

TABLE 1. Three different perturbation setups used in the dynamic balance test.

	Perturbation velocities		
	Speed (cm/s)	**Acceleration (m/s2)**	**Displacement (cm)**
Low	8.00	1.70	12.5
Mid	17.00	2.10	12.5
High	24.00	2.90	12.5

9.5.3 EMG

EMG-activity during the bilateral isometric MVCs. Measurements were done with Bipolar Ag/AgCl electrodes (5-mm diameter, 20-mm interelectrode distance, input impedance >100 MOhms, baseline noise < 1 mV rms) that were positioned to the right leg according to SENIAM guidelines. EMG signals were sampled at 2,000 Hz and amplified at a gain of 500 (sampling bandwidth 10–500 Hz). Raw signals

were sent from a hip-mounted pack to a receiving box (Telemyo 2400R; Noraxon, Scottsdale, AZ, USA), then were relayed to an AD converter (Micro1401; Cambridge Electronic Design, Cambridge, United Kingdom) and recorded by Signal 4.04 software Cambridge Electronic Design).

9.6 Data processing

Data processing for each variable is described in the following chapters.

9.6.1 Foot kinematics

Acceleration values were first averaged within subjects with respect to the stimulus. Acceleration peak- and slope-values were manually analyzed. Peak acceleration of the foot during the movements was measured in order to make sure that the movement stimuli was similar in PRE and POST situations.

9.6.2 MEG

MEG data was manually checked to reject noisy channels from the analysis. Data was preprocessed using MaxFilter temporal signal-space-separation (tSSS; Taulu & Simola 2006) with head movement compensation in order to suppress external interferences, to correct for head movements and to transfer the coordinates to average (the average of the PRE- and POST-measurement coordinates) reference head position. The MEG and acceleration signals were band-pass filtered offline at 0.5–195 Hz.

Data was averaged over trials for each subject and then passive movement evoked fields were analyzed from sensorimotor areas in the contralateral side of the stimulated leg. Field potentials were analyzed from a single gradiometer showing greatest evoked responses (pMEF) and also from a peak value of a gradiometer-pair (VectSum).

Stretch reflex induced cortical potential amplitudes were averaged with respect to stimulus onset (-500–1000 ms window). Onset latencies and peak amplitudes were manually analyzed from the pMEFs (Fig. 7). Onset latency was defined as the first clear deviation from signals baseline. Peak value was determined as the highest value within the analyzed window.

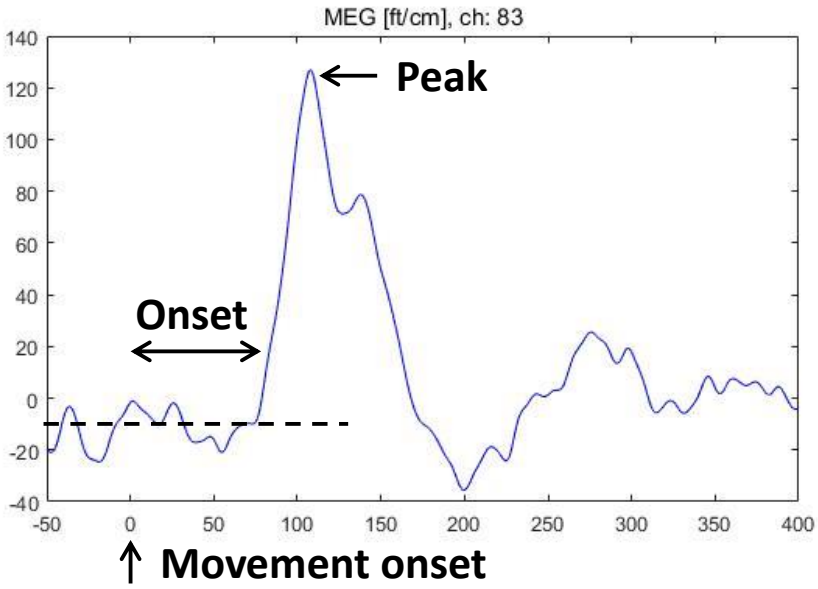

FIGURE 7. Example of the averaged pMEF-response analysis. Also latency within the stimulus system (time from sent stimulus to actual movement onset) was taken into account during the analysis.

9.6.3 EMG

EMG data obtained during stretct reflex protocol was first averaged with respect to the stimulus for every subject. EMG signals were then manually analyzed for stretch reflex component peak latencies (Fig. 8). M3 component latencies were reported only from subjects that showing a clear M3 response. Because of high variability in the shape and size of M3 responses no statiscal analysis was done for stretch reflex M3 component responses.

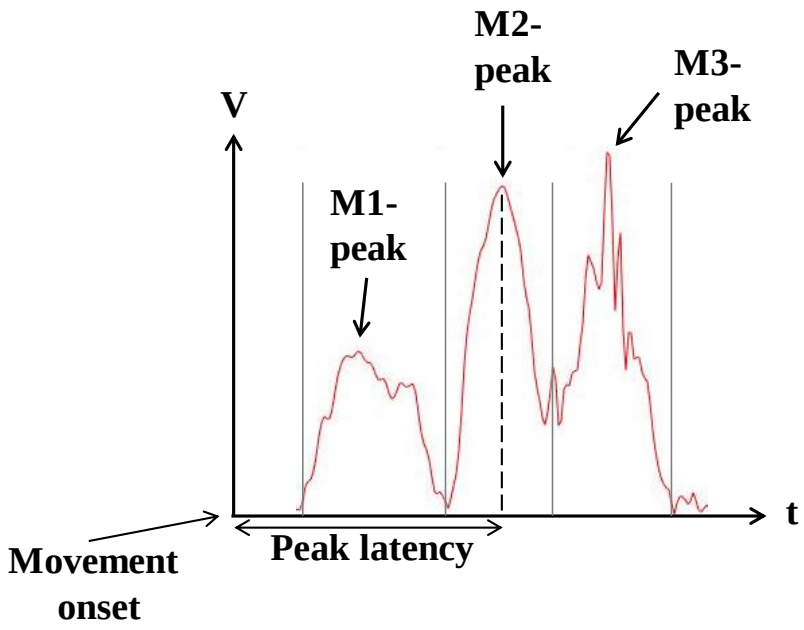

FIGURE 8. Stretch reflex component peak latencies were defined manually as the time point in which greatest EMG-activity was measured.

RMS-amplitude of bilateral plantar flexor MVC was analyzed from the first 100 ms of the contract after EMG signals were band-pass filtered at 20–350 Hz.

9.6.4 Maximal strength

Maximal strength was determined as a single greatest force value obtained from the completed maximal bilateral isometric plantar flexion contractions.

9.6.5 Balance measurements

The peak-to-peak values of single center of pressure displacements were determined from the following 500 ms after the perturbations end. Successful trials were averaged within subjects so that only single average value from all the different perturbations velocities was reported.

9.6.6 Statistical analysis

Statistical analysis was done with IBM SPSS statistics 24 –software. Individual data was first averaged within groups. Non-parametric related samples Wilcoxon signed rank

test was then used to study the differences from PRE to POST within the groups in every variable. Average and standard deviation results were reported from every variable.

10 RESULTS

Results for maximal plantar flexor strength and dynamic balance are presented in section 10.1. Plantar flexor EMG-activity during the isometric is presented in section 10.2.

Ankle acceleration values induced with the ankle actuator are measured during the MEG-protocol are shown in section 9.3. Ankle movement induced somatosensory evoked fields latency and peak values are presented in the section 9.4. At the same time EMG-activity was measured from ankle plantar and dorsiflexors to study the stretch reflex components. The latencies of the measured muscle activity are presented in the section 9.5.

10.1 Strength and balance tests

Results of plantar flexor maximal isometric force measurements are presented in table 2. The young group showed statistically different improvements after the training period while combined and elderly group did not. Dynamic balance was tested in three different velocities by measuring the movements of the center of pressure after perturbations. Both the combined and the young groups had a statistically different score after the intervention. Results are shown in table 3. Both plantar flexor maximal isometric force and balance test results are shown in Fig.9.

TABLE 2. Plantar flexor maximal isometric force.

Group	**Group size, n=**	**PRE (Newton)**	**POST (Newton)**	**Wilcoxon p-value**
Combined	11	1077 ± 230	1139 ± 235	0.091
Young	7	1052 ± 246	1168 ± 266	0.018*
Elderly	4	1121 ± 226	1089 ± 195	0.465

Statistically significant difference *$p < 0.05$

TABLE 3. Center of pressure displacements measured during the balance test.

