Differential modulation of spinal and corticospinal excitability during drop jumps

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Abstract

Previously it was shown that spinal excitability during hopping and drop jumping is high in the initial phase of ground contact when the muscle is stretched, but decreases towards take off. To further understand motor control of stretch-shortening cycle this study aimed to compare modulation of spinal and corticospinal excitability at distinct phases following ground contact in drop jump. Motor evoked potentials (MEPs) induced by transcranial magnetic stimulation (TMS) and H-reflexes were elicited at the time of the short (SLR), medium (MLR) and long (LLR, LLR$_2$) latency responses of the soleus muscle (SOL) after jumps from 31 cm height. MEPs and H-reflexes were expressed relative to the background EMG activity.

H-reflexes were highly facilitated at SLR (172%) and then progressively decreased (MLR=133%; LLR=123%; LLR$_2$=110%). TMS showed no effect at SLR, MLR and LLR whereas MEPs were significantly facilitated at the LLR$_2$ (122%; p=0.003). Background EMG was highest at LLR and lowest at LLR$_2$.

Strong H-reflex facilitation at the beginning of the stance phase indicated significant contribution of Ia-afferent input to the $\alpha$-motoneurons during this phase which then progressively declined towards take off. Conversely, corticospinal excitability was exclusively increased at the phase of push off (LLR$_2$, ~120 ms). It is argued that corticomotoneurons increased their excitability at LLR$_2$. At LLR (~90 ms), Ia-afferent transmission as well as corticospinal excitability was low whereas background EMG was high. Therefore it is speculated that other sources, presumably subcortical in origin, contributed to the EMG activity at LLR in drop jumps.
Introduction

Muscular activation during the drop jump is organized in a stretch-shortening cycle. The efficiency of the stretch-shortening cycle is dependent on the immediate transfer from the pre-activated and eccentrically stretched muscle-tendon complex to the concentric push-off phase. Reflex contribution induced by stretch of the antigravity muscles during touch down (eccentric phase) is thought to enhance muscular stiffness and therefore increase the performance during the concentric phase when compared to isolated concentric action (7; 14; 63). In hopping and during drop jumping it was shown that the H-reflex excitability was very low at take off, still low in the flight phase but increased before touch down. During the stance phase, H-reflexes remained facilitated but decreased again before take off (9; 41; 63). The increase of the Ia-transmission towards touch down supports the assumption of anticipatory spinal stretch reflex contribution to the EMG in hopping and drop jumping. Further studies showed that the reflex activity shortly after touch down is specifically modulated dependent on the jump condition: Reflex excitability was enhanced in jumps from “low” heights (20 to 60 cm) compared to jumps from “great” heights (76 to 80 cm) (29; 32). This adaptation in the reflex behavior was argued to be pre-programmed by supraspinal centers (32). Similarly, centrally pre-programmed muscular activity was considered to be important for the pre-innervation, the reflex modulation and the stiffness regulation during stretch-shortening cycle and landing movements in men and monkeys (8; 18; 19). Therefore, it is assumed that supraspinal centers not only initiate jumping and landing movements but also pre-program part of the muscular activation pattern after touch down. The question now is, whether and how corticospinal pathways contribute to this muscular activity after ground contact.

In stretch reflex experiments during sitting and walking as well as in a postural regulation task it was shown that for the lower leg muscles corticospinal excitability was low at the time of the short- (SLR) and the medium-latency response (MLR) but highly facilitated at later reflex components (long-latency responses = LLR) (3; 47; 57). If motor control during drop jump is similar we would expect cortical contribution to be apparent at the time of the LLR but not at SLR and MLR. However, drop jump is a self-initiated movement whereas muscular responses induced by stretch or postural disturbance are not. In other words, the initial muscular activation following postural disturbance is derived from spinal α-motoneurons activated by stretch of the muscle spindles. Drop jump on the other hand allows supraspinal centers to accurately predetermine the time of ground contact and thus the instance of muscular stretch.
Therefore, it can be speculated that during drop jump, pre-programmed activation of corticospinal pathways may contribute to the muscular activity at the time of the SLR, MLR and LLR. The aim of the present study was to address this issue by comparing changes in spinal and corticospinal excitability during the drop jump at distinct phases after ground contact (SLR after ~ 45 ms, MLR ~ 70 ms, LLR ~ 90 ms and LLR2 ~ 120 ms). For this purpose, peripheral nerve stimulation and transcranial magnetic stimulation (TMS) were applied so that H-reflexes and motor evoked potentials (MEPs) coincided with the respective reflex peaks in the soleus muscle.

Methods and Materials

Subjects

Fifteen healthy subjects without record of neurological or orthopedic disorder participated in the main experiment of the present study. However, as 5 subjects did not fulfill the criteria for constant electrical stimulation intensity (see data analysis) only 10 subjects were included for later analysis (3 females; 7 males; 23 ± 1.5 years). Seven subjects participated in an additional test protocol. Three of them took also part in the main protocol. Before testing, all subjects were informed about the experiments and gave written consent about the experimental procedure. The experiments were run in accordance with the Declaration of Helsinki and approved by the local ethics committee.

General experimental procedure

Following a 5 minute warm-up (hopping and stretching) subjects performed drop jumps from 31 cm. Before every jump, both arms were held akimbo, the left leg remained stable in order to secure upright stance whereas the right leg was lifted and projected in front of the body. The jump off was performed with the left leg to keep the right leg relaxed during this phase. Subjects were instructed to touch down on both feet and jump off the ground as quickly as possible while bending the knees as little as possible. Subjects were instructed to keep the jumping procedure similar throughout the whole experiment. At touch down, the ankle plantarflexor muscles were stretched and several responses could be observed in the SOL electromyogram (EMG). Response latencies were calculated with respect to the moment of
touch down, which was measured by a force plate (AMTI®, Watertown, Massachusetts, USA). According to their latencies, the first three response peaks were termed short (SLR), medium (MLR) and long latency response (LLR). The fourth peak in the EMG was assumed to be a later component of the LLR and subsequently called LLR₂. The onsets and peaks of the SLR, MLR, LLR and LLR₂ were determined on the basis of the background EMG activity and previously reported latencies and durations of SLR, MLR, LLR and LLR₂ (15; 23; 47; 54; 57). Stimulation was timed, so that the motor-evoked potentials (MEPs; elicited by TMS) as well as the peaks of the soleus H-reflexes (elicited by electrical stimulation) were triggered to coincide with the peaks of SLR, MLR, LLR or LLR₂ (see figure 1). TMS as well as peripheral nerve stimulation were applied with low and high stimulation intensity. Consequently, subjects were tested in three conditions during drop jumping:

1) **Drop jumps alone** to elicit SLR, MLR, LLR and LLR₂.

2) **Drop jumps + electrical stimulation** (soleus H-reflexes) to assess spinal excitability at SLR, MLR, LLR and LLR₂:
   a) Stimulation intensity adjusted to elicit H-reflexes of 20% of M_max during stance (“low intensity stimulation”)
   b) Stimulation intensity adjusted to elicit M-waves of 50% of M_max during stance (“high intensity stimulation”)

3) **Drop jumps + TMS** to assess corticospinal excitability at SLR, MLR, LLR and LLR₂ peaks:
   a) Stimulation intensity subthreshold to elicit MEPs during stance (0.9 MT; in accordance with Day et al. (5))
   b) Stimulation intensity of 1.2 MT to elicit MEPs during stance (suprathreshold stimulation at MLR, LLR and LLR₂; in accordance with Petersen et al. (47)).

TMS- and peripheral nerve stimulation-experiments were conducted on two separate days to avoid fatigue. At each test day, subjects performed approximately 120 drop jumps. After every 10 jumps, subjects were instructed to rest for 2 minutes. After familiarization with the jumping procedure, 15 jumps without stimulation were performed in order to determine the latencies for the SLR, MLR, LLR and LLR₂. Based on these latencies and the latencies of the MEP and the H-reflex, stimulation instances were calculated so that the peak of the MEP or the H-reflex coincided with the peak of the SLR, MLR, LLR and LLR₂, respectively (methodological approach in accordance with Petersen et al. (47); Taube et al. (56; 57); Leukel et al. (32)). For each stimulation point (SLR, MLR, LLR and LLR₂) 15 jumps with
subthreshold TMS and 15 jumps without stimulation took place in a pseudo-randomized order, i.e. in each “round” all stimulation points as well as the control trials were measured in a random sequence. This procedure ensured that all stimulation points and control trials were subject to the same potential disturbances (fatigue, electrode displacement, sweating, etc.). The 15 trials of every condition were averaged afterwards based on the trigger signal, which had been derived from the first increase in ground reaction force (ground contact). Additionally, 5 jumps with suprathreshold TMS were applied at MLR, LLR and LLR2. Similarly, 15 jumps with peripheral nerve stimulation and 15 jumps without stimulation were recorded at each stimulation point. To monitor changes in the stimulation intensity, 5 trials with higher stimulus intensity were performed at each stimulation point. As small M-waves are not very sensitive to changes in the intensity, the stimulation intensity was adjusted to elicit M-waves of 50% of the maximum M-wave ($M_{\text{max}}$). At this intensity, the recruitment curve is steepest for the M-wave and therefore, it is most sensitive to small alterations in the stimulus intensity (49).

**EMG recordings**

EMGs were recorded from the soleus (SOL), tibialis anterior (TA), and gastrocnemius medialis muscles of the right leg. AgAg-Cl surface electrodes (Hellige, diameter 9 mm, centre to centre distance 3 cm) were filled with electrode jelly and attached to the skin in line with the presumed direction of the underlying muscle fibers. The reference electrode was placed on the anterior aspect of the tibia. EMG signals were amplified (×1000), bandpass-filtered (30 - 1000 Hz) and sampled at 4 kHz.

**H-reflex recording**

H-reflexes were elicited with an electrical stimulator (DS 7, Digitimer Ltd., UK) from the right SOL muscle by stimulating the posterior tibial nerve. The stimulus was a square-wave pulse of 1 ms duration. The anode, a 10 × 5 cm dispersal pad, was fixed on the anterior aspect of the knee just underneath the patella. The cathode (2 cm in diameter) was placed in the popliteal fossa and moved stepwise until the best position for eliciting and H-reflex was found. It was ensured that stimulation evoked no response in the TA muscle. The cathode was fixed with rigid tape. An H-reflex recruitment curve was recorded during upright stance. As the sensitivity of the H-reflex to facilitation and inhibition varies with respect to the size of
the control H-reflex, the stimulation intensity was adjusted to elicit H-reflexes with the size of 20% of $M_{\text{max}}$ (Crone et al., 1990). This ensured that the same portion of the motor pool was activated in each subject. In all cases, this resulted in an H-reflex being on the ascending slope of the H-reflex recruitment curve. In some trials (see general experimental procedure 2b) stimulation intensity was increased to elicit M-waves of 50% of $M_{\text{max}}$ to obtain a sensitive measure for changes in the stimulation intensity (49).

**TMS**

TMS was applied over the left motor cortex using a Magstim 200 (Magstim Company Ltd, Dyfed UK) with a 90 mm circular coil. The stimulus waveform was monophasic and had a pulse width of 200 µs. For each subject, the initial coil position was chosen so that the centre of the coil was situated approximately 0.5 cm posterior to the vertex and over the midline. The final position for the stimulation was determined by moving the coil anterior and left from the vertex while MEP size of SOL and TA were monitored on an oscilloscope. The optimal position for eliciting MEPs in the SOL muscle during stance with minimal stimulator output was marked on the scalp with a felt pen. Motor threshold (MT) of the SOL was determined during stance. MT was defined as the intensity at which an MEP reached 150 µV in 3 out of 5 trials (30). The threshold was set to 150 µV as some subjects showed relatively high tonic muscular activation during stance (around 100 µV). Stimulation intensity during jumps was adjusted to 0.9 MT (subthreshold trials). At MLR, LLR and LLR$_2$ corticospinal excitability was additionally assessed with 1.2 MT (suprathreshold trials; see general experimental procedure). At SLR, stimulation with 1.2 MT disrupted the jumping behavior of most subjects so that they were unable to absorb the impact of touch down smoothly which consequently caused discomfort.

A crucial point for TMS-measurements during highly dynamic movements is the fixation of the coil to the scalp. To ensure a constant stimulation site throughout drop jumps the coil was fixed to a custom designed helmet (Petzl, Garmisch-Partenkirchen, Germany) which provided a flexible adjustment to head-sizes from 53 cm to 63 cm. The helmet was additionally secured by straps to the chin and to the back of the head. The coil could be moved relative to the helmet through a pin-jointed system made of light plastic, which guaranteed a highly flexible handling. To minimize any forces acting on the helmet, the cable of the coil was separately fixed to a backpack-like system carried by the subject. In preliminary tests with a high speed camera it was ensured that there was minor movement of the coil relative to the skull during
drop jump (0 – 1 mm). For measurements during jumping, the “helmet construction” therefore provided several advantageous compared to the halo vest used in the same lab before, which was introduced by Schubert et al. (52): First, subjects could move their head relative to their trunk, which was a prerequisite to perform drop jumps. Second, the carrying system was lighter than the harness. Third, the coil was neatly supported and fixed in every direction to counteract the high forces acting on the coil.

**Additional protocol**

An additional protocol was performed in order to evaluate the modulation of the H-reflex and the MEP in response to changes of the background EMG. Subjects were asked to execute isometric ramp and hold contractions between 0 and 100% of their maximal voluntary contraction (MVC). Before every contraction, the subject was instructed how strong to contract (not at all, mild, 50%, submaximal, maximal, stronger/less strong as before, etc.). H-reflexes and MEPs were elicited as soon as the force curve showed a plateau. The stimulation intensities resembled the ones used in the drop jump condition, i.e. H-reflexes were adjusted to 20% of \( M_{\text{max}} \) and the TMS intensity to 0.9 MT. Each subject performed 40 contractions with electrical stimulation and 40 contractions with TMS. Additionally, each subject performed 10 drop jumps without any stimulation to assess the background EMG activity.

**Data analysis**

The size of the H-reflexes and MEPs were determined by calculating root mean square (RMS) values over a 20 ms time frame around SLR, MLR, LLR and LLR\(_2\). The RMS values were chosen according to Kiers et al. (25). They showed that the MEP area corresponded better to the muscle twitch than the MEP amplitude and therefore, was a more accurate measure of the muscular response. Regarding the analysis of SOL stretch and H-reflexes it was shown that there were no differences whether choosing reflex amplitude or area (53; 63). Muscular responses with stimulation (peripheral nerve stimulation or TMS) were expressed as a percentage of the corresponding background EMG activity (RMS-value analyzed in the respective time window) at SLR, MLR, LLR and LLR\(_2\).

In order to ensure comparable stimulation intensity of the peripheral nerve stimulation, the intensity was adjusted to elicit M-waves of 50% of \( M_{\text{max}} \) (M-wave\(_{50\%}\)) during stance. Subjects who showed significant deviations of their M-wave\(_{50\%}\) amplitudes between SLR, MLR, LLR
and LLR2 were excluded from any analysis. For this purpose, the median of the M-wave50% amplitudes of SLR, MLR, LLR and LLR2 was calculated. Subjects were excluded if the mean of one of their M-wave50% deviated more than ± 12.5% of Mmax (recorded during stance) from this median. Additionally, the H-reflexes were analyzed with respect to their corresponding M-waves on a trial to trial basis. The effect of the electrical stimulation was calculated in the following way: The background EMG (mean of all trials without stimulation) was subtracted from the H-reflex (mean of all trials obtained with peripheral nerve stimulation) and then normalized to the corresponding M-wave50% (formula: \[(HR – bEMG) / M-wave50%\] *100).

In the additional test protocol, RMS values were calculated 20 ms around each H-reflex; Mmax and MEP peak as well as in a 20 ms time interval prior to the stimulation. H-reflexes and MEPs were normalized with respect to Mmax. The background EMG was normalized to the maximal EMG activity obtained at 100% MVC. For better visualization data obtained during 60 to 100% MVC were z-normalized according to the mean and the standard deviation (S.D.) of the respective SOL activation obtained with peripheral nerve stimulation or TMS; e.g. a z-transformed H-reflex value of -10 is a value of 10 S.D. below the mean of the H-reflexes obtained during contractions between 60 to 100% MVC.

Statistics

To evaluate differences in the modulation of spinal and corticospinal excitability during drop jump, H-reflexes and MEPs were expressed in percentage of the background EMG. In order to reveal differences between the background EMG activity and trials with H-reflexes or MEPs, a one way ANOVA was accomplished. The influence of different TMS intensities on the EMG response at each stimulation point was evaluated with a two way repeated measures ANOVA [3 (stimulation point) * 2 (stimulus intensity)]. In case of significant F-values (p < 0.05) differences between values at selected points in time or between groups were compared by Bonferroni corrected paired two-sided tests (Student’s t-test). Data are presented as group mean values ± SEM. In the additional protocol, slopes of linear regression were compared using the Z-test for parallelism (26).


Results

Reflex peak latencies and EMG recording

Reflex peak latencies during drop jump were 45.6 ± 3.5 ms (SLR), 68.9 ± 6.4 ms (MLR), 91.9 ± 4.9 ms (LLR) and 118.1 ± 5.5 ms (LLR_2), respectively (an example is illustrated in figure 1). Background EMG activity of TA did not differ between H-reflex and TMS trials (table 1). Similarly, SOL background EMG activity (RMS values measured 20 ms around each reflex peak) was almost identical in H-reflex trials and TMS trials (see table 1) but showed differences between stimulation points (ANOVA p = 0.027; LLR versus LLR_2, p = 0.028).

Peripheral nerve stimulation

Electrical stimulation with low intensity (see general experimental procedure 2a) revealed facilitation of the muscular responses at SLR, MLR and LLR compared to the EMG responses without stimulation when expressed with respect to the background EMG activity (ANOVA “EMG activity” p = 0.002; figure 2A). This facilitation progressively decreased at MLR, LLR and LLR_2 (ANOVA “stimulation point” p = 0.01; figure 2A). Electrical stimulation with intensity sufficient to elicit an M-wave of 50% of M_max (M-wave_50%; see general experimental procedure 2b; M_max see figure 2B) showed a very similar modulation (H-reflex modulation see figure 2A). Additionally, the H-reflexes were analyzed with respect to their corresponding M-waves on a trial to trial basis (formula: [(HR – bEMG) / M-wave_50%] *100). This analysis revealed also a progressive decrease in H-reflex size from SLR (47 ± 5%) to MLR (36 ± 7%), LLR (24 ± 8%) and LLR_2 (10 ± 11%) (ANOVA “stimulation point” p = 0.023).

TMS

MEPs in the soleus muscle

Subthreshold (0.9 MT) and suprathreshold (1.2 MT) TMS to the SOL had no effect at SLR (SLR only measured with 0.9 MT; see method section “TMS”), MLR and LLR when compared to the background EMG (ANOVA “EMG activity” not significant (n.s.)). At LLR_2,
TMS significantly facilitated the SOL response (figure 2C; ANOVA "stimulation point" \( p = 0.046 \)). The augmentation at LLR\(_2\) obtained with suprathreshold TMS (1.2 MT) exceeded the one recorded with subthreshold TMS (140 ± 12 % versus 122 ± 4 %). However, this difference was not significant as only 7 out of 10 subjects revealed greater MEPs with enhanced stimulation intensity (ANOVA “intensity” n. s.; “stimulation point” * “intensity” n. s.).

### MEPs in the TA muscle

TMS subthreshold to evoke MEPs in the SOL muscle during stance (0.9 MT) was sufficiently high to elicit MEPs in the TA (figure 3). The same stimulation intensity applied during jumping further enhanced MEPs in the TA (ANOVA “EMG activity” \( p = 0.021 \); figure 2D and 3). Thereby, absolute MEP-amplitude was greatest at SLR and progressively decreased at MLR, LLR and LLR\(_2\) (figure 3). Taking background EMG activity into account, comparable facilitation occurred at SLR, MLR, LLR and LLR\(_2\) (see figure 2D; ANOVA “stimulation point” n. s.). An increase in the stimulus intensity enhanced MEPs in the TA to a similar extent at MLR, LLR and LLR\(_2\) (see figure 2D; ANOVA “stimulus intensity” \( p = 0.038 \); “stimulus intensity” * “stimulation point” n. s.; “stimulation point” n. s.).

### Additional protocol

H-reflexes as well as MEPs increased with increasing background EMG (\( r = 0.49; p < 0.01; r = 0.54; p < 0.01 \); see figure 5A/C). The slopes of the two regression lines were statistically not different (\( Z = 1.9; p > 0.05 \)). Considering background EMG levels with comparable activation as in drop jumps (60 to 100% MVC), neither H-reflexes nor MEPs correlated with the background EMG activity (\( r = -0.07; p = 0.55; r = -0.02; p = 0.88 \); see figure 5B/D). The slopes of the regression lines were not different (\( Z = -0.5; p > 0.05 \)). The facilitatory effects of peripheral electrical and transcranial magnetic stimulation were additionally expressed in percentage of the background EMG. Four groups were formed according to the EMG activity observed during drop jumps at SLR (= 70-80% MVC), MLR (= 80-95% MVC), LLR (= 100% MVC) and LLR\(_2\) (= 60-70% MVC). Peripheral nerve stimulation facilitated the EMG response about 268 ± 53% at background EMG levels adjusted to the ones seen at the SLR during drop jump, about 226 ± 69% when matched to the MLR, about 199% ± 70% when matched to the LLR and about 266 ± 61% when matched to the LLR\(_2\). During voluntary contractions, MEPs expressed in percentage of the background EMG were 239 ± 45%, 219 ±
43%, 206 ± 36% and 223 ± 35% when the background EMG was matched to the responses (SLR, MLR, LLR and LLR₂) during jumping without stimulation.

**Discussion**

The present results show a differential modulation of the H-reflex and the MEP during the contact phase of drop jump. To ensure that this modulation was due to mechanisms at the spinal and cortical level and not simply caused by differential responses of H-reflexes and MEPs to changes in the background EMG activity, an additional test protocol was executed. In accordance with the literature both, H-reflexes and MEPs were positively correlated with the background EMG (1; 13; 31; 34; 39; 51; 61). However, on closer examination it became apparent that these correlations were strongly developed at weak contraction levels but almost disappeared at higher levels. As a consequence, there was no further increase in the H-reflex and the MEP size from 60 to 100% of MVC. Concerning the soleus H-reflex, Löscher et al. (33) reported a similar modulation in 3 out of 8 subjects at high contraction levels: H-reflexes increased up to 30-50% of MVC and then remained at a constant amplitude. To our knowledge, modulation of the MEP at high contraction levels has not been reported for the soleus muscle, yet. In the present study, responses to peripheral nerve stimulation and TMS were similarly affected by alterations in the background EMG for soleus, indicating that there was no stimulus specific effect. This may surprise as the MEP involves multiple descending volleys which may cause motoneurons to discharge more than once. At the same time, dispersed volleys can lead to phase cancellation of action potentials which in turn reduce the size of the MEP (24). In contrast, the H-reflex is elicited by a single synchronized volley and in this respect resembles the cervicomedullary motor evoked potential (CMEP) evoked by electric or magnetic stimulation at the cervicomedullary junction. CMEPs evoked in biceps brachii and brachioradialis were shown to behave comparable to MEPs for isometric contractions of 50, 75, 90 and 100% MVC (36). The authors concluded that alterations in the contraction strength lead to changes in the excitability of the motoneuron pool which affected different stimuli similarly. Our results support this assumption. Therefore, it is unlikely that differences in the H-reflex and MEP modulation primarily relied on different background EMG levels at SLR, MLR, LLR and LLR₂.
Modulation of the SOL H-reflex

Previous studies investigating spinal excitability during hopping and drop jumping revealed a phase dependent modulation of the H-reflex: The H-reflex excitability was high during the stance phase but decreased before take off and stayed low during the flight phase (9; 41; 63). Our results support these earlier observations, also showing facilitated H-reflexes in the early stance phase (SLR, MLR) which then decrease towards take off (LLR, LLR\textsubscript{2}). Thereby, a similar modulation was seen with stimulation intensity adjusted to evoke H-reflexes of 20% of M\textsubscript{max} and with stimulation intensity sufficient to elicit M-waves of approximately 50% of M\textsubscript{max}. It has to be considered that in the latter case the H-reflex cannot be regarded to arise solely from Ia-afferent input and had either reached a plateau or lay on the descending part of the recruitment curve. Consequently, it was presumably subject to substantial antidromic collision. However, a similar modulation with low and high stimulus intensity suggests that most of the reflex modulation could be attributed to changes in the Ia-afferent transmission onto the SOL motoneuron pool. The functional significance of enhanced Ia-afferent transmission in the early stance phase may be that impulses from the Ia-afferents can add a reflex response on top of the ongoing EMG activity or produce a peak by synchronizing muscular activation in the already active SOL muscle at the time of touch down (9). In this way, the stretch reflex response most likely contributes to the enhanced efficiency of the stretch-shortening cycle compared to concentric action (7; 14). The progressive decline of the H-reflex amplitude relative to the background EMG at MLR, LLR and LLR\textsubscript{2} suggests that muscular activity at the later points is not as depending on Ia-afferent input as at the time of the SLR. Functionally, reduction in Ia-afferent transmission thus may help to enhance movement control and efficiency by preventing poorly timed muscular activation arising from spinal reflex circuits. Supporting evidence comes from Funase et al. (12) who reported time consistent appearance of the SLR at different hopping frequencies but could not find a time-locked long-latency EMG response. They argued that the overall EMG activity during hopping is composed of a time consistent short-latency reflex component induced by landing and a “voluntary component” for launching take-off, which is pre-programmed and triggered dependent on the frequency. Thus, if a strong contribution of Ia-afferents to muscular activity was present during the entire stance phase this would probably interfere with such a flexible modulation of the “voluntary” component. However, it has to be mentioned that other factors than changes in the Ia-transmission may have influenced the size of the H-reflex: Muscle
length was shown to affect the H-reflex size (44; 46) as well as activity in upper leg muscles like the biceps femoris or the rectus femoris (20; 28; 40). Furthermore, cutaneous afferent input can also alter the excitability of the H-reflex (27). As drop jump is a highly dynamic movement involving many muscles and sensory information from numerous different sources it was not possible to determine the main mechanism(s) which is/are responsible for the H-reflex modulation in the present study. However, irrespective of the exact underlying mechanism, our results as well as observations from previous stretch-shortening cycle studies suggest that spinal reflex excitability assessed via the Ia-afferents is relatively high at touch down (SLR) and reduced towards take off (LLR2).

**Modulation of the MEP**

To test for cortical contribution, Day et al. (5) superimposed MEPs on stretch reflex responses of the flexor digitorum profundus muscle in the forearm. They observed that only long-latency reflex responses were facilitated by TMS whereas short latency reflex responses were not. Similarly, Petersen et al. (47) observed facilitatory responses in the TA muscle following magnetic stimulation when MEPs were triggered to coincide with the LLR but not when MEPs were superimposed on the SLR or the MLR. Additionally, no extra facilitation was seen with transcranial electrical stimulation. The authors concluded that the LLRs of the flexor digitorum profundus and the TA are – at least partly – mediated by a transcortical reflex. Very recently, such a transcortical reflex loop was also shown for the LLR of the SOL muscle during a postural regulation task (57).

In the present study, MEPs were superimposed on muscular activation peaks following touch down in order to assess changes in the corticospinal excitability during drop jump. Stimulation with *subthreshold* TMS during drop jump had no effect on SLR (~ 45 ms after touch down), MLR (~ 70s) and LLR (~ 90 ms) of the SOL. Interestingly, *suprathreshold* TMS evoked no facilitation of the EMG responses at MLR and LLR either. Only the LLR2, which occurred after approximately 120 ms, was significantly facilitated following both, sub- and suprathreshold stimulation. This may indicate enhanced corticospinal excitability at LLR2. However, it has to be pointed out that the rise time of the MEP is long enough to allow several pathways to contribute to its facilitation. Accordingly, the modulation of motor responses evoked by TMS reveals only the net effect of the activation of several different corticospinal projections (48). Consequently, excitability changes assessed by TMS alone can
hardly be attributed to specific structures of the CNS (43). Thus, the facilitation of MEPs at LLR\textsubscript{2} observed in our study may be caused by changes in cortical, subcortical and/or spinal excitability. Based on the H-reflex results obtained in this and previous studies (9; 41), it seems unlikely that changes at the spinal level were responsible for enhanced efficiency of the TMS at LLR\textsubscript{2}. Although presynaptic mechanisms may have caused the reduced H-reflex excitability at LLR\textsubscript{2}, this would not explain facilitated MEPs at this time. Similarly, the on average slightly lower background EMG activity at LLR\textsubscript{2} should have influenced – if at all – H-reflexes and MEPs in equal measure. As spinal mechanisms fail to explain the facilitation of the MEP at LLR\textsubscript{2}, supraspinal centers most likely accounted for the MEP modulation during drop jump. As TMS is believed to activate the corticomotoneurons indirectly (trans-synaptically or at the axon hillhock) and the corticospinal fibers are considered to be free from presynaptic inhibition (21; 42) changes in the MEP size which can not be ascribed to changes in the motoneuronal excitability are most likely caused by alterations in the excitability of cortical interneurons and/or of the corticomotoneurons themselves. Thus, the same stimulus to the motor cortex at LLR\textsubscript{2} probably activated a significantly greater portion of cortical neurons than at SLR, MLR or LLR.

**Organization of the long-latency response during drop jump**

The present results indicated increased corticospinal excitability at the LLR\textsubscript{2}, which occurred after approximately 120 ms. Based on studies using TMS to evaluate cortical contribution following muscle stretch, the corticospinal influence would have been expected earlier – at the time of the LLR around 90 ms. In the TA enhanced corticospinal excitability was reported to occur after 85 ms with a maximum at 95 ± 9 ms following stretch to the TA during sitting (47) and from 90 ms onwards with a peak at 110 ms following stretch applied during walking (3). Similarly, contribution of transcortical pathways in the SOL muscle during posterior translation of the support platform was evident after 86 to 100 ms (57). These studies showed that there is sufficient time for a transcortical loop within the first 100 ms following muscle stretch. It has to be emphasized that the present study did not determine the exact onset of enhanced corticospinal excitability in each subject because no intermediate measurements were made between the LLR and the LLR\textsubscript{2}. Nevertheless, it seems peculiar that the time for cortical involvement via the corticospinal tract is not apparent at the LLR as drop jump constitutes a voluntary initiated movement in which the instance of ground contact – and thus the time when the muscle is stretched – can be accurately predicted (8; 37; 50). One
explanation might be that movements of the coil or the brain avoided activation of corticomotoneurons. If this was the case, TA-MEPs recorded during drop jump would have been expected to decline compared to standing. This however was not the case for any of the stimulation points (see figure 3). A similar facilitation was observed at MLR, LLR and LLR₂ when expressed relative to the background EMG activity (see figure 2D). More importantly, with increased stimulation intensity (from 0.9 MT to 1.2 MT) MEPs in the TA were significantly augmented at each stimulation point. These results imply that neither movement of the coil nor the brain prevented activation of corticomotoneurons. Another possibility to explain the non-facilitated MEPs at SLR, MLR and LLR would be saturation of the muscular output. The muscular responses may have been maximal and therefore no further increment could have been evoked by TMS. However, two observations contradict this reasoning: First, peripheral nerve stimulation induced highly facilitated H-reflex responses at these stimulation points during jumping. Second, H-reflexes and MEPs evoked with the same stimulation intensity as during drop jumps resulted in significantly facilitated responses (expressed in percentage of the background EMG) during maximal voluntary ramp and hold contractions (see the additional protocol). Although maximal voluntary contractions are considered to activate all motor units of a respective muscle, stimulation (e.g. peripheral nerve stimulation) can easily lead to a 3 fold increase in the muscular activity (4; 35). This is thought to rely on the synchronized activation of all motorneurons with nerve stimulation. In contrast, during voluntary contractions non-synchronous discharges lead to amplitude cancellation (10). In the cat soleus muscle it was shown that amplitude cancellation in the surface EMG can reach over 60% at maximal activation (6). Based on these observations, it may be assumed that the muscular response of the SOL muscle around 90 ms (called LLR in the present study) is not mediated via corticospinal projections during drop jump.

If there is in fact no – or only a minor – cortical contribution via the corticospinal tract to the LLR during drop jump, two main questions arise: First, which source(s) of the central nervous system is/are responsible for generating the muscular response around 90 ms in jumping? Second, what functional benefit may emerge from the non-involvement of corticospinal pathways in this situation? Based on results obtained in the upper extremity in monkeys and humans it is conceivable that cortical as well as subcortical regions contribute to the generation of the LLR (2; 38; 55; 58). Similarly, the LLR of the TA muscle seems to depend on both corticospinal and subcortical/spinal pathways as activation of intracortical inhibitory circuits by low intensity TMS depressed the LLRs but generally did not extinguish them (62).
The functional advantage of subcortical levels to control the LLR during drop jump may lay in their temporal precision. For example, cooling or lesions of the cerebellum caused hypermetria in fast wrist movements (11; 17). The cerebellum also proved to be important for precise timing of the finger opening in throwing (59; 60). In mice, the formation of the cerebellum seems to be associated with their jumping behavior (16).

Our methodological approach does not allow us to draw any conclusion about the role of the cerebellum or other subcortical structures during drop jump. Furthermore, it has to be emphasized that the studies cited above neither investigated the LLR response of the SOL muscle nor did they evaluate cerebellar dysfunction in this muscle. Consequently, their results can not directly be transferred to the SOL and to drop jumps in particular. However, it is remarkable that at LLR: i) the H-reflex excitability is low, ii) there is no effect of the TMS onto the EMG but iii) the background EMG activity is still high. Thus it seems reasonable to assume contributions from sources other than the spinal cord or the corticospinal tract in this phase of the movement. Based on the literature cited above and the observation that jumping abilities are impaired in cerebellar patients (45), an involvement of the cerebellum seems conceivable. Nevertheless, our results do not exclude the participation of the cortex at any time during jumping. It is possible that cortical projections other than the corticospinal tract, which are not activated by TMS, contributed to muscular activity during the early phase after ground contact.

**Conclusion**

The results of the present study propose that cortical, spinal and presumably subcortical sources contribute to muscular activation in drop jump in a very time dependent manner. The first EMG peak after touch down strongly relies on Ia-afferent feedback as indicated by highly facilitated H-reflexes. Thereafter, the spinal contribution progressively declines suggesting other sources to become more relevant. Since the susceptibility of corticomotoneurons was not enhanced until the LLR$_2$ (~120 ms), cortical influence via the corticospinal tract is supposed to be minor in the early stance phase. The non-facilitation of the MEP at the LLR during drop jump in contrast to the facilitation of the LLR obtained after postural displacement (57) may point towards task-specific gating of transcortical reflex loops.

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References


38. **Miller AD and Brooks VB.** Late muscular responses to arm perturbations persist during supraspinal dysfunctions in monkeys. *Exp Brain Res* 41: 146-158, 1981.

39. **Morita H, Olivier E, Baumgarten J, Petersen N, Christensen LOD and Nielsen JB.** Differential changes in corticospinal and Ia input to tibialis anterior and soleus motor


corticospinal pathways contribute to neuromuscular control of perturbed stance. *J Appl

58. Taylor JL, Fogel W, Day BL and Rothwell JC. Ipsilateral cortical stimulation
inhibited the long-latency response to stretch in the long finger flexors in humans. *J

59. Timmann D, Citron R, Watts S and Hore J. Increased variability in finger position
occurs throughout overarm throws made by cerebellar and unskilled subjects. *J

60. Timmann D, Watts S and Hore J. Failure of cerebellar patients to time finger opening
precisely causes ball high-low inaccuracy in overarm throws. *J Neurophysiol* 82: 103-
114, 1999.

61. Ugawa Y, Terao Y, Hanajima R, Sakai K and Kanazawa I. Facilitatory effect of
tonic voluntary contraction on responses to motor cortex stimulation.

62. van Doornik J, Masakado Y, Sinkjaer T and Nielsen JB. The suppression of the
long-latency stretch reflex in the human tibialis anterior muscle by transcranial magnetic

63. Voigt M, Dyhre-Poulsen P and Simonsen EB. Modulation of short latency stretch
Figure legends

**Figure 1.** EMG pattern of the SOL muscle during drop jump of one subject. Time 0 corresponds to the time of ground contact. The consecutive activation peaks in the SOL muscle were termed short- (SLR), medium- (MLR) and long-latency response (LLR and LLR₂). Peripheral nerve stimulation and transcranial magnetic stimulation (TMS) were triggered so that the H-reflexes and the MEPs coincided with the peaks of SLR, MLR, LLR and LLR₂, respectively. Stimulation intensity of the peripheral nerve stimulation and of the TMS was kept constant at SLR, MLR, LLR and LLR₂. Modulation of H-reflexes and MEPs are exemplary displayed. Background EMG, H-reflexes and MEPs were compared by analyzing root mean square values (RMS) 20 ms around each reflex peak (grey boxes).

**Figure 2.** Changes in the size of the H-reflex (HR) and the motor evoked potential (MEP) during the time course of drop jumping. H-reflexes and MEPs are expressed as percentage of the SOL (2A, C) and TA (2D) background EMG. 2A, H-reflexes were greatest shortly after ground contact and then progressively decreased (p < 0.05 *; p = 0.054 #). H-reflexes obtained with low (□) and high (◆) stimulus intensity were modulated in the same way. M-waves measured in the trials with high stimulation intensity were similar in size at SLR, MLR, LLR and LLR₂ and showed no statistical differences (2B). 2C, TMS to the SOL had no influence on SLR, MLR and LLR but significantly facilitated the LLR₂ (◆ subthreshold TMS with 0.9 MT, p = 0.003**; ▲ suprathreshold TMS with 1.2 MT, p = 0.016). 2D shows facilitation of TA MEPs following sub- (◆ 0.9 MT) and suprathreshold (▲ 1.2 MT) stimulation. An increase in the stimulus intensity enhanced MEPs in the TA to a similar extent at MLR, LLR and LLR₂ (ANOVA “stimulus intensity” p = 0.038; “stimulus intensity” * “stimulation point” n. s.; “stimulation point” n. s.).

**Figure 3.** Tibialis anterior motor evoked potentials (◆ MEP TA) during drop jump (SLR, MLR, LLR and LLR₂) and quiet stance. MEPs in the TA during jumping were always greater than MEPs during standing (p = 0.07 #, p < 0.05 *; p < 0.01 **). At each stimulation point, the stimulus intensity was adjusted to 90% of the motor threshold (0.9 MT) of the SOL muscle during standing. This intensity was sufficiently high to elicit MEPs in the TA during stance as can be seen when comparing EMG activity without stimulation (□ background EMG TA) and with TMS (◆ MEP TA at STANCE; p < 0.001 ***).
**Figure 4.** Modulation of the H-reflex (A) and the MEP (B) during the stance phase of drop jump. Stimulation instances (SLR, MLR, LLR and LLR₂) were determined by analyzing the rectified background EMG (bEMG; grey lines) obtained in drop jumps without stimulation. Thereupon, stimulation was timed so that the H-reflex and the MEP, respectively, coincided with the activation peaks of SLR, MLR, LLR and LLR₂. In A, changes in the H-reflex size at SLR, MLR, LLR and LLR₂ of one subject are displayed. The H-reflex (H) was greatest at SLR and then progressively decreased although the M-wave (M) indicated constant stimulation intensity. In B, transcranial magnetic stimulation (TMS) to the SOL muscle of another subject revealed only minor effects at SLR, MLR and LLR whereas at LLR₂ the response was strongly facilitated (MEPs are expressed in percentage of the background EMG; s = stimulus artifact).

**Figure 5.** Modulation of the H-reflex and the MEP during voluntary contractions. In A and C, regression lines are illustrated for each participating subject (solid lines) as well as the mean (dashed line). Both, H-reflexes (A) and MEPs (C) increased with increasing contraction levels. However, H-reflexes (B) and MEPs (D) obtained with contraction levels between 60% and 100% of the MVC do not show any contraction dependent modulation (shown are all data from all subjects obtained with peripheral nerve stimulation (B) and TMS (D) and the corresponding regression lines; for better visualization data in B and D are expressed in z-normalized values).
Table 1  Background EMG (bEMG) of SOL and TA in mV at SLR, MLR, