ROLE OF MOTOR CORTEX DURING DROP JUMP: MOTOR CORTICAL EXCITABILITY ASSESSED WITH TMS AND H-REFLEX STIMULATION

Timo Rantalainen

ABSTRACT

Rantalainen, Timo. 2005. Role of motor cortex during drop jump: motor cortical excitability assessed with TMS and H-reflex stimulation. Department of biology of physical activity, university of Jyväskylä. Master's thesis of biomechanics. 32 pages.

Pre-programmed activity has major influence on drop jump. The efficiency of a drop jump is dependent on the amount of preactivation. The source of voluntary muscle activation can be assessed with evoked potentials at different levels of motor control. The goal of the study was to clarify the roles of cortical and spinal segments in producing the muscle activation levels required in drop jumping. The motor program used in drop jumps with low drop heights was examined by stimulating the motor system at spinal (peripheral electrical stimulation, H-reflex) and cortical level (transcranial magnetic stimulation [TMS], motor evoked potential [MEP]) at different times from 50 ms prior to ground contact to ~120 ms after the ground contact. The motor program was studied by determining the spinal and corticospinal excitability changes during the drop jump (DJ) cycle. The cortical excitability changes were extracted from the corticospinal excitability with a novel correction factor approach in which the spinal excitability changes were extracted from corticospinal excitability. The assumption was made that the spinal excitability changes measured with H-reflex measurements cause similar relative facilitation to the MEP values. The DJs were conducted from a drop height of 30 cm and there were a total of 9 healthy men subjects with prior jumping exercise experience. The cortical excitability is high prior to ground contact lowered at ground contact and heightens through first 100 ms of ground contact. The spinal excitability is high from the beginning of ground contact to approximately 100 ms after the beginning of ground contact. The results indicate that the motor programming of drop jump takes reflex activity into account. The muscle activation is initiated with cortical drive after which the spinal drive is accounted for by lowering cortical excitability levels. Functionally the high spinal excitability during the first 100 ms of ground contact enables the maintenance of high neural drive to muscles until volitional corrections are available.

KEYWORDS: TMS, drop jump, cortical, spinal, excitability, motor program

CONTENTS

1   INTRODUCTION

1. 1   Supraspinal movement control

The output of motor programming is called the central command. The central command is directed to lower nerve centres, brain stem and spinal cord and back to higher nerve centres which take part in motor programming. (Enoka 1994.)

By EEG recordings it has been discovered that premotor cortex activates before primary motor cortex during preparations of voluntary movement (Brunia et al. 1985). The pyramidal e.g. corticospinal tract begins from motor cortex and part of the neurons has monosynaptic connections to motoneurons. Corticospinal tract directs precise voluntary movements. (Carpenter 1984.)
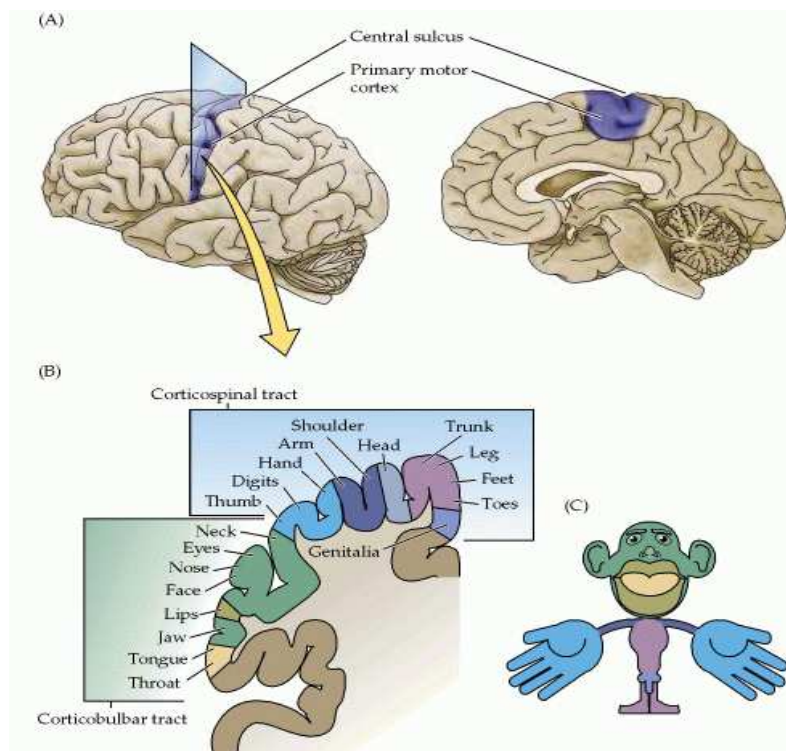
FIGURE 1. Purves et al. 2001. The location of motor cortex in the brain. (a), topographical map of the body parts (b) and the relative representation of body parts (c) on the motor cortex.

The motor cortex is situated in front of the central sulcus of the brain on both cerebral lobes. Body parts are represented on the motor cortex as a topographical map (figure 1). The neurons, which innervate foots are situated between the brain lobes. The pyramidal cell dendrites have branches to all layers of cortex. (Carpenter 1984.)
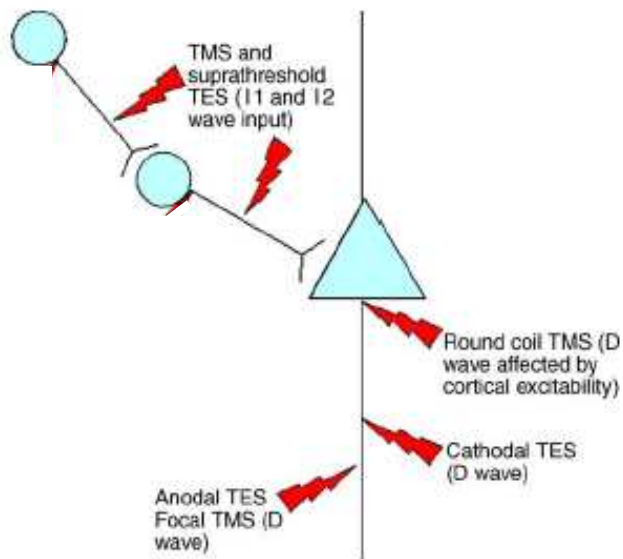
1.2 Spinal movement control

In addition to efferent nervation the sensory activation contributes to the movement (Enoka 1994). In some instances the supraspinal command needs to inhibit spinal reflexes (Carpenter 1984). Spinal cord makes fluent series of extensions and flexions available for standing, walking and running (Sage 1984). The spinal control enables the motor apparatus to adapt to surprising disturbances (Enoka 1994).

Spinal control is not largely under voluntary (supraspinal) control. Long latency reflexes can be mediated with earlier information. Mono- oligo- and polysynaptic neural connections regulate the excitability of motoneuron pool. Typically spinal control is conservative and tries to resist perturbations. (Latash 1998.)

1.3 Stimulation of motor cortex

Barker et al. (1985) discovered that the cortex of the brain could be stimulated with transcranial magnetic stimulation (TMS). They discovered that a rapidly changing magnetic field can be used to stimulate motor cortex painlessly and as a consequence of the stimulation a measurable EMG response will be evoked. (Barker et al. 1985.) Di lazarro et al. (2004) reported that at motor treshold TMS results in I-waves (indirect) and only with increased intensity TMS results with D-waves (caused by direct excitation of corticospinal axons). 100 mV in 50% of stimulations. Voluntary activation increases all I-waves. Because of this and facilitation of the motoneuron pool TMS during voluntary activation results in larger motor evoked potential (MEP) than TMS when the subject is passive. (Di lazzaro et al. 2004).

Di lazzaro et al. (2004) The intensity of the stimulation can be represented in relation to motor threshold (MT). which can be defined as an intensity that results in a response of FIGURE 2. Modified from Di Lazzaro et al. 2004. The possible sites of activation of corticospinal cells using different techniques of transcranial stimulation. Transcranial electrical stimulation (TES) and Transcranial magnetic stimulation (TMS).

A single D-wave can result in a measurable EMG-signal at rest. In some cases voluntary contraction is needed (Di lazarro et al. 2004). TMS and TES can result in multiple firings of α-motoneuron, which does not happen with peripheral stimulation (Day et al. 1989). The amplitude of D-wave when stimulating with circular coil is dependent on the cortical excitability and is therefore induced near the soma of the neuron (figure 2). (Di lazzaro et al. 2004).
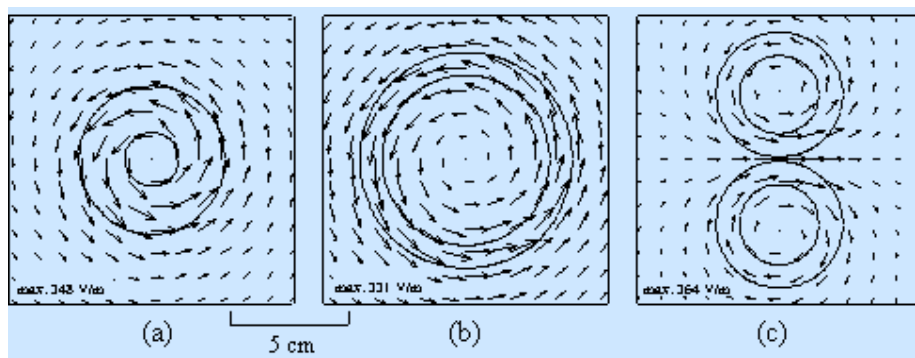

FIGURE 3. Ruohonen 1997. The electric field induced by different kinds of coils.

In magnetic stimulation the stimulation energy is transferred with magnetic field (Malmivuo 1992). The direction of the induced current in the brains (figure 3) is determined by the direction of the current in the coil and is opposite to the current in the coil (Ruohonen 1997).

The activation caused by TMS is affected by: the shape of the coil, the orientation of the coil, the induced current and the intensity of the stimulus. If stimulus intensity is to be varied, monophasic current pulse should be used. (Brasil-Neto et al. 1992.)

The TMS stimulation site has to be determined experimentally for each measurement. For circular coil the site of the coil is near the vertex. Circular coil should be oriented tangentially to the skull. If the right side of the brain is the side, which is to be stimulated the current should flow clockwise in the coil. The highest induced current will occur between the centre and the ring of the coil. Deeper cortical areas can be stimulated with circular coil compared to figure of eight shaped coil. (Reid 2003).

The reliability of TMS increases if stimulations are analyzed as blocks of more than one. Evidently the intraindividual intraclass reliability (ICC) of MEP settles to a value of ICC = 0.70 independent of the amount of stimulation blocks and measurement days. (Kamen 2004.) The variability of the MEP is greater than compound muscle action potential (CMAP) evoked by peripheral stimulation. For this reason several MEPs needs to be averaged in order to examine the amplitude of TMS response. (Reid 2003.) With high stimulation intensities MEP reaches a plateau but the twitch force continues to increase. The twitch force would thus give a more accurate estimate of the amount of motor units activated by TMS. (Reid 2003., Kiers et al. 1995.) The MEP area corresponds better to the twitch force than the MEP amplitude does. (Kiers et al. 1995).

Single pulse magnetic stimulation (sTMS) has no known side effects for healthy people (Wasserman 1998). Metal and magnetic objects, life supporting electrical devices and heightened intracranial pressure are contraindications to TMS (Wassermann 1998, Ruohonen 1999, Reid 2003). The same precautions should be taken in application of TMS as in magnetic resonance imaging. Within these limits the use of TMS is safe. (Reid 2003.).

During magnetic stimulation the coil makes a clicking sound so subjects should wear earplugs (Ruohonen 1999, Reid 2003). In some cases the application of TMS causes mild headache for sensitive subjects. The headache is most likely caused by the activation of neck and skull musculature. (Ruohonen 1999.) With sTMS headache is rarely a problem (Wassermann 1998). The stimulation frequency of sTMS is not to exceed 0,5 Hz in order to avoid the facilitatory and inhibitory effects of previous stimulation. (Reid 2003).

1.4   Spinal and corticospinal excitability

TMS can be used to assess the excitability of motor cortex if the function of more peripheral neural system is taken into account (Taylor & Gandevia 2001).

During fatiguing muscle activation MEP increases for 10 – 15 s from the beginning of activation and plateaus after that. During fatigue also M-wave increases, but even when MEP is normalized with M-wave, MEP still increases. (Taylor et al. 1996, Taylor & Gandevia 2001, Taylor et al 1999.) After even a brief period of activity the MEP induced to a passive muscle is facilitated. The facilitation disappears after 2 – 4 min. (Taylor & Gandevia 2001.)

During maximal voluntary contraction (MVC) fatiguing protocol application of TMS increases produced force. Therefore the output of motor cortex is not maximal during MVC. (Taylor & Gandevia 2001, Wolfgang & Nordlund 2002.) Also in non-fatigued MVC application of TMS increases produced force. The deficit during fatiguing voluntary activation is especially marked during eccentric activation. (Wolfgang & Nordlund 2002.)

MEP is facilitated instantly after fatiguing exercise (Liepert et al. 1996, Kato et al 2003). Fatigue is compensated with activation of nearby motor cortical areas. Thus TMS excites more neurons on motor cortex during fatigue. (Kato et al. 2003.) Central fatigue is not caused by fatigue of motor cortex during isometric MVC (Gandevia et al.

1996). After MVC fatiguing protocol the MEP falls to minimal value in 2 min after the cessation of exercise and stays depressed for 10 min afterwards. (Gandevia et al. 1999).

When TMS intensity is lower than active motor threshold (ATM) during voluntary activation, there is no facilitation of H-reflex detected due to TMS. (Ziemann et al. 1996.) 60 - 80 ms before voluntary movement facilitation of MEP starts in the agonist (Pascual-Leone et al. 1992, Hoshiyama et al. 1996, Leocani et al. 2000) and the inhibition of MEP starts in the antagonist. Because there is no movement induced at that time the inhibition can not be caused by proprioceptic factors. The motor program of cerebral cortex regulates the facilitation and inhibition of the movement inducing muscle and this regulation is ready 60 ms before the movement. The regulation can take place at cortical and spinal level. (Hoshiyama et al. 1996.) Prior to movement onset the corticospinal facilitation starts to increase and is the greatest at onset of movement initiation. Corticospinal facilitation is greater in phasic contractions than in tonic contractions and facilitation increases with movement speed. (Nielsen & Petersen 1995.)

During voluntary activation MEP caused by TMS is increased with increasing EMG levels. The increment of MEP is dependent on the recruitment order of the specific muscle. (Taylor & Gandevia 2001, Nowicky et al. 2001.) Contralateral inhibition caused by voluntary contraction does not affect cervicomedullarry evoked muscle action potential (CMEP) and MEP is facilitated during contralateral voluntary activation in hand muscles (Hortobágyi et al. 2003). During voluntary contraction the M_{max} tends to increase. When H_{max} / M_{max} ratio is compared during maximal voluntary contraction and at rest there is no significant difference, which suggests that the inhibitory mechanisms remain similar to rest during MVC. (Pensini & Martin 2004.)

Much of H-reflex modulation during voluntary movement occurs pre-synaptically (Stein 1995). In foot Ia-afferent presynaptic inhibition is decreased during voluntary activation in both the agonist and the antagonist muscles (Meunier & Pierrot-Deseilligny 1998). H-reflex is more affected by presynaptic inhibition than stretch reflex. Previously activated afferents are less affected by the presynaptic inhibition. (Morita et al. 1998, Enríquez-Denton et al. 2002.) According to Crone et al. (1990) H-reflex modulation is largely dependent on the test reflex size. If test H-reflex is 20 –

40% of M_{max} the effects of inhibition and facilitation will be most evident. (Crone et al. 1990).

The facilitation of corticospinal system probably takes place at cortical level before onset of voluntary activation (Davey et al. 1998). Long latency reflexes have transcortical segments (Petersen et al. 1998, Christensen et al. 2001). Stretch elicits three bursts of reflex activity (M1 – M3). The M3 reflex loop has transcortical segments. (Petersen et al. 1998.)

1.5   Motor programming

Motor program is a series of preplanned motor commands, which enable the execution of a movement without peripheral feedback (Klapp 1996). The afferent nervation of muscles makes studying of the motor control challenging. If movement is evoked by stimulation the stimulation will cause sensory feedback, which can affect the activation. (Carpenter 1984.) In rapid movements the motor program has to be fully programmed before execution of the movement (Carpenter 1984). The shortest movement in which volitional corrections are possible is 100-200 ms (Kerr 1978).

For drop jumps there are different kinds of motor strategies that have been discovered to been used. Some subjects seem to land first and after that perform a subsequent jump whereas others seem to not bother themselves with landing. (Dyhre-Poulsen et al. 1991, Viitasalo et al. 1998, Horita et al. 2002.) When comparing jump trained and untrained subjects in drop jumps it is evident that from higher drop heights (> 40 cm) the untrained subjects are unable to jump efficiently (Viitasalo et al. 1998).

During drop jumps higher levels of preactivation (~50 ms before ground contact) of gastrocnemius medialis are associated with more efficient jumping pattern (the exploitation of stretch shortening cycle). (Horita et al. 2002.) Agonist premotor silent period (PMS) is associated with increased velocity and acceleration of the upcoming activity (Walter 1988). Premovement silent period in ballistic movements is produced at supraspinal level. H-reflex is not surpressed during the PMS. The likely role of

premovement silent period is to enhance the performance during the ensuing ballistic movement. (Aoki et al. 2002.)

Voluntary movement requires planning of the movement prior to movement onset. Before short lasting movements (less than 200 ms) the whole movement needs to be pre-programmed and corrections during the movement are impossible. It is known that preactivation heightens the efficiency of stretch shortening cycle. Is the level of preactivation decided at spinal or supraspinal (cortical) level? The goal of the study was to clarify the roles of cortical and spinal segments in producing the muscle activation levels required in drop jumping. The results would thus shed light on the magnitude of activity produced at spinal level compared to activity produced at cortical level in voluntary activation.

The goal of this study is to examine the role of motor cortex during the execution of a drop jump. The role of motor cortex is assessed by examining the corticospinal excitability changes during drop jump.

2   METHODS

2.1   Subjects and experimental protocol

The study was conducted with written consent form the subjects. Nine healthy male university student volunteers with prior jumping exercise training were subjects to this study. The measurements were conducted on two separate days. On both of the measurement days the subjects conducted drop jumps from 30 cm drop height. On the other day subjects were peripherally electrically stimulated for soleus H-reflex and on the other stimulated with transcranial magnetic stimulation (TMS) for soleus MEP.

TABLE 1. The protocol.

1	10 drop jumps to measure temporal pattern of EMG and ground reaction force	
2		
	20 to 50 stimuli to determine motor threshold, H-max, M-max and excitability curves	
3	3 times 5 times 8 jumps plus control stimulus with stimulation at different temporal sites 5 times 8 jumps plus control stimulus with higher stimulation intensity	
4		Intended timing
	Stimulus 1	50 ms before ground contact
	Stimulus 2	~15 ms after ground contact
	Stimulus 3	M1 reflex response
	Stimulus 4	M2 reflex response
	Stimulus 5	M3 reflex response
	Stimulus 6/ Control	Control stimulus while standing still
	Control 1 no stimulus	Background EMG
	Control 2 no stimulus	Background EMG
	Stimulus 7	EMG peak after ~120 ms

First the subjects were asked to perform 10 drop jumps to reveal the temporal pattern of contact time and EMG (Table 1). Secondly the stimulating intensity and the excitability curve for H-reflex or TMS was determined. In case of TMS measurements the first two measurements were conducted in the opposite order. For TMS the motor threshold (MT) intensity was determined while subjects were standing. TMS intensity was

accepted as MT intensity when 3 of 5 stimulations produced a response of 100 mV or more.

The temporal patterns for EMG and ground reaction force were determined from the rectified EMG and ground reaction force traces separately for each individual and once for each of the separate measurement days (table 1). All EMG and force signals were averaged after which the onset of ground contact and contact time were determined from the force signals. The EMG spikes needed and the stimulus latencies were determined from the EMG signal.

After the variables for temporal stimulating sites and the temporal pattern before the ground contact were determined the drop jumps were conducted. The subjects performed four times five times nine completions. Five times eight jumps and one control stimulus were completed with six second intervals after which a five to 10 min break was taken according to the subjects wishes after which the same five times nine completions were performed again until all the completions were finished. In the TMS measurement the last set of five times nine was omitted and replaced with a few more control stimuli with higher stimulation intensity to control for the possible ceiling effect.

2.2   Stimulation

For determining the stimulation site the EMG tibialis anterior and soleus were projected with oscilloscope and the EMG response resulting from the stimulus was observed visually. When at constant stimulus intensity the response was maximal in soleus and minimal in tibialis anterior the stimulating site was accepted for the rest of the measurements and the stimulating apparatus was fixated properly.

The timing of stimulation was decided from the temporal pattern of EMG. Stimulation was conducted at the time of wished stimulation point subtracted by the latency of stimulation. In practice the stimulation was triggered by photocell or by ground reaction force. For the two earliest stimulation points the photocell was used for triggering and for the latter four points the force plate was used for triggering. The trigger threshold for force plate was 2 N. The average stimulation intensity for the peripheral electrical

stimulation was $13,4 \pm 5,5$ mA for the H-reflex amplitude of 20% of M_{max} and $18,7 \pm 6,9$ mA for M-wave 50% of M_{max}. The latency of peripheral stimulation was $38,4 \pm 2,4$ ms. For timing of the stimuli (and 8) refer to table 2

Table 2. Average values of the actual timing of stimuli in milliseconds. The first value is before ground contact and all the others are after ground contact.

	Stimulus 1	Stimulus 2	Stimulus 3	Stimulus 4	Stimulus 5	Stimulus 7
TMS	$48,7 \pm 3,4$	$17,6 \pm 7,7$	$47,4 \pm 4,6$	$74,0 \pm 4,4$	$91,3 \pm 4,6$	$119,7 \pm 5,3$
H-reflex	$49,6 \pm 7,5$	$16,4 \pm 9,8$	$43,9 \pm 4,4$	$70,4 \pm 6,3$	$92,4 \pm 6,4$	$117,8 \pm 5,7$

2.2.1 Transcranial magnetic stimulation

TMS stimulation was conducted with Magstim 200 Mono Pulse magnetic stimulator using circular coil. TMS stimulation site was defined moving the coil near the vertex

FIGURE 4. Fixating the magnetic coil with the custom made helmet.

and tangentially to the skull. The proper stimulating site was usually so that the centre of the coil was a few centimetres to the side of the stimulated foot in which case the vertex was situated between the ring of the coil and the centre of the coil. The magnetic coil was fixated for the duration of the measurement with a custom made helmet which allowed negligible movement of the coil relative to the skull (figure 4). The intensity of

stimulation was kept constant for drop jump trials. The intensity for TMS was 95% of the motor threshold. The intensity for ceiling effect control trials was 120% of MT. The average stimulation intensity for TMS measured by the coiled wire attached to the stimulating coil was 58 ± 10 % of the stimulator output for stimulation intensity of 95% of MT. The intensity was and 69 ± 9 % of the stimulator output for 120% of stimulation intensity of MT. The latency of TMS was $37,8 \pm 3,5$ ms. For timing of the stimuli (figure 6) refer to table 2

2.2.2 Peripheral electrical stimulation

Electrical stimulation was conducted with Digitimer DS7A constant current stimulator using 500 µs rectangular pulse. Peripheral electrical stimulation was conducted placing the rectangular anode below the patella and searching the optimal stimulating site from popliteal fossa with the circular cathode. The stimulating electrode for the peripheral stimulation was fixated with tape.

During the first three sets of drop jumps the intensity of stimulation was kept constant and for the last set the intensity was increased. The intensity for electrical stimulation was set so that the H-reflex amplitude was 20% of M_{max} in for the first free sets. The intensity was set so that M-wave was 50% of M_{max} for the last set with the higher stimulation intensity. After each rest period the intensity of electrical stimulation was adjusted to the correct value. The intensity of TMS was controlled with a coiled wire attached to the magnetic coil. The coiled wire measured the percentage of the induced field compared to the maximal field that the coil was able to induce.

2.3 Registering and collecting the signals

EMG, force signal and stimulation intensity were registered. EMG was registered with single reusable electrodes with circular terminals. The electrodes were used as bipolar electrodes. The electrodes were placed on the skin with inter electrode distance of approximately 2,5 cm and the reference electrode was placed on top of tibia. EMG was

registered from right leg from the tibialis anterior, gastrocnemius lateralis & medialis and soleus muscles. To minimize artefact the wires from the electrodes were fixated with tape to the leg of the subject and held apart from the stimulating coil and the cords to the stimulating electrodes.

Force signal was registered from the force plate. Stimulation intensity was registered from the actual output of electrical stimulation or with a coiled wire from the magnetic stimulation coil.

The sampling frequency for all signals was 4000 Hz and the signals were 50 – 2000 Hz band pass filtered. After filtering the data was collected with IMAGO data collection program.

2.4   The experimental setup

The experimental setup for drop jumps consisted of a 30 cm high platform, photocells which were positioned at a height that corresponded to approximately 100 ms before ground contact and of a force plate. The force plate was surrounded with additional platforms to prevent injury if subject lost balance. (figure 5)

FIGURE 5. The setup for drop jumps.

The drop jumps were performed with the subjects' arms positioned freely to help sustain balance. The subjects were instructed to make the ground contact as short as possible and to make each repetition as identical as possible. The ground reaction force

was observed online with an oscilloscope to control that all the jumps were approximately identical.

2.5   Analysis

The H-reflex and TMS values for each stimulation point were obtained from the averaged curve of all stimuli stimulated with the same stimulation intensity. The value of the response was determined with 20 ms root mean square (RMS) value and with the peak-to-peak amplitude of the response. The 20 ms window was situated around the highest peak of the H-reflex and at the earliest visible peak of the control MEP response. The earliest visible peak was used for MEP values because MEP latency is decreased during voluntary activation.

To assess the spinal and cortical excitability the background EMG value for corresponding time span was subtracted from the H-reflex and MEP values. Background EMG value was determined by calculating the 20 ms RMS or peak-to-peak values around the intended stimulation sites from the background EMG trials.
To cancel the effect of spinal excitability changes from the cortical excitability a correction factor ($C = H_m/H$) was formed. The percentage of spinal excitability change compared to the control value was calculated by dividing the H-reflex value at certain stimulation site (H_m) with control H-reflex value (H). The TMS values were divided by correction factor to compensate for the spinal excitability changes.

To control the possible stimulation intensity changes of peripheral electrical stimulation the M-wave from the higher stimulation intensity trials was collected. The M-waves were analysed similarly to the H-reflex and TMS values.

After all the individual subject values had been determined, the values for individuals were averaged to produce single value for each variable for the purposes of creating plots and diagrams. These values are the values found in the results but the statistical analysis was done with the average results of each individual so that the n of the study was the same as the number of subjects.

For the ground reaction force all the trials for determining the background EMG were averaged to obtain the standard deviation of ground reaction force during the ground contact. The TMS ceiling control stimuli were analyzed the same way as the drop jump control trials.

The stimulation intensity was averaged over all trials with supposedly the same stimulation intensity and the standard deviation of the stimulation intensity was determined for both TMS and peripheral electrical stimulation.

Statistical analysis was performed using the SigmaStat statistical analysis program. To determine if there were differences between the different stimulation times the resulting responses were compared with repeated measures ANOVA and if there were differences the differences were assessed with Bonferroni's against control measurement multiple comparisons tests. The stimulation intensity and ceiling control measurements were assessed with paired t-test and Pearson correlation coefficient. RMS and peak to peak (AMP) values for soleus TMS trials were tested for correlation with Pearson correlation coefficient. The significance level was set at $p = 0.05$. All significance statements are against the control value.

3   RESULTS

The average ground reaction force during the ground contact for H-reflex trials and TMS trials were 2227 ± 517 N and 2237 ± 556 N respectively (coefficient of variation (CV) 23 % and 25% respectively) (figure 6). The average contact times were 213 ± 20 ms and 219 ± 30 ms for H-reflex trials and TMS-trials respectively (CV 9% and 14% respectively).

FIGURE 6. A typical sample of 500 ms's of average ground reaction force with standard deviation and soleus background EMG plotted against time from 15 jumps of individual subject from control H-reflex (Left) and TMS trials (Right). The stimuli 1 through 5 and 7 are plotted in ascending order under the background EMG in corresponding temporal placing. The bottom most trace is from control stimuli. The analysed window is highlighted with grey rectangle.

The TMS ceiling control stimuli MEP values were significantly (p = 0,05) different from the control stimuli recorded during the trials. The correlation between the ceiling control and control stimuli was 0.663, which is nearly significant (p = 0.073). The correlation between soleus TMS RMS and AMP values was significant (p = 0.000) and the correlation was 0.572.

The H-reflex and M-wave of the higher stimulation intensity didn't correlate significantly. The correlation coefficient was 0.084. There were significant correlations between higher and lower intensity H-reflex values at some stimulation times. There was significant correlation with stimulation times 1, 2 and 4 between H-reflex values of lower and higher intensity electrical stimulations. The higher stimulation intensity H-reflex values were lower than the H-reflex values of lower stimulation intensity and the difference was significant. There were no significant differences between the stimulation intensities measured from the corresponding M-wave values. If the average M-wave values were compared there was an apparent intensity drop of 10 % at stimulation time 4 but there was no correlation between the control and stimulation time 4 M-wave values (p =0.904).

For average MEP values at different temporal sites during the drop jumps refer to table 3 All values are in mVs. Significances were calculated compared to control.

TABLE 3. Soleus, gastrocnemius medialis, gastrocnemius lateralis and tibialis anterior MEP RMS values

Soleus	Control	Stimulus 1	Stimulus 2	Stimulus 3	Stimulus 4	Stimulus 5	Stimulus 7
Average	0.044	0.098	0.128	0.174	0.165	0.221	0.097
STDEV	0.024	0.051	0.054	0.088	0.125	0.117	0.095
Significance				**	**	***	
Gastr. med	Control	Stimulus 1	Stimulus 2	Stimulus 3	Stimulus 4	Stimulus 5	Stimulus 7
Average	0.116	0.256	0.181	0.181	0.238	0.214	0.058
STDEV	0.084	0.203	0.160	0.161	0.262	0.239	0.085
Significance							
Gastr. lat.	Control	Stimulus 1	Stimulus 2	Stimulus 3	Stimulus 4	Stimulus 5	Stimulus 7
Average	0.073	0.203	0.140	0.158	0.152	0.137	0.110
STDEV	0.060	0.130	0.093	0.094	0.102	0.077	0.129
Significance		*					
Tibialis ant	Control	Stimulus 1	Stimulus 2	Stimulus 3	Stimulus 4	Stimulus 5	Stimulus 7
Average	0.120	0.262	0.289	0.264	0.204	0.194	0.124
STDEV	0.120	0.105	0.142	0.223	0.215	0.221	0.133
Significance		***	**	**			

For average H-reflex values refer to table 4.

TABLE 4. Soleus, gastrocnemius medialis, gastrocnemius lateralis and tibialis anterior H-REFLEX RMS values. All values in mVs

Soleus	Control	Stimulus 1	Stimulus 2	Stimulus 3	Stimulus 4	Stimulus 5	Stimulus 7
Average	0.196623	0.194642	0.440823	0.385201	0.364694	0.321288	0.21357
STDev	0.090285	0.187165	0.220921	0.21724	0.162999	0.175757	0.15071
Significance			*				
Gastr. Med.	Control	Stimulus 1	Stimulus 2	Stimulus 3	Stimulus 4	Stimulus 5	Stimulus 7
Average	0.140642	0.217663	0.39269	0.330647	0.32704	0.342002	0.071051
STDev	0.039372	0.210746	0.282479	0.270075	0.254665	0.278272	0.103719
Significance			*				
Gastr. lat.	Control	Stimulus 1	Stimulus 2	Stimulus 3	Stimulus 4	Stimulus 5	Stimulus 7
Average	0.145133	0.233073	0.397947	0.380391	0.375269	0.36873	0.190451
STDev	0.110408	0.155404	0.155058	0.166837	0.15007	0.201332	0.134104
Significance			***	**	**	**	
Tibialis ant.	Control	Stimulus 1	Stimulus 2	Stimulus 3	Stimulus 4	Stimulus 5	Stimulus 7
Average	0.041921	0.094343	0.128992	0.08896	0.058477	0.050501	0.029113
STDev	0.022313	0.054533	0.084547	0.037681	0.023121	0.020348	0.013483
Significance		*	*				

For Background EMG values at times corresponding to stimuli refer to table 5.

TABLE 5. Soleus, gastrocnemius medialis, gastrocnemius lateralis and tibialis anterior background EMG values at times corresponding to the stimuli from TMS and H-reflex trials. All values in mVs

TMS BG	Simulus 1	Stimulus 2	Simulus 3	Stimulus 4	Simulus 5	Stimulus 7
Soleus	0.015829	0.075282	0.082998	0.117759	0.113126	0.095302
STDev	0.009262	0.120025	0.104331	0.12533	0.100609	0.072894
Gastro Med	0.079291	0.085639	0.10989	0.125329	0.123866	0.11365
STDev	0.054106	0.061237	0.094258	0.08688	0.070166	0.07416
Gastro Lat	0.060947	0.070965	0.079589	0.119057	0.119135	0.093416
STDev	0.040062	0.04744	0.05865	0.071399	0.055112	0.045409
Tibialis Ant	0.023048	0.01995	0.014464	0.020001	0.019738	0.020751
STDev	0.023401	0.013257	0.009103	0.01647	0.010873	0.015126
H-reflex BG	Simulus 1	Stimulus 2	Simulus 3	Stimulus 4	Simulus 5	Stimulus 7
Soleus	0.020629	0.063642	0.088733	0.08899	0.115015	0.06728
STDev	0.016137	0.042117	0.100471	0.036309	0.097365	0.06364
Gastro Med	0.063761	0.102125	0.083777	0.091611	0.088241	0.106384
STDev	0.050583	0.076658	0.054965	0.064617	0.056263	0.082069
Gastro Lat	0.0477	0.078743	0.070795	0.106028	0.11089	0.107921
STDev	0.015913	0.036833	0.026869	0.044644	0.050549	0.061262
Tibialis Ant	0.019397	0.025378	0.016607	0.0223	0.019679	0.020129
STDev	0.010447	0.016635	0.005854	0.016348	0.006814	0.015599

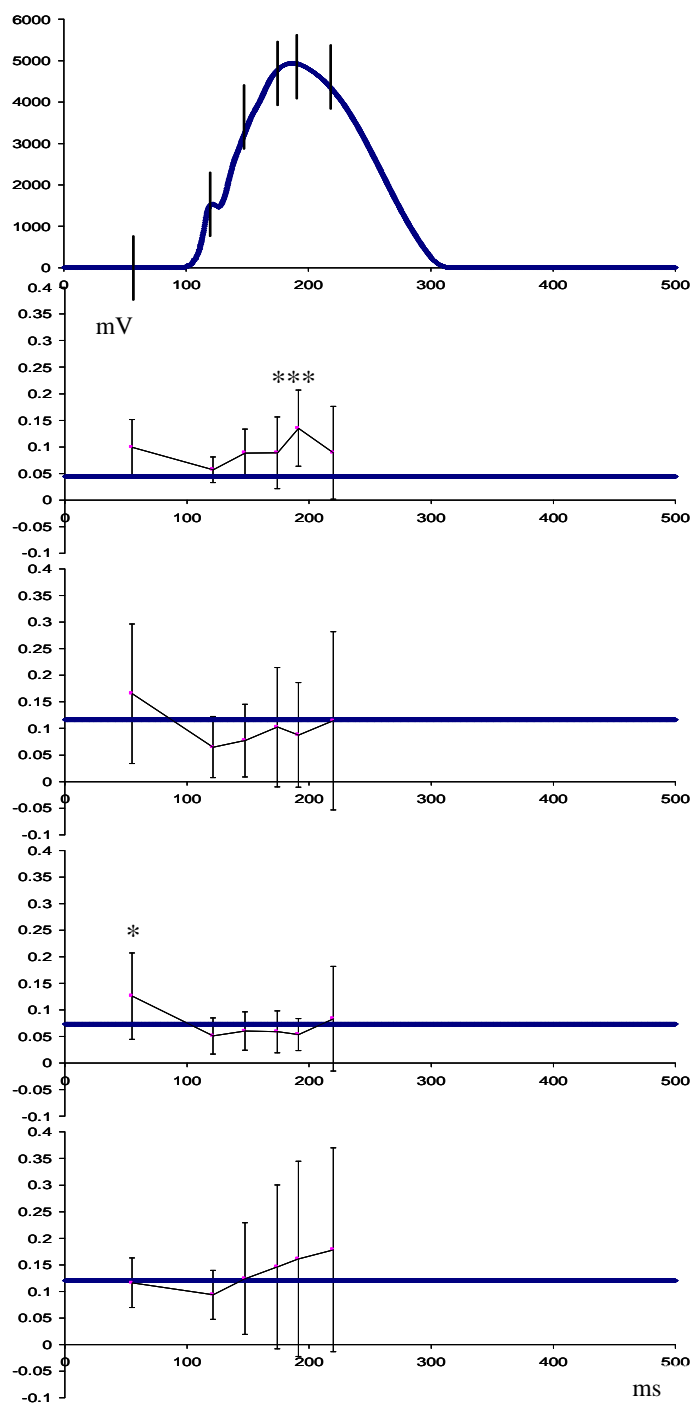For TMS values corrected with correction factor refer to figure 7.

FIGURE 7. Corrected MEP RMS values with standard deviation. Soleus, gastronemius medialis, gastrocnemius lateralis and tibialis anterior from up to down. Horizontal line is the control value of TMS. The uppermost trace is a typical ground reaction force plot from one of the subjects. Vertical lines on the force trace imply the application times of stimuli.

4   DISCUSSION

The results of this study support the conclusion by Nielsen & Petersen (1995) that corticomotoneuronal cells have a role in initiation of rapid contractions. Also the conclusion by Petersen et al. (1998) and Christensen et al. (2001) that long latency reflexes have cortical segments is supported by the results of this study. It was also evident that the pre movement silent period is produced at cortical level as suggested by Aoki et al. (2002) revealed by the corrected TMS values at stimulus 2 (15 ms after ground contact), which corresponds to the timing of PMS. Horita et al. (2002) reported that higher preactivation levels are associated with enhanced performance. The enhancement is likely produced by increased preservation of kinetic energy as elastic energy in the tendon and parallel elastic element. If the level of preactivation were determined at spinal level, the control of stretch shortening cycle would be less precise and cumbersome as there would be need for specialized circuits for many kinds of movements. Also it is known that the shortest time at which volitional corrections are possible is approximately 100 ms. The cortical segment of long latency reflexes allow the addition of cortically dependent neural drive in advance to the first volitional drive changes, which is probably significant in the efficiency of drop jump by increasing the amount of force produced.

The spinal excitability and the corticospinal excitability had different kinds of temporal patterns when the spinal excitability was measured with H-reflex amplitude and corticospinal excitability with MEP amplitude. The spinal excitability was low before ground contact the highest shortly after the beginning of ground contact and tended to descend after ground contact which is well in line with previous results (Dyhre-Poulsen et al. 1991). The corticospinal excitability tended to rise until the stimulus time 5 after which the corticospinal excitability started to fall.

The pattern of corticospinal facilitation measured with the corrected MEP values resembles the corticospinal excitability pattern found by Nielsen & Petersen (1995) of step contraction in which the subjects were instructed to produce a certain force level as fast as possible. Therefore seems to be possible that the motor strategy used for

achieving a certain submaximal force level rapidly is probably the same as the strategy used for drop jumps.

The present TMS results are in line with previous studies on the effect of voluntary activation on MEP (Taylor & Gandevia 2001, Nowicky et al. 2001) and the effect of preparation of voluntary movement on MEP (Nielsen & Petersen 1995, Hoshiyama et al. 1996, Davey et al. 1998). During the highest peak of background EMG the MEP was highest. Before the ground contact MEP was relatively high in spite of lack of background EMG, which would indicate that the preactivation is being prepared at the cortical level at that time.

When the spinal excitability was accounted for (figure 7) in the corticospinal excitability by normalizing the MEP values with the ratios of spinal excitability changes in corresponding stimulation times the rise of corticospinal excitability became even more evident, which is in line with studies by Pascual-Leone et al. (1992), Hoshiyama et al. (1996) and Leocani et al. (2000) who perceived that corticospinal facilitation begins before onset of static voluntary movement. From the corrected TMS values, which are assumed to represent the level of cortical excitability in this study, it can be seen that before ground contact the corticospinal excitability is relatively high even when EMG levels and spinal excitability levels are low. These results would indicate that the preactivation EMG is produced at cortical or supracortical level, which was also suggested by Davey et al. (1998), who didn't measure the level of spinal excitability.

The assumption that the corrected MEP values represent the cortical excitability changes is based on the conclusion by Hortobágyi et al. (2003), who found that contralateral inhibition does not affect CMEP and the MEP was even facilitated in hand muscles. Based on their results they concluded that the changes perceived on MEP had to represent cortical changes because the changes were not seen on CMEP, which shows that at least for hand muscles the MEP represents cortical excitability level when spinal excitability changes are accounted for. The spinal excitability can be measured with H-reflex measurement if it is not polluted by presynaptic inhibition.

Stein (1995) concluded that H-reflex is modulated predominantly presynaptically when H-reflex does not follow the background EMG level. In the present study the H-reflex

values followed the increase of background EMG except for the last stimulus time. It can therefore be assumed that presynaptic inhibition didn't take place in significant magnitude before the last stimulation time. Since according to Nielsen & Petersen (1995) the descending fibers are not likely to be affected by presynaptic inhibition it seems that the H-reflex values can be used as a measurement of spinal excitability and the MEP values can be used as a measurement of corticospinal excitability.

The ratio of stimulus/control value can be used as correction factor to separate the spinal excitability changes from corticospinal excitability changes to produce the cortical excitability changes as corrected MEP values in this study. Apart from the last stimulus time at which time presynaptic inhibition seemed to take place the correction used in this study should produce the true magnitude or underestimation of cortical excitability changes. According to Crone et al. (1990) the H-reflex is most sensitive to facilitation and inhibition with low stimulus intensities, which were used in this study. This would also indicate that the cortical excitability revealed in this study is not likely an overestimation although the apparent stimulation intensity at stimulus times 4 and 5 would probably lead to overestimation of cortical excitability at those stimulus times.

According to Enríquez-Denton et al. (2002) the stretch reflex is less affected by presynaptic inhibition than H-reflex, which would suggest that the correction factor of the last stimulation is too small, which is likely to lead in overestimation of cortical excitability at the last stimulation time.

According to Kerr (1978) volitional corrections are possible if movement takes more than 100 – 200 ms. Because the ground contact of drop jump lasts about 200 ms it is reasonable to assume that pre-programmed activation has major influence on drop jump and Horita et al. (2002) have made the same assumption. As only the first 120 ms after the ground contact are analyzed the results should represent the pre-programmed part of motor control in drop jump.

There was significant increase in TMS ceiling effect control MEP values compared to control trials during the drop jump trials. Because there is an increase in MEP value after drop jump trials it can be assumed that there was no ceiling effect, which would have led to underestimation of corticospinal excitability.

It is assumed that there was no significant neural or physiological fatigue induced in light of results by Dyhre-Poulsen et al. (1991) who used even higher amount of drop jumps and higher drop heights in single session and reported no deterioration of performance.

During dynamic movement it is possible that peripheral electrical stimulation intensity changes due to relative electrode movement. In this study the stimulation intensity was controlled with M-wave measurements. Although there was an apparent stimulation intensity drop at stimulation time 4 and 5 compared to control value, it could not be accounted for because the H/M-excitability curve was not measured during all parts of the jump cycle for practical reasons. It could be deduced that the intensity of stimulation was likely at the descending side of the H-reflex excitability curve with higher stimulation intensity, which is in accordance with the results of Crone et al. (1991). This deduction is of little practical value since the lower stimulation intensity was probably on the ascending side of the H-reflex excitability curve. As it is known that the form of the H-reflex excitability curve is that of an inverted parabola it is impossible to make any assumptions of the dependency of the H-reflex and stimulation intensity without a third stimulation intensity and H-reflex value from the same stimulation site in order to construct a hypothetical H-reflex excitability curve to account for the stimulation intensity drop.

Dyhre-Poulsen et al (1991) discovered that electromyographic pattern and H-relfex excitability is adapted for different kinds of jumps. They also discovered that skilled jumpers were able to reproduce jumps so that the EMG pattern remained the same between the jumps. This seems to be in line with the low standard deviation of ground reaction force measured in this study and it can be assumed that the subjects had sufficient jumping abilities to produce highly identical jumps with highly identical excitability changes of corticospinal system.

The background EMG patterns of this study were mostly in line with the results of Dyhre-Poulsen et al (1991). The EMG patterns of soleus, gastrocnemius lateralis and medialis were quite similar in shape as discovered also by Dyhre-Poulsen et al (1991). However the preactivation of gastrocnemius muscles seemed to be higher compared to

soleus prior to ground contact, which was coupled also on the MEP results. This difference could be due to much lower drop height in this experiment, which would require different kinds of stiffness properties around the ankle joint and different kinds of angle changes around the knee joint. Since gastrocnemii are biarticulate muscles the difference of drop heights could explain the different activation patterns.

For the purposes of simplicity only the RMS values were considered in discussion. The correlation between RMS and AMP values was significant however the correlation was only 0.572 which could have resulted in conflicting results. The RMS values was chosen to be presented as results according to Kiers et al. (1995) who suggested that MEP area corresponds more to the muscle twitch than MEP amplitude does and therefore is more accurate measurement of the TMS response.

In conclusion, the results indicate that the role of motor cortex in drop jumps is to initiate the muscle activation prior to the beginning of ground contact and to continue to produce activity after that. From the results it is evident that preactivation level is controlled from supraspinal, possibly cortical, level. It seems that the motor programming of drop jump takes reflex activity into account as indicated by lowering of cortical excitability during the pre movement silent period. The muscle activation is initiated with cortical drive after which the spinal drive is accounted for by lowering cortical excitability level.

ACKNOWLEDGEMENTS

the data. Author would also like to thank Mr. Taube for His advice concerning the analysis. Author would like to thank undergraduate student Christian Leukel for His valuable help with the measurements.

REFERENCES

Aoki, H., Tsukahara, R. & Yabe, K., 2002. Cortical and spinal motor excitability during the premovement EMG silent period prior to rapid voluntary movement in humans. Brain research, 949. 178 – 187.

Barker, A. T., Jalinous, R. & Freeston, I. L., 1985. Non-invasive magnetic stimulation of motor cortex. The Lancet, 8437. 1106 - 1107.

Brasil-Neto, J. P., Cohen, L. G., Panizza, M., Nilsson, J., Roth, B. J. & Hallet, M., 1992. Optimal focal transcranial magnetic activation of the human motor cortex: effects of coil orientation, shape of the induced current pulse, and stimulus intensity. Journal of clinical neurophysiology, 9(1). 132 – 136.

Brunia, C. H. M., Haagh, S. A. V. M. & Scheirs, J. G. M., 1985. Waiting to respond: electrophysiological measurements in man during preparation for a voluntary movement. Teoksessa Heuer, H., Kleinbeck, U. & Schmidt, K.-H., 1985. Motor behavior programming, control & acquistion. Saksa. 35 – 78.

Carpenter, R. H. S., 1984. Neurophysiology. Lontoo. 210 – 240, 256 – 276.

Christensen, L. O. D., Andersen, J. B., Sinkjòr, T. & Nielsen, J., 2001. Transcranial magnetic stimulation and stretch reflexes in the tibialis anterior muscle during human walking. Journal of Physiology, 531(2). 545 — 557.

Crone, C., Hultborn, H., Mazieres, L., Morin, C., Nielsen, J. & Pierrot-Deseilligny, E., 1990. Sensitivity of monosynaptic test reflexes to facilitation and inhibition as a function of the test reflex size: a study in man and the cat. Experimental Brain Research, 81. 35-45.

Davey, N. J., Rawlinson, S. R., Maskill, D. W. & Ellaway, P.H., 1998. Facilitation of a hand muscle response to stimulation of motor cortex preceding a simple reaction task. Motor control, 2(3). 241 – 250.

Day, B. L., Dressler, D., Maertens de Noordhout, A., Marsden, C. D., Nakashima, K., Rothwell, J. C. & Thompson, P. D., 1989. Electric and magnetic stimulation of human motor cortex: surface emg and single motor unit responses. Journal of physiology, 412. 449 – 473.

Di Lazzaro, V., Oliviero A., Pilato F., Saturno E., Dileone M., Mazzone P., Insola A., Tonali P.A. & Rothwell J.C., 2004. The physiological basis of transcranial motor

cortex stimulation in conscious humans. Clinical Neurophysiology, 115. 255 – 266.

Dyhre-Poulsen, P., Simonsen, E. B., & Voigt, M., 1991. Dynamic control of muscle stiffness and H reflex modulation during hopping and jumping in man. Journal of Physiology, 437. 287-304.

Enoka, R. M. 1994. Neuromechanical basis of kinesiology. USA. 123-200, 231-261.

Enríquez-Denton, M., Morita, H., Christense, L. O. D., Petersen, N., Sinkjaer, T. & Nielsen, J. B., 2002. Interaction Between Peripheral Afferent Activity and Presynaptic Inhibition of Ia Afferents in the Cat. Journal of Neurophysiology, 88. 1664 – 1674.

Gandevia, S. C., Allen, G. M., Butler, J. E. & Taylor, J. L., 1996. Supraspinal factors in human muscle fatigue: evidence for suboptimal output from the motor cortex. Journal of physiology, 490(2), 529 – 536.

Gandevia, S. C., Petersen, N., Butler, J. E. & Taylor, J. L., 1999. Impaired response of human motoneurones to corticospinal stimulation after voluntary exercise. Journal of Physiology, 521(3). 749 — 759.

Horita, T., Komi, P. V., Nicol, C. & Kyröläinen, H., 2002. Interaction between pre-landing activities and stiffness regulation of the knee joint musculoskeletal system in the drop jump: implications to performance. European journal of applied physiology, 88. 76–84.

Hortobágyi, T., Taylor, J. L., Petersen, N. T., Russell, G. & Gandevia, S. C., 2003. Changes in segmental and motor cortical output with contralateral muscle contractions and altered sensory inputs in humans. Journal or neurophysiology, 90. 2451 – 2459.

Hoshiyama, M., Yoshikiro, K., Koyama, S., Watanabe, S., Shimojo, M. & Kakigi, R., 1996. Reciprocal change of motor evoked potentials preceding voluntary movement in humans. Muscle & Nerve, 19. 125 – 131.

Kamen, G., 2004. Reliability of motor-evoked potentials during resting and active contraction conditions. Medicine & science in sports & exercise, 36(9). 1574 – 1579.

Kato, T., Takeda, Y., Tsuji, T. & Kasai, T., 2003. Further insights into post-exercise effects on H-reflexes and motor evoked potentials of flexor carpi radialis muscles. Motor control, 7(1), 82 – 99.

Kerr, B., 1978. Task factors that influence selection and preparation for voluntary movements. Teoksessa Stelmach, G. E. (toim.), 1978 Information processing on motor control and learning. 62-64.

Kiers, L., Clouston, P.,Chiappa, K. H. & Cros, D., 1995. Assessment of cortical motor output: compound muscle action potential versus twitch force recording. Electroencephalography and clinical Neurophysiology, 97. 131 - 139.

Klapp, S. T., 1996. Reaction Time Analysis of Central Motor Control. Teoksessa Zelaznik, H. N. (toim.) Advances in motor learning and control. USA, 13-31.

Latash, M. L. 1998. Neurophysiological basis of human movement. USA. 55-71, 85, 98-101.

Leocani, L., Cohen, L. G., Wassermann, E. M., Ikoma, K. & Hallet, M., 2000. Human corticospinal excitability evaluated with transcortical magnetic stimulation during different reaction time paradigms. Brain, 123. 1161 – 1173.

Liepert, J., Kottebra, S., Tegenthoff, M. & Malin, J-P., 1996. Central fatigue assessed by transcranial magnetic stimulation. Muscle & nerve, 19. 1429 – 1434.

Malmivuo, J., 1992. Distribution of electrical fields in the tissue.Teoksessa toim. Suihko, V. & Eskola, H., 1992. Proceedings of the first Ragnar granit symposium. Electrical and magnetic stimulation of motor nervous system. Tampere university of technology, Ragnar granit institute. 5 – 30.

Meunier, S. & Pierrot-Deseilligny, E.,1998. Cortical control of presynaptic inhibition of Ia afferents in humans. Experimental brain research, 119. 415 – 426.

Morita, H., Petersen, N., Christensen, L. O. D., Sinkjær, T. & Nielsen., J., 1998. Sensitivity of H-reflexes and stretch reflexes to presynaptic inhibition in humans. Journal of neurophysiology, 80. 610 – 620.

Nielsen, J. & Petersen, N., 1995. Changes in the effect of magnetic brain stimulation accompanying coluntary dynamic contraction in man. Journal of Physiology, 484(3). 777–789.

Nowicky, A. V., McGregor, A. H. & Davey, N. J., 2001. Corticospinal control of human erector spinae muscles. Motor control, 5(3). 270 – 280.

Pascual-Leone, A., Valls-Solé. J., Wasserman, E. M., Brasil-Neto, J., Cohen, L. G. & Hallet, M., 1992. Effects of focal transcranial magnetic stimulation on simple reaction time to acoustic, visual and somatosensory stimuli. Brain, 115. 1045 – 1059.

Pensini, M. & Martin, A., 2004. Effect of voluntary contraction intensity on the H-reflex and V-wave responses. Neuroscience Letters, 367. 369–374.

Petersen, N., Christensen, L. O. D., Morita, H., Sinkjaer, 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.

Purves, D., Fitzpatrick, D., Augustine, J. G., Katz, L.C., Williams, M. S., McNamara, J. O. & Lamantia, A-S., 2001. Neuroscience. Osoitteessa http://www.ncbi.nlm.nih.gov/books/bv.fcgi?rid=neurosci.figgrp.1170 29.11.2004.

Reid, V., 2003. Transcranial magnetic stimulation. Physical medicine & rehabilitation clinics of North America, 14. 307 – 325.

Ruohonen, J., 1997. Basic principles. http://www.biomag.hus.fi/tms/basic.html 5.10.04.

Ruohonen, J., 1999. Safety of TMS. http://www.biomag.hus.fi/tms/safety.html 5.10.04.

Sage, G. H., 1984. Motor learning and control. Dubuque, Iowa. 24, 59-60.

Stein, R. B., 1995. Presynaptic inhibition in humans. Progress in Neurobiology, 47. 533 –544.

Taylor, J. L., Butler, J. E., Allen, G. M. & Gandevia, S. C., 1996. Changes in motor cortical excitability during human muscle fatigue. Journal of physiology, 490 (2). 519 – 528.

Taylor J. L., Butler J. E., Gandevia S. C., 1999. Altered responses of human elbow flexors to peripheral nerve and cortical stimulation during a sustained maximal voluntary contraction. Experimental Brain Research, 127. 108–115.

Taylor, J. L. & Gandevia, S. C, 2001. Tranacranial magnetic stimulation and human muscle fatigue. Muscle & nerve, 24(1). 18 - 29.

Viitasalo, J. T., Salo, A. & Lahtinen, J., 1998. Neuromuscular functioning of athletes and non-athletes in the drop jump. European journal of applied physiology, 78. 432-440.

Walter, C. B., 1988. The influence of agonist premotor silence and the stretch-shortening cycle on contractile rate in active skeletal muscle. European journal of applied physiology, 57. 577 – 582.

Wassermann, E. M.,1998. Risk and safety of repetitive transcranial magnetic stimulation: report and suggested guidelines from the international workshop on the safety of repetitive transcranial magnetic stimulation, June 5–7, 1996. Electroencephalography and clinical Neurophysiology, 108. 1 - 16.

Wolfgang, N. L. & Nordlund, M. M., 2002. Central fatigue and motor cortical excitability during repeated shortening and lenghtening actions. Muscle & nerve, 25(6). 864 – 872.

Ziemann, U., Rothwell, J. C. & Ridding, M. C., 1996. Interaction between intracortical inhibition and facilitation in human motor cortex. Journal of physiology, 496(3). 873 – 881.