Group	Group size, n=	PRE (mm)	POST (mm)	Wilcoxon p-value
Combined	11	102 ± 7	95 ± 9	0.026*
Young	7	101 ± 7	91 ± 6	0.018*
Elderly	4	104 ± 7	101 ± 12	0.465

Statistically significant difference *p < 0.05

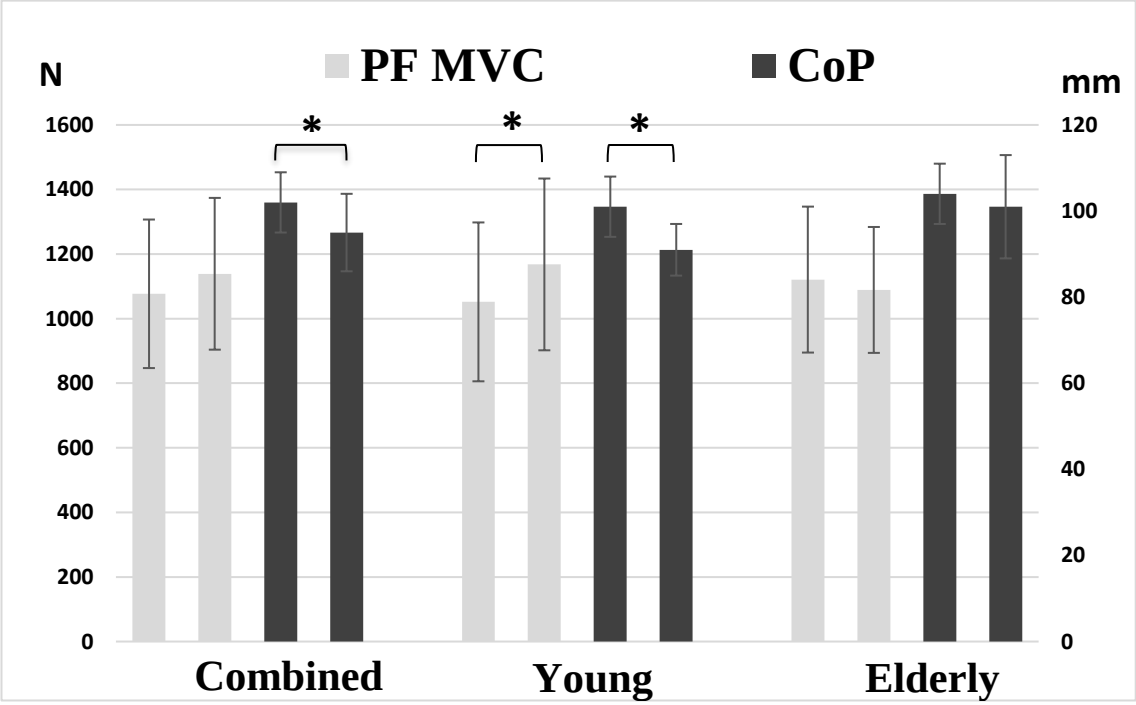

FIGURE 9. Plantar flexor MVC (PF MVC) and center of pressure amplitude (CoP) results presented. Adjacent bars of the same color present PRE-POST values. Statistically significant difference *p < 0.05

10.2 Plantar flexor EMG-activity during 0-100 ms of MVC

Results for m. gastrocnemius (MG), m. soleus (SOL) and for m. tibialis anterior (TIB) are presented in the Fig 10.

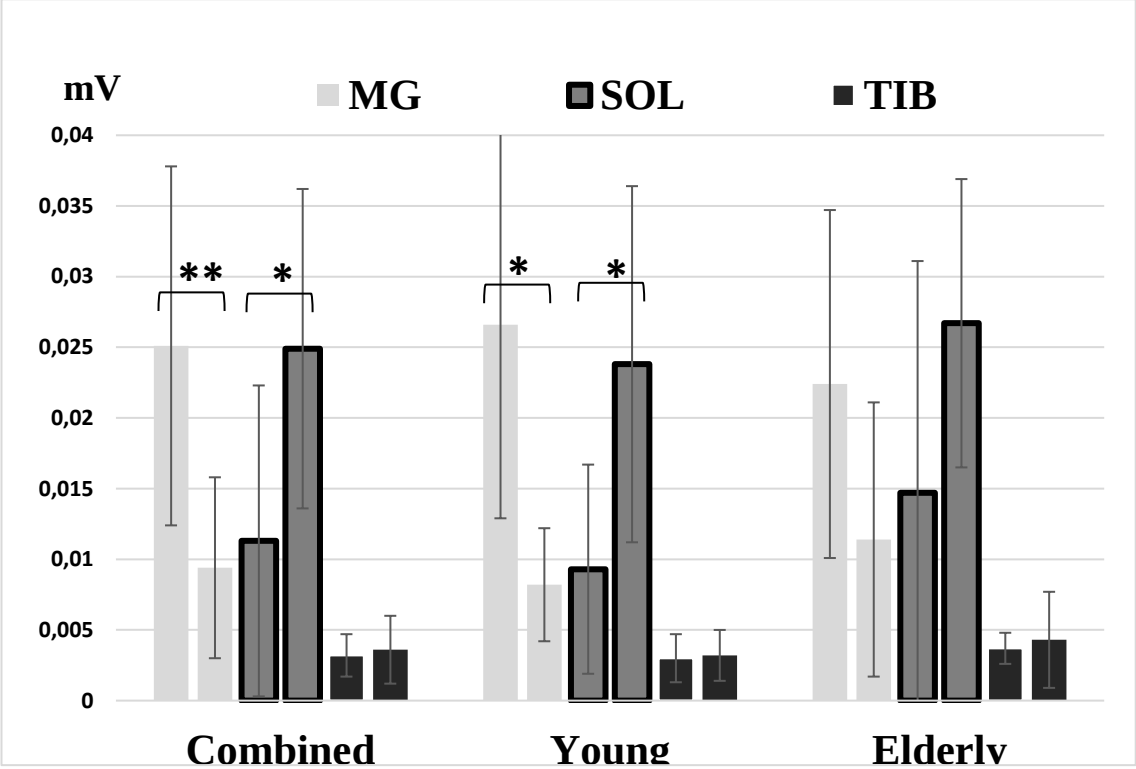

FIGURE 10. EMG-activity measured from m. gastrocnemius (MG), m. soleus (SOL) and m. tibialis anterior (TIB) in different groups. Adjacent bars of the same color are results from PRE (left) and POST (right) measurements.

10.3 Foot kinematics

No statistically significant changes were found between PRE-POST in the group level (table 5).

TABLE 5. Peak acceleration values measured for each group during the SR protocol.

Group	Group size, n=	PRE (G)	POST (G)	Wilcoxon p-value
Combined	12	1.213 ± 0.127	1.177 ± 0.123	0.182
Young	7	1.198 ± 0.158	1.202 ± 0.144	0.735
Elderly	5	1.235 ± 0.077	1.142 ± 0.090	0.08

10.4 Passive movement evoked fields (pMEF)

pMEF onset latencies for each group are presented in table 6. Combined-group had a statistically significant increase in pMEF onset latency due to intervention.

pMEF peak-values from a single gradiometer (table 6) and also from a peak value of a gradiometer-pair (VectSum) are shown below (table 7). All pMEF results are summarized in figure 11.

TABLE 6. Passive movement evoked field onset latency

Group	Group size, n=	PRE (ms)	POST (ms)	Wilcoxon p-value
Combined	12	36.9 ± 7.6	44.8 ± 10.3	0.045*
Young	7	36.6 ± 6.4	44.9 ± 9.4	0.176
Elderly	5	37.4 ± 9.8	44.6 ± 12.6	0.225

Statistically significant difference *$p < 0.05$

TABLE 7. pMEF-values for each group.

Passive movement evoked field peak values

Group	Group size, n=	PRE (fT/cm)	POST (fT/cm)	Wilcoxon p-value
Combined	12	108.0 ± 41.4	107.2 ± 44.3	1
Young	7	107.5 ± 47.9	108.7 ± 52.1	0.5
Elderly	5	108.6 ± 35.5	105.2 ± 36.4	0.735

Passive movement evoked field - VectSum

Group	Group size, n=	PRE (fT/cm)	POST (fT/cm)	Wilcoxon p-value
Combined	12	129.6 ± 48.2	118.8 ± 48.9	0.117
Young	7	131.4 ± 54.8	122.7 ± 55.6	0.398
Elderly	5	127.1 ± 43.3	113.3 ± 43.3	0.08

Statistically significant difference *p < 0.05, **p < 0.01

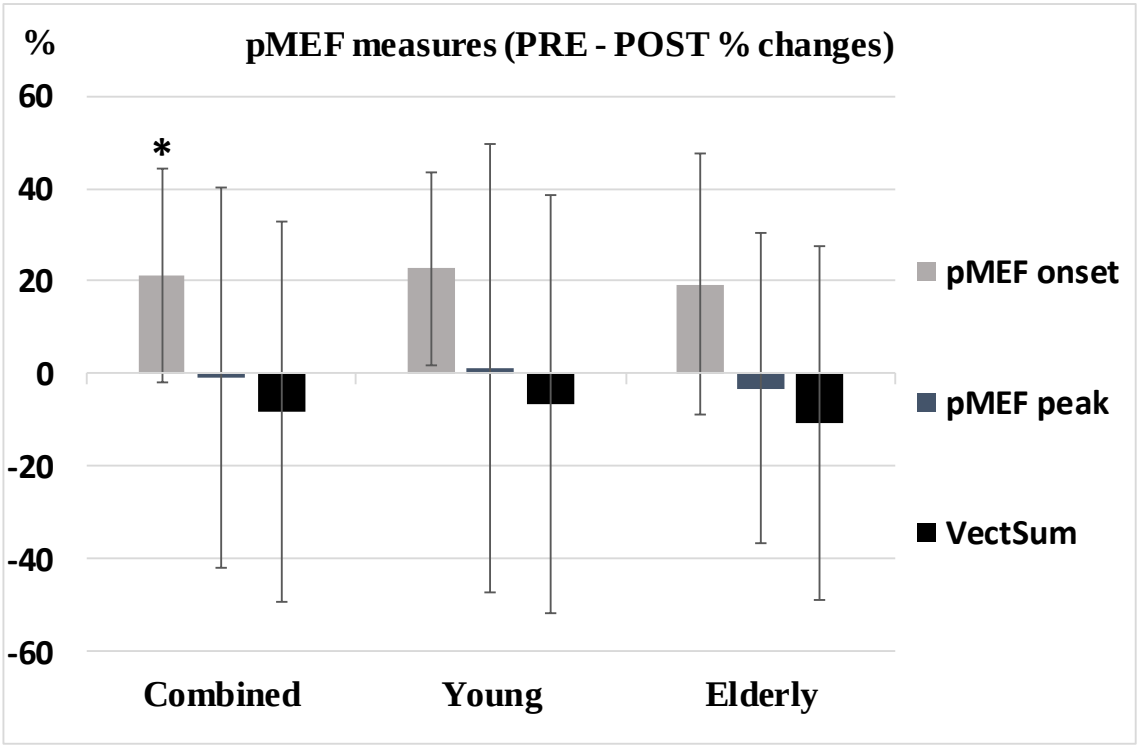

FIGURE 11. pMEF measurements included in the study. Blocks describe percentual changes from PRE to POST.

10.5 EMG stretch reflex component latencies

Below are shown the measured SR component latencies (table 8). No changes were observed measured in M1 component latencies that are shown for m. gastrocnemius and m. soleus.

Combined group had a statistically significant decrease in stretch reflex M2 peak latency in m. gastrocnemius. No other statistically significant changes were observed for other groups in m. gastrocnemius or in m. soleus.

TABLE 8. Stretch reflex component latencies measured from the muscle with EMG.

m. gastrocnemius M1 peak latency

Group	Group size, n=	PRE (ms)	POST (ms)	Wilcoxon p-value
Combined	11	64.9 ± 11.9	60.6 ± 8.9	0.306
Young	7	63.6 ± 12.4	61.9 ± 9.5	0.611
Elderly	4	67.3 ± 12.3	58.5 ± 8.3	0.465

m. soleus M1 peak latency

Group	Group size, n=	PRE (ms)	POST (ms)	Wilcoxon p-value
Combined	12	65.3 ± 9.8	68.6 ± 13.9	0.583
Young	7	62.3 ± 9.5	67.6 ± 9.9	0.31
Elderly	5	69.4 ± 9.6	70.0 ± 19.5	0.893

m. gastrocnemius M2 peak latency

Group	Group size, n=	PRE (ms)	POST (ms)	Wilcoxon p-value
Combined	11	108.7 ± 17.5	96.1 ± 16.5	0.026*
Young	6	107.8 ± 19.9	94.5 ± 15.6	0.116
Elderly	5	109.8 ± 16.5	98 ± 19.2	0.078

m. soleus M2 peak latency

Group	Group size, n=	PRE (ms)	POST (ms)	Wilcoxon p-value
Combined	12	92.2 ± 19.3	110.3 ± 17.4	0.06
Young	7	82.6 ± 14.0	105.7 ± 17.8	0.063
Elderly	5	105.6 ± 18.5	116.8 ± 16.5	0.686

Statistically significant difference *p < 0.05, **p < 0.01

M3 latencies were not observed in all of the subjects or in both PRE and POST. Therefore no further statistical analysis was completed for a small and varying group size. However the average results acquired are presented in the table 9.

TABLE 9. Stretch reflex M3 peak latencies.

	Stretch reflex M3 peak latency			
	m. soleus		**m. gastrocnemius**	
Group	**PRE (ms)**	**POST (ms)**	**PRE (ms)**	**POST (ms)**
Combined	152.9± 23.8	139 ± 22.9	153.3 ± 25.7	145.1 ± 24.6
Elderly	156.6 ± 20.2	169.7 ± 13.9	160 ± 31.1	159.2 ± 19.7
Young	149.8 ± 26.0	125.9 ± 9.6	148 ± 18.6	133.3 ± 21.9

11 DISCUSSION

Present study found strength training to improve isometric maximal force of plantar flexors and the ability to maintain balance in the young while this was not the case with the elderly. This was accompanied with changes in the relative EMG-activity between the working muscles that implying that changes in the motor control in this particular task has occurred. This trend was evident even though not all groups showed statistically significant changes. Changes in the motor control were also supported by decreased m. gastrocnemius M2 peak latency ($p < 0.026$) together with other not statistically significant changes in the SR latencies. Besides the maximal strength different age groups had similar adaptation trends implying that aging does not cancel these adaptations from occurring due to strength training.

Cortical measures showed pMEF onset latency to increase in the combined group and on average the pMEF amplitudes were decreased after training but this finding was not statistically different. Even though in line with previous literature these findings should be interpreted with discretion.

Main findings in the present study are discussed below in more detail.

11.1 Effects of training on strength and balance

Strength training is known to benefit functioning of the neuromuscular system and that strength abilities can be improved no matter age or sex (Ciolac et al. 2010a; Ciolac et al. 2010b) Even though strength training in the elderly has been shown to improve maximal force that was not the case in the present study that included a training period of 14 weeks. Increased the maximal voluntary contraction of the plantar flexors was found in the younger group but the effect was not statiscally significant in the elderly or when both of the groups were pooled together. The range of dynamic repetitions used in plantar flexor movements that varied from 7 to 12 repetitions during the training period, might not have had a great effect on isometric MVC as has been shown by Baker et al. (1994). Although not clear, the young have been shown to have greater adaptations due to strength training (Kosek et al. 2006). It is also possible that in the elderly the probable lower amount of fast type muscle fibers (Nilwik et al. 2013) that are more prone to hypertrophy could diminish the strength improvements (Kosek et al. 2006).

The elderly might also be more cautious and careful while training with heavy weights and therefore not able to reach their full potential in maximal strength.

EMG-activity measurements done during the plantar flexion MVCs first 100 ms offer a glimpse of the strategy the movement is initiated with. Results show that plantar flexors seem to be recruited in a different pattern after the training period. Training induced changes in the muscle activity related to better movement execution have been shown to occur (Darling et al. 1989; Häkkinen et al. 1998). Also Piirainen et al. (2014) showed RFD to have an important role in maintaining balance implying that the initiation of movement/force production is crucial for balance. Both the young and the elderly seemed to have decreased the activity of m. gastrocnemius muscles while increasing the activity of m. soleus muscle after the training period. The changes were statiscally significant in both combined and young groups and the elderly group also had a similar trend towards it. Therefore it seems that strength training has lead to changes of similar type in the movement strategies in all of the groups. Even though EMG-activity was not normalized these results seem justified since the changes in all of the groups share a similar trends as shown in Fig.5.

In the balance tests only the young and the pooled group had improved while the elderly had no significant effect. When concluding with the previous results it could be that for the elderly training twice per week was not enough to induce changes with muscle strength although that was not the case with Krist et al. (2013). It could be that strength training improvements in dynamic movements might not be that apparent in isometric contractions (Baker et al. 1994). Also getting used to training at higher intensity and maximal effort might take more time for the elderly. Previously strength training has been shown to reduce falls in the elderly although no effects have been seen in balance tests involving both static and dynamic tests (Buchner et al. 1997). Joshua et al. (2014) however showed that strength training induced even better improvements on balance than balance training itself. In the present study the lack strength improvements in the elderly might therefore affect the scores in balance as well.

11.2 Effects of training on passive movement evoked fields

In order to study the changes in the nervous system in a longitudinal setup an ankle actuator was implemented into MEG environment. Acceleration magnitudes were

measured in the PRE-POST measurements and were used as a inclusion criteria. Peak acceleration and slope values showed no statistically significant changes in the groups included in the study so examining of the results is meaningful.

From the physiological point of view some changes were observed due to the training period. pMEF onset latency increased ~8 ms the combined-group and similar trends towards longer latencies were present in both of the age groups. Age has been shown to increase latencies of auditory evoked potentials (Goodin et al. 1978) and middle-latency somatosensory evoked potentials (Zumsteg & Wieser 2002). Even though it seems conflicting that training induced changes similar to aging it could be that the increase in latency is somehow beneficial for functioning of the movement system as a whole. One possibility is that movements become more automatized due to training. This could mean lessened role of cortical involvement and increased role of spinal processes in the automated movement compensation.

No statistically significant changes in the pMEF peak values were observed due to training although on average smaller cortical response was seen in the elderly when vector sum of potential fields was measured. Possible explanation for this trend could be that improvents in balance and proprioception lead to smaller cortical activity. Piitulainen et al. (2018) observed higher amounts of proprioceptive processing, indicated by a rise of corticokinematic coherence to be related to poorer balance performance. This proprioceptive processing that reflects higher cortical activity was greater with elderly and with non-dominant leg again supporting the hypothesis that higher cortical activity is related to poorer use of proprioceptive feedback or at least poorer task-spesific performance (Piitulainen et al. 2018a). The trend of decreased cortical activity could therefore caused by training related adaptations. However it should be noted that the elderly group also had a trend of decreased ankle acceleration, although not statistically different, that could have some effect on the change in the measured cortical activation and is in line with previous findings from Spitzer & Claus (1992) who concluded that cortical potentials from thenar muscles were greater when rise-time and amplitude of muscle stretch were increased (Spitzer & Claus 1992).

Earlier Goble et al. (2011) showed that increased brain activity induced by afferent feedback from the muscle spindles and cutaneous receptors was related to better balance performance. Also Taube et al. (2007) reported cortical changes to have occurred after

improved balance due to training. Because present findings did not show any statistical changes in the peak values of pMEFs in any of the groups whether strength or balance had improved or not it could be hypothesized that changes observed in the cortical functioning by Taube et al. (2007) were not related to afferent feedback (down-top) or its immeadiate processing but more likely to top-down mechanisms.

11.3 Effects of training on stretch reflex EMG component latencies

Training did not affect the M1 latencies measured from m. soleus or m. gastrocnemius in any group. However m. gastrocnemius M2 responses occurred with shorter latencies (~13 ms) after the training period in the combined group. This change was mainly accompanied by the changes occurring in the elderly group. M2 responses measured from the m. soleus did not show any statistically significant changes but the opposite trend towards longer latencies was clear. Interestingly this change was more evident in the younger group. Based on the results it seemed that in the m. gastrocnemius changes in the elderly were towards shorter latencies and while the younger group had changes towards longer latencies in the m.soleus muscle.

M2 latencies in m. soleus were on average increasing while M2 latencies in the m. gastrocnemius were shortening. Changes occurring on opposite directions might again reflect changes in the activation patterns of different muscles due to training. The last 6 weeks of training had only calf exercises done in a standing position or jumping which primarily activate the m. gastrocnemius muscle (Signorile et al. 2002). It is known that M2 responses are task dependent and can partly be affected by voluntary actions. However it should be noted that in the present study only latencies were measured instead of amplitudes and the environment was stable in terms of visual or cognitive stimuli. Therefore in this case it seems that the nervous system has reorganized its functioning of m. soleus towards longer latencies to match the demands of the environment or training as was shown too by Häkkinen et al. (1998).

It is possible that the latency changes observed stem from top-down-changes rather than changes in the reflex loops or other more peripheral parts of the nervous system. In other words M2 changes could be affected by fast voluntary responses as was speculated by Rothwell (1980) and Pruszynski et al. (2011).

11.4 Existence of a transcortical loop?

One of the main questions in the present study was whether it was temporally possible that the measured cortical activity induced by the stretch reflex would eventually effect the measured M2 or M3 components? In the present study the onset of the cortical fields were around 40 ms (PRE: 36.9 ms, POST: 44.8 ms). Previous TMS studies could be used to estimate whether M2 or M3 could possibly be affected by a cortical loop. The onset latencies in the corticospinal tract observed in previous studies were 32 ms for m. tibialis anterior (Petersen et al. 1998) and around 30 ms for m. soleus (Sammut et al. 1995; Suga et al. 2001).Studies have shown m. soleus M2 peak-responses to occur at a latency of around 80 ms (Grey et al. 2001; Sinkjaer et al. 1999; Uysal et al. 2009). M3 responses occur later, for m. soleus around 115 ms (Sinkjaer et al. 1999).

Taken together the present findings of pMEF onsets, stretch reflex components and previous studies of the latencies in central processing, ascending and descending tracts it can be concluded that the existence of a cortical loop is indeed timewise possible but could only affect the later part of M2 or M3 response.

11.5 Limitations

Surprisingly big amount of subjects had to be excluded from the analysis because of the changes in the measured foot kinematics components. Multiple possible factors or a combination of those might have caused changes in the measured foot kinematics. Firstly, a change in the air pressure that moves the ankle actuator and finally causes the ankle to move. Secondly a change in the levers because of different positioning of the leg in the ankle actuator could affect the acceleration values since the actuators ability to produce force is limited. Even though measures of the ankle actuator's placement in relation to the MEG and joint-angles from knee and ankle were controlled, the foot's exact center of the pressure in the actuator might have changed leading to minor changes in lever arm length. Changes in the acceleration might also be an outcome of the training through stiffening of the muscle-tendon complex. The peak acceleration could therefore be affected by the force that resists the movement caused by the actuator. Finally the exact location of the accelerometer can also affect the measured acceleration values. Things described above underlie the the need of careful preparation

involved when measuring movements in a MEG environment. In our case most of these problems could have been dealt with better preparation since the reproducibility of the actuator itself is not an issue (Piitulainen et al. 2018b). This fact itself underlines how challenging the MEG environment as a whole can be. Another limitation regarding the results is that the measured EMG-responses were not normalized to M-wave or to MVC EMG-activity and it is should be noted when interpreting the results.

11.6 Conclusions

Findings of the present study are in line with previous studies related to transcortical loop. However findings cannot unambiguously prove the existence of the transcortical loop. Since the timely possibility of a transcortical loop has been shown now by us and previous studies for example by Van Doornik (2014) and Perenboom (2015) future studies should focus on distinguishinging the actual functioning of the transcortical loop. This would mean studying whether a separate cortical loop actually exists or are the previous findings only showing that sensory feedback affects the cortical pyramidal neurons eventually causing alterations in the gain or the excitation of the motoneuron pool as was hypothesized by Rothwell et al. (1980).

Other findings suggesting changes in the muscle recruitment due to training need further clarification but provide interesting hints of the neural mechanisms affected by training. Understanding these mechanisms could enhance the developing of successful training methods for example to improve performance for sports or balance for the elderly. Different age groups showed similar trends in the adaptations highlighting the possibilities of strength training even with greater age.

ACKNOWLEDGMENTS

Thanks to Helge Kainulainen and Aalto Neuroimaging for technical support and collaboration in the project. Also thanks to CIBR staff including Hanna-Maija Lapinkero, Jarno Mikkonen, Simo Monto, Tiina Parviainen and Viki-Veikko Elomaa for all the support given during the process.

12 REFERENCES

Alary, F., Doyon, B., Loubinoux, I., Carel, C., Boulanouar, K., Ranjeva, J.P., Celsis, P. & Chollet, F. 1998. Event-Related Potentials Elicited by Passive Movements in Humans: Characterization, Source Analysis, and Comparison to fMRI. Neuroimage 8, 377–390.

Aman, J.E., Elangovan, N., Yeh, I-L. & Konczak, J. 2015. The effectiveness of proprioceptive training for improving motor function: a systematic review. Frontiers in Human Neuroscience 8, 1075.

Baker, D., Wilson, G. & Carlyon, B. 1994. Generality versus specificity: a comparison of dynamic and isometric measures of strength and speed-strength. European Journal of Applied Physiology and Occupational Physiology 68 (4), 350–355.

Buchner, D.M., Cress, M.E., de Lateur, B.J., Esselman, P.C., Margherita, A.J., Price, R. & Wagner, E.H. 1997. The effect of strength and endurance training on gait, balance, fall risk, and health services use in community-living older adults. The Journals of Gerontology 52 (4), 218–224.

Capaday, C., Forget, R., Fraser, R. & Lamarre, Y. 1991. Evidence for a contribution of the motor cortex to the long-latency stretch reflex of the human thumb. Journal of Physiology 440, 243–255.

Chen, R, Corwell, B. & Hallett, M. 1999. Modulation of motor cortex excitability by median nerve and digit stimulation. Experimental Brain Research 129, 77–86.

Christakos, C.N., Papadimitrou, N.A. & Erimaki, S. 2006. Parallel neuronal mechanisms underlying physiological force tremor in steady muscle contractions of humans. Journal of Neurophysiology 95, 53–66.

Ciolac, E.G., Garcez-Leme, L.E., Greve, J.M. 2010a. Resistance exercise intensity progression in older men. International Journal of Sports Medicine 31 (6), 433–438.

Ciolac, E.G., Brech, G.C. & Greve, J.M. 2010b. Age does not affect exercise intensity progression among women. Journal of Strength and Conditioning Research 24 (11), 3023–3031.

Corna, S., Grasso, M., Nardone, A. & Schieppati, M. 1995. Selective depression of medium-latency leg and foot muscle responses to stretch by an α_2-agonist in humans. Journal of Physiology 484 (3), 803–809.

Crago. P.E., Houk, J.C. & Hasan, Z. 1976. Regulatory actions of human stretch reflex. Journal of Neurophysiology Vol. 39 no. 5, 925–935.

Cronin, N.J., Rantalainen, T. & Avela, J. 2015. Triceps surae fascicle stretch is poorly correlated with short latency stretch reflex size. Muscle Nerve 52, 245–251.

Darling, W.G., Cooke, J.D. & Brown, S.H. 1989. Control of simple arm movements in elderly humans. Neurobiology of Aging 10 (2), 149–157.

Durbaba, R., Cassidy, A., Budini, F. & Macaluso, A. 2013. The effects of isometric resistance training on stretch reflex induced tremor in the knee extensor muscles. Journal of Applied Physiology 114, 1647–1656.

Enoka, R.M. 2008. Neuromechanics of human movement. Fourth edition. Champaign, IL: Human Kinetics.

Erim, Z., Beg, M.F., Burke, D.T., de Luca, C.J. 1999. Effects of Aging on Motor-Unit Control Properties. Journal of Neurophysiology 82 (5), 2081–2091.

Falvo, M.J., Sirevaag, E.J., Rohrbaugh, J.W. & Earhart, G.M. 2010. Resistance training induces supraspinal adaptations: evidence from movement-related cortical potentials. European Journal of Applied Physiology 109, 923–933.

Feldman, A.G. 1966. Functional tuning of the nervous system with control of movement or maintenance of a steady posture. II. Controllable parameters of the muscle. Biophysics 11, 565–578.

Feldman, A.G. 1986. Once more on the equilibrium-point hypothesis (λ-model) for motor control. Journal of Motor Behavior 18, 17–54.

Feldman, A.G., Goussev, V., Sangole, A., Levin, M.F. 2007. Threshold position control and the principle of minimal interaction in motor actions. Progress in Brain Research 165, 267–281.

Ferrucci, L., Guralnik, J.M., Buchner, D., Kasper, J., Lamb, S.E., Simonsick, E.M., Corti, M.C., Bandeen-Roche, K. & Fried, L.P. 1997. Departures from linearity in the relationship between measures of muscular strength and physical performance of the lower extremities: the Women's Health and Aging Study. The Journals of Gerontology. Series A, Biological Sciences and Medical Sciences 52 (5), 275–285.

Gandevia, S.C., Burke, D .& Mckeon, B.1984. The projection of muscle afferents from the hand to the cerebral cortex in man. Brain 107, 1–13.

Gardner, E.P. & Johnsson, K.O. 2013. The somatosensory system: Receptors and central pathways. In a writing of Kandel, E.R., Schwartz, J.H., Jessell, T.M., Siegelbaum, S.A. & Hudspeth, A.J. (publ.) Principles of neural science. 5th edition. McGraw-Hill-companies.

Gauchard, G.C., Jeandel, C., Tessier, A. & Perrin, P.P. 1999. Beneficial effect of proprioceptive physical activities on balance control in elderly human subjects. Neuroscience Letters 273 (2), 81–84.

Goble, D.J., Coxon, J.P., Van Impe, A., Geurts, M., Doumas, M., Wenderoth, N. & Swinnen, S.P. Brain Activity during Ankle Proprioceptive Stimulation Predicts Balance Performance in Young and Older Adults. Journal of Neuroscience 31 (45), 16344–16352.

Goodin, D.S., Squires, K.C., Henderson, B.H. & Starr, A. 1978. Age-related variations in evoked potentials to auditory stimuli in normal human subjects. Clinical Neurophysiology, vol 44 (4), 447–458.

Gottlieb, G.L., Agarwal, G.C. & Jaeger, R.J. 1983. Response to sudden torques about ankle in man: V effects of peripheral ischemia. Journal of Neurophysiology 50 (1), 297–312.

Grey, M.J., Ladouceour, M., Andersen, J.B., Bo Nielsen, J. & Sinkjaer, T. 2001. Group II muscle afferents probably contribute to the medium latency soleus stretch reflex during walking in humans. Journal of Physiology 534 (3), 925–933.

Gruber, M., Taube, W., Gollhofer, A., Beck, S., Amtage, S. & Schubert, M. 2007. Training spesific adaptations of H- and stretch reflexes in human soleus muscle. Journal of Motor Behavior 39, 68–78.

Hari, R. & Forss, N. 1999. Magnetoencephalography in the study of human somatosensory cortical processing. Philosophical Transactions of the Royal Society B: Biological Sciences 354, 1145–1154.

Heath, C.J., Hore, J., Phillips, C.G. 1976. Inputs from low threshold muscle and cutaneous afferents of hand and forearm to areas 3a and 3b of baboon's cerebral cortex. The Journal of Physiology 257, 199–227.

Hollman, J.H., Kovash, F.M., Kubik, J.J., Linbo, R.A. 2007. Age-related differences in spatiotemporal markers of gait stability during dual task walking. Gait & Posture 26 (1), 113–119.

Hortobágyi, T. & DeVita, P. 1999. Muscle pre- and coactivity during downward stepping are associated with leg stiffness in aging. Journal of Electromyography and Kinesiology 10, 117–126.

Häkkinen, K., Kallinen, M., Izquierdo, M., Jokelainen, K., Lassila, H., Mälkiä, E., Kraemer, W.J., Newton, R.U. & Alen, M. 1998. Changes in agonist-antagonist EMG, muscle CSA, and force during strength training in middle-aged and older people. Journal of Applied Physiology 84 (4), 1341–1349.

Joshua, A.M., D'Souza, V., Unnikrishnan, B., Mithra, P., Kamath, A., Acharya, V. & Venugopal, A. 2014. Effectiveness of Progressive Resistance Strength Training Versus Traditional Balance Exercise in Improving Balance Among the Elderly - A Randomised Controlled Trial. Journal of Clinical and Diagnostic Research 8 (3), 98–102.

Kaasinen, V., Vilkman, H., Hietala, J., Nagren, K., Helenius, H., Olsson, H., Farde, J., 2000. Age-related dopamine D2/D3 receptor loss in extrastriatal regions of the human brain. Neurobiology of Aging 21, 683–688.

Kamen, G. & Knight, C.A. 2004. Training-related adaptations in motor unit discharge rate in young and older adults. The Journals of Gerontology: Series A 59 (12), 1334–1338.

Kaplan, F.S., Nixon, J.E., Reitz, M. Rindfleish, L. & Tucker, J. 1985. Age-related changes in proprioception and sensation of joint position. Acta Orthopaedical Scandinavica 56 (1), 72–74.

Kawashima, N., Nakazawa, N., Yamamoto, S.I., Nozaki, D., Akai, M. & Yano, H. 2004. Stretch reflex excitability of the anti-gravity ankle extensor muscle in elderly humans. Acta Physiologica 180, 99–105.

Kistemaker, D.A., Knoek Van Soest, A.J., Wong, J.D., Kurtzer, I. & Gribble, P.L. 2013. Control of position and movement is simplified by combined muscle and Golgi tendon organ feedback. Journal of Neurophysiology 109, 1126–1139.

Korhonen, MT., Christea, A., Alen, M., Häkkinen, K., Sipilä, S., Mero, A., Viitasalo, JT., Larsson, L. & Suominen, H. 2006. Aging, muscle fiber type, and contractile function in sprint-trained athletes. Journal of Applied Physiology 101 (3), 906–917.

Krist, L., Dimeo, F. & Keil, T. 2013. Can progressive resistance training twice a week improve mobility, muscle strength, and quality of life in very elderly nursing-home residents with impaired mobility? A pilot study. Clinical Interventions in Aging 8, 443–448.

Kumpulainen, S., Mrachacz-Kersting, N., Peltonen, J., Voigt, M. & Avela, J. 2012. The optimal interstimulus interval and repeatability of paired associative stimulation when the soleus muscle is targeted. Experimental Brain Research 221 (3), 241–249.

Kurusu, K. & Kitamura, J. 1999. Long-latency reflexes in contracted hand and foot muscles and their relations to somatosensory evoked potentials and transcranial magnetic stimulation of the motor cortex. Clinical Neurophysiology 110, 2014–2019.

Lambert, C.P. & Evans, W.J. 2002. Effects of aging and resistance exercise on determinants of muscle strength. Journal of the American Aging Association 25 (2), 73–78.

Latash, M. 2010. Motor synergies and equilibrium-point hypothesis. Motor Control 14 (3), 294–332.

Latash, M. 2012. Fundamentals of motor control. First edition.

Lee, R.G. & Tatton, W.G. 1975. Motor responses to sudden limb displacements in primates with specific CNS lesions and in human patients with motor system disorders. The Canadian Journal of Neurological Sciences 2 (3), 285–293.

Lewis, G.N., Polych, M.A. & Byblow, W.A. 2004. Proposed cortical and sub-cortical contributions to the long-latency stretch reflex in the forearm. Experimental Brain Research 156, 72–79.

Liddell, E.G.T. & Sherrington, C. 1924. Reflexes in response to stretch (myotatic reflexes). Proceedings of the royal society of London: Biological Sciences 96, 212–242.

Ling, S.M., Conwit, R.A., Ferrucci, L. & Metter, E.J. 2017. Age-Associated Changes in Motor Unit Physiology: Observations From the Baltimore Longitudinal Study of Aging. Archives of Physical Medicine and Rehabilitation 90 (7), 1237–1240.

Lippold, O.C. 1970. Oscillation in the stretch reflex arc and the origin of the rhythmical, 8-12 C-S component of physiological tremor. The Journal of Physiology 206 (2), 359–382.

Mackinnon, C.D., Verrier, M.C. & Tatton, W.G. 2000. Motor cortical potentials precede long-latency EMG activity evoked by imposed displacements of the human wrist. Experimental Brain Research 131, 477–490.

Maki, B.E., Holliday, P.J. & Topper, A.K. 1994. A prospective study of postural balance and risk of falling in an ambulatory and independent elderly population. Journal of Gerontology 49 (2), 72–84.

Manning, C.D., Tolhurst, S.A. & Bawa, P. 2012. Proprioceptive reaction times and long-latency reflexes in humans. Experimental Brain Research 221, 155–166.

Mattay, V.S., Fera, F., Tessitore, A., Hariri, A.R., Das, S., Callicott, J.H. & Weinberger, D.R. 2002. Neurophysiological correlates of age-related changes in human motor function. Neurology 58(4): 630–635.

Matthews, P.B. 1989. Long-latency stretch reflexes of two intrinsic muscles of the human hand analysed by cooling the arm. Journal of Physiology 419, 519–538.

Merletti, R., Lo Conte, L.R., Cisari, C. & Actis, M.V. 1992. Age related changes in surface myoelectric signals. Scandinavian Journal of Rehabilitation Medicine 24 (1), 25–36.

Moore, A.Z., Caturegli, G., Metter, E.J., Makrogiannis, S., Resnick, S.M. Harris, T.B. & Ferrucci, L. 2014. Difference in muscle quality over the adult life span and biological correlates in the Baltimore Longitudinal Study of Aging. Journal of American Geriatrics Society 62 (2), 230–236.

Morita, H., Petersen, N., Christen, L.O., Sinkjaer, T. & Nielsen, J. 1998. Sensitivity of H-reflexes and stretch reflexes to presynaptic inhibition in humans. Journal of Neurophysiology 80 (2), 610–620.

Mrachacz-Kersting, N., Grey, M.J. & Sinkjær, T. 2006. Evidence for a supraspinal contribution to the human quadriceps long-latency stretch response. Experimental Brain Research 168, 529–540.

Mutha, P.K., Boulinguez, P. & Sainburg, R.L. 2008. Visual modulation of proprioceptive reflexes during movement. Brain Research 1246, 54–69.

Nardone A, Grasso M, Giordano A, Schieppati M. 1996. Different effect of height on latency of leg and foot short- and medium- latency EMG responses to perturbation of stance in humans. Neuroscience Letters 206, 89-92.

Nascimento, C.M., Ingles, M., Salvador-Pascual, A., Cominetti, M.R., Gomez-Cabrera, M.C. & Viña, J. 2018. Sarcopenia, frailty and their prevention by exercise. Free Radical Biology and Medicine, in press.

Nilwik, R., Snijders, T., Leenders, M., Groen, B.B.L., van Kranenburg, J., Verdijk, L.B. & van Loon, L.J.C. 2013. The decline in skeletal muscle mass with aging is mainly attributed to a reduction in type II muscle fiber size. Experimental Gerontology 48 (5), 492–498.

Obata, H., Kawashima, N., Akai, N., Nakazawa, K. & Ohtsuki, T. 2010. Age-related changes of the stretch reflex excitability in human ankle muscles. Journal of Electromyography and Kinesiology 20 (1), 55–60.

Obata, H., Kawashima, N., Akai, N., Ohtsuki, T. & Nakazawa, K. 2012. Aging effects on posture-related modulation of stretch reflex excitability in the ankle muscles in humans. Journal of Electromyography and Kinesiology 22 (1), 31–36.

Ogawa, T., Kawashima, N., Suzuki, S. & Nakazawa, K. 2012. Different modulation pattern of spinal stretch reflex excitability in highly trained endurance athletes. European Journal of Applied Physiology 112, 364–3648.

Palmer, E. & Ashby, P. 1992. Evidence that a long latency stretch reflex in humans is transcortical. Journal of Physiology 449, 429–440.

Perenboom, M.J.L, Van de Ruit, M., De Groot,J.H., Schouten,A.C. & Meskers, C.G.M. 2015. Evidence for sustained cortical involvement in peripheral stretch reflex during the full long latency reflex period. Neuroscience Letters 584, 214–218.

Petersen, N., Christensen, L.O.D., Morita, H., Sinkjær, T. & Nielsen, J. 1998. Evidence that a transcortical pathway contributes to stretch reflexes in the tibialis anterior muscle in man. Journal of Physiology 512 (1), 267–276.

Pfurtscheller, G., Stancák, Jr.A. & Neuper, C. 1996. Event-related synchronization (ERS) in the in the alpha band – an electrophysiological correlate of cortical idling: A review. International Journal of Psychophysiology 24, 39–46.

Piirainen, J.M, Linnamo, V., Cronin, N.J. & Avela, J. 2013. Age-related neuromuscular function and dynamic balance control during slow and fast balance perturbations. Journal of Neurophysiology 110, 2557–2562.

Piirainen, J.M., Cronin, N.J., Avela, J. & Linnamo, V. 2014. Effects of plyometric and pneumatic explosive strength training on neuromuscular function and dynamic balance control in 60–70 year old males. Journal of Electromyography and Kinesiology 24 (2), 246–252.

Piitulainen, H., Bourguignon, M., Hari, R. & Jousmäki, V. 2015. MEG-compatible pneumatic stimulator to elicit passive finger and toe movements. Neuroimage 112, 310–317.

Piitulainen, H., Seipäjärvi, S., Avela, J., Parviainen, T. & Walker, S. 2018a. Cortical proprioceptive processing is altered by aging. Frontiers in Aging Neuroscience 10, 147.

Piitulainen, H., Illman, M., Laaksonen, K., Jousmäki, V. & Forss, N. 2018b. Reproducibility of corticokinematic coherence. Neuroimage 179, 596–603.

Pruszynski, J.A., Kurtzer, I. & Scott, S.H. 2011. The long-latency reflex is composed of at least two functionally independent processess. Journal of Neurophysiology 106, 449–459.

Rothwell, J.C., Traub, M.M. & Marsden, C.D. 1980. Influence of voluntary intent on the human long-latency stretch reflex. Nature 286, 496–498.

Rothwell, J.C., Traub, M.M., Day, B.L., Obeso, J.A., Thomas, P.K. & Marsden, C.D. 1982. Manual motor performance in a deafferented man. Brain 105 (3), 515–542.

Salenius, S., Schnitzler, A., Salmelin, R., Jousmäki, V. & Hari, R. 1997. Modulation of human cortical rolandic rhythms during natural sensorimotor tasks. Neuroimage 5 (3), 221–228.

Salles, J.I., Velasques, B., Cossich, V., Nicoliche, E., Ribeiro, P., Amaral, M.V. & Motta, G. 2015. Journal of Athletic Training 50 (3), 277–280.

Sammut, R., Thickbroom, G.W., Wilson, S.A. & Mastaglia, F.L. 1995. The origin of the soleus late response evoked by magnetic stimulation of human motor cortex. Electroencephalography and Clinical Neurophysiology 97 (3), 164–168.

Scholz, J.P. & Schöner, G. 1999. The uncontrolled manifold concept: Identifying control variables for a functional task. Experimental Brain Research 126, 289–306.

Schuurmans, J., de Vlugt, E., Schouten, A.C., Meskers, C.G.M, de Groot, J.H. & van der Helm, F.C.T. 2009. The monosynaptic Ia afferent pathway can largely explain the stretch duration effect of the long latency M2 response. Experimental Brain Research 193, 491–500.

Shemmell, J., Krutky, M.A., Perreault, E.J. 2010. Stretch sensitive reflexes as an adaptive mechanism for maintaining limb stability. Clinical Neurophysiology 121 (10), 1680–1689.

Signorile, J.F., Applegate, B., Duque, M., Cole, N. & Zink, A. 2002. Selective recruitment of the triceps surae muscles with changes in knee angle. Journal of Strength and Conditioning Research 16 (3), 433–439.

Siegelbaum, S.A., Kandel, E.R. & Yuste, R. 2013. Synaptic integration in the central nervous system. In a writing of Kandel, E.R., Schwartz, J.H., Jessell, T.M., Siegelbaum, S.A. & Hudspeth, A.J. (publ.) Principles of neural science. 5th edition. McGraw-Hill-companies.

Sinkjaer, T., Andersen, J.B. Larsen, B. 1996. Soleus stretch reflex modulation during gait in humans. Journal of Neurophysiology 76 (2), 1112–1120.

Sinkjaer, T., Andersen, J.B., Nielsen, J.F. & Hansen, H.J. 1999. Soleus long-latency stretch reflexes during walking in healthy and spastic humans. Clinical Neurophysiology 110 (5), 951–959.

Smith, C.D., Umberger, G.H., Manning, E.L., Slevin, J.T., Wekstein, D.R., Schmitt, F.A., Markesbery, W.R., Zhang, Z., Gerhardt, G.A., Kryscio, R.J., Gash, D.M., 1999. Critical decline in fine motor hand movements in human aging. Neurology 53, 1458–1461.

Smeds, E., Piitulainen, H., Bourguignon, M., Jousmäki, V. & Hari, R. 2017. Effect of interstimulus interval on cortical proprioceptive responses to passive finger movements. The European Journal of Neuroscience 45 (2), 290–298.

Spitzer, A. & Claus, D. 1992. The influence of the shape of mechanical stimuli on muscle stretch reflexes and SEP. Clinical Neurophysiology 85 (5), 331–336.

Stackhouse, S.K., Stevens, J.E., Lee, S.C.K., Pearce, K.M., Snyder-Mackler, L. & Binder-Macleod, S.A. 2001. Physical Therapy 81 (5), 1102–1109.

Suga, R., Tobimatsu, S., Taniwaki, T., Kira, J. & Kato, M. 2001. The soleus late response elicited by transcranial magnetic stimulation reflects agonist-antagonist postural adjustment in the lower limbs. Clinical Neurophysiology 112 (12), 2300–2311.

Taube, W., Gruber, M., Beck, S., Faist, M., Gollhofer, A. & Schubert, M. 2006. Cortical and spinal adaptations induced by balance training: correlation between stance stability and corticospinal activation. Acta Physiologica 189 (4), 347–358.

Taulu, S. & Simola, J. 2006. Spatiotemporal signal space separation method for rejecting nearby interference in MEG measurements. Physics in Medicine and Biology 51 (7), 1759–1768.

Timothy, J. & Brown, W.D. 1993. The estimated numbers and relative sizes of thenar motor units as selected by multiple point stimulation in young and older adults. Muscle Nerve 16 (4), 355–366.

Thilmann, A.F., Schwarz, M., Töpper, R., Fellows, S.J. & Noth, J. 1991. Different mechanisms underlie the long-latency stretch reflex response of active human muscle at different joints. Journal of Physiology 444, 631–643.

Trans, T., Aaboe, J., Henriksen, M., Christensen, R., Bliddal, H. & Lund, H. 2009. Effect of whole body vibration exercise on muscle strength and proprioception in females with knee osteoarthritis. The Knee 16(4), 256–261.

Uysal, H., Larsson, L-E., Efendi, H., Burke, D. & Ertekin, C. 2009. Medium-latency reflex response of soleus elicited by peroneal nerve stimulation. Experimental Brain Research, vol 193 (2), 275–286.

Van Doornik, J., Masakado,Y., Sinkjaer,T. & Nielsen, J.B. 2004. The suppression of the long-latency stretch reflex in the human tibialis anterior muscle by transcranial magnetic stimulation. Experimental Brain Research 157, 403–406.

Xiang, J., Kakigi, R., Hoshiyama, M., Kaneoke, Y., Naka, D., Takeshima, Y. & Koyama, S. 1997. Somatosensory evoked magnetic fields and potentials following passive toe movements in humans. Electroencephalography and Clinical Neurophysiology 104, 393–401.

Yavuz, S.U., Mrachacz-Kersting, Sebik, O., Ünver, M.B., Farina, D. & Türker, K.S. 2014. Human stretch reflex pathways reexamined. Journal of Neurophysiology 111, 602–612.

Zumsteg, D. & Wieser, H.G. 2002. Effects of aging and sex on middle-latency somatosensory evoked potentials: normative data. Clinical Neurophysiology, vol 113 (5), 681–685.

APPENDICES

APPENDIX 1 Strength training intervention summary

Weeks 1-2 (familiarization)

Training twice per week

14-16 reps with 40-60% max weights, 1 min rest between sets

2-3 Sets per movement, 8-9 movements. “Whole body program” with alternating movements.

Weeks 3-5

Training twice per week

10-12 reps with 70-80% of max weights, 1 min rest between sets

3 sets in main “movements”, 2 in others

Calfs 10-12 reps with 70-80%

Whole body program with alternating movements.

Weeks 6-7

Training twice per week

8-10 reps with 80-85% of max weights, 1 min rest between sets

3 sets in main “movements”, 2 in others

Calfs 8-10 x 80-85

Whole body program with alternating movements.

Weeks 8-9

Training twice per week

5-8 reps per with 85-90% of max weights, 1-2 min rest

Calfs12-14 x 60-70%, 8-10 x 80-85

3 sets in main “movements”, 2 in others

Whole body program with alternating movements.

Core exercises done as “circuits”

Weeks 10-12

Training twice per week

2 sets of 4-6 reps with 90-95% of max in the leg press. In other movements 6-8 reps with 85-90%. 2 min rests.

Calfs 7-8 x 85-90%, Jumps included this week 7-8 x

Whole body program” with alternating movements

Weeks 13-14

Training twice per week

4-6 reps with 90-95% of max in several movements. Also longer sets with lower weights

Calfs 7-8 x 85-90%, Jumps 7-8 x

Alternating movements

POST Tests

Movements done for calf muscles during the intervention

- Calfs in sitting position
- Calfs in standing position
- Calf jumps

APPENDIX 2 Strength training program

Week 1-2: Day 1				
Movement	Set 1	Set 2	Set 3	Set 4
Horizontal leg press	14-16x40-60%	14-16x40-60%	14-16x40-60%	
Knee extension	14-16x40-60%	14-16x40-60%		
Knee flexion	14-16x40-60%	14-16x40-60%		
Bench press (machine)	14-16x40-60%	14-16x40-60%		
Lat pulldown	14-16x40-60%	14-16x40-60%		
Tricep pushdown	14-16x40-60%	14-16x40-60%		
Trunk flexion (machine)	14-16x40-60%	14-16x40-60%		
Back extension (machine)	14-16x40-60%	14-16x40-60%		
Day 2				
Horizontal leg press	14-16x40-60%	14-16x40-60%	14-16x40-60%	
Knee extension	14-16x40-60%	14-16x40-60%		
Knee flexion	14-16x40-60%	14-16x40-60%		
Military press (machine)	14-16x40-60%	14-16x40-60%		
Seated lat row	14-16x40-60%	14-16x40-60%		
Low pulley biceps curl	14-16x40-60%	14-16x40-60%		
seated calf raise	14-16x40-60%	14-16x40-60%		
Trunk flexion (machine)	14-16x40-60%	14-16x40-60%		
Back extension (machine)	14-16x40-60%	14-16x40-60%		

Week 3-5: Day 1				
Horizontal leg press	10-12x70-80%	10-12x70-80%	10-12x70-80%	
Knee extension	10-12x70-80%	10-12x70-80%	10-12x70-80%	
Knee flexion	10-12x70-80%	10-12x70-80%		
Bench press (machine)	10-12x70-80%	10-12x70-80%	10-12x70-80%	
Lat pulldown	10-12x70-80%	10-12x70-80%		
Tricep pushdown	10-12x70-80%	10-12x70-80%		
Standing calf raises (Hack)	10-12x70-80%	10-12x70-80%	10-12x70-80%	
Trunk flexion (machine)	14-16x50-70%	14-16x50-70%		
Back extension (machine)	14-16x50-70%	14-16x50-70%		
Day 2				
Horizontal leg press	10-12x70-80%	10-12x70-80%	10-12x70-80%	
Knee extension	10-12x70-80%	10-12x70-80%		
Knee flexion	10-12x70-80%	10-12x70-80%	10-12x70-80%	
Military press (machine)	10-12x70-80%	10-12x70-80%		
Seated lat row	10-12x70-80%	10-12x70-80%	10-12x70-80%	
Low pulley biceps curl	10-12x70-80%	10-12x70-80%		
Seated calf raise	10-12x70-80%	10-12x70-80%	10-12x70-80%	
Trunk flexion (machine)	14-16x50-70%	14-16x50-70%		
Back extension (machine)	14-16x50-70%	14-16x50-70%		

Week 6-7: Day 1				
Horizontal leg press	8-10x80-85%	8-10x80-85%	8-10x80-85%	8-10x80-85%
Knee extension	8-10x80-85%	8-10x80-85%	8-10x80-85%	
Knee flexion	8-10x80-85%	8-10x80-85%		
Bench press (machine)	8-10x80-85%	8-10x80-85%	8-10x80-85%	8-10x80-85%
Lat pulldown	8-10x80-85%	8-10x80-85%		
Tricep pushdown	8-10x80-85%	8-10x80-85%		
Standing calf raises (Hack)	8-10x80-85%	8-10x80-85%	8-10x80-85%	
Trunk flexion (machine)	10-12x70-80%	10-12x70-80%		
Back extension (machine)	10-12x70-80%	10-12x70-80%		
Day 2				
Horizontal leg press	8-10x80-85%	8-10x80-85%	8-10x80-85%	8-10x80-85%
Knee extension	8-10x80-85%	8-10x80-85%		
Knee flexion	8-10x80-85%	8-10x80-85%	8-10x80-85%	
Military press (machine)	8-10x80-85%	8-10x80-85%		
Seated lat row	8-10x80-85%	8-10x80-85%	8-10x80-85%	8-10x80-85%
Low pulley biceps curl	8-10x80-85%	8-10x80-85%		
Seated calf raise	8-10x80-85%	8-10x80-85%	8-10x80-85%	
Trunk rotation (machine)	10-12x70-80%	10-12x70-80%		
Back extension (machine)	10-12x70-80%	10-12x70-80%		

Week 8-9: Day 1				
Horizontal leg press	5-8x85-90%	5-8x85-90%	5-8x85-90%	
Knee extension	6-8x85-90%	6-8x85-90%	6-8x85-90%	
Knee flexion (laying)	6-8x85-90%	6-8x85-90%	6-8x85-90%	
Lunge (with bar)	12-14x60-70%	12-14x60-70%		
Standing calf raises (Smith)	12-14x60-70%	12-14x60-70%		
Seated calf raise	8-10x80-85%	8-10x80-85%		
Bench press	12-14x60-70%	12-14x60-70%		
Military press (dumbbells)	12-14x60-70%	12-14x60-70%		
Sit-ups	12-14x60-70%	12-14x60-70%		
Lying back extensions	12-14x60-70%	12-14x60-70%		
Day 2				
Squat (Smith)	5-8x85-90%	5-8x85-90%	5-8x85-90%	
Knee extension	6-8x85-90%	6-8x85-90%	6-8x85-90%	
Knee flexion (laying)	6-8x85-90%	6-8x85-90%	6-8x85-90%	
Deadlift (barbells)	12-14x60-70%	12-14x60-70%		
Leg press calf raises	12-14x60-70%	12-14x60-70%	12-14x60-70%	12-14x60-70%
Standing lat row (barbell)	12-14x60-70%	12-14x60-70%		
Assisted pull-up (machine)	12-14x60-70%	12-14x60-70%		
Sit-ups	12-14x60-70%	12-14x60-70%		
Lying back extensions	12-14x60-70%	12-14x60-70%		

Week 10-12: Day 1				
Horizontal leg press	4-6x90-95%	4-6x90-95%	4-6x50-60%	

Knee extension	6-8x85-90%	6-8x85-90%	6-8x85-90%
Knee flexion (laying)	6-8x85-90%	6-8x85-90%	6-8x85-90%
Standing calf raises (Smith)	7-8x85-90%	7-8x85-90%	7-8x85-90%
Calf jumps	7-8x		
Split squat jumps	5 x	5 x	
Bench press	10-12x70-80%	10-12x70-80%	10-12x70-80%
Military press (dumbbells)	10-12x70-80%	10-12x70-80%	
Sit-ups	14-16 x	14-16 x	
Lying back extensions	14-16 x	14-16 x	
Day 2			
Horizontal leg press	4-6x90-95%	4-6x90-95%	4-6x50-60%
Knee extension	6-8x85-90%	6-8x85-90%	6-8x85-90%
Knee flexion (laying)	6-8x85-90%	6-8x85-90%	6-8x85-90%
Leg press calf raises	7-8x85-90%	7-8x85-90%	7-8x85-90%
Calf jumps	7-8x		
Countermovement jump	5 x	5 x	
Standing lat row (barbell)	10-12x70-80%	10-12x70-80%	10-12x70-80%
Assisted pull-up (machine)	10-12x70-80%	10-12x70-80%	
Sit-ups	14-16 x	14-16 x	
Lying back extensions	14-16 x	14-16 x	

Week 13-14: Day 1			
Horizontal leg press	4-6x90-95%	4-6x50-60%	4-6x50-60%
Knee extension	4-6x90-95%	4-6x90-95%	4-6x90-95%
Knee flexion (laying)	4-6x90-95%	4-6x90-95%	4-6x90-95%
Standing calf raises (Smith)	7-8x85-90%		
Calf jumps	7-8x	7-8x	7-8x
Split squat jumps	5 x	5 x	5 x
Pec-deck	8-10x80-85%	8-10x80-85%	8-10x80-85%
Assisted dip machine	8-10x80-85%	8-10x80-85%	8-10x80-85%
Sit-ups	16-20x	16-20x	
Lying back extensions	16-20x	16-20x	
Day 2			
Horizontal leg press	4-6x90-95%	4-6x50-60%	4-6x50-60%
Knee extension	4-6x90-95%	4-6x90-95%	4-6x90-95%
Knee flexion (laying)	4-6x90-95%	4-6x90-95%	4-6x90-95%
Leg press calf raises	7-8x85-90%		
Calf jumps	7-8x	7-8x	7-8x
Countermovement jump	5 x	5 x	5 x
Seated lat row	8-10x80-85%	8-10x80-85%	8-10x80-85%
Upright row	8-10x80-85%	8-10x80-85%	8-10x80-85%
Sit-ups	16-20x	16-20x	
Lying back extensions	16-20x	16-20x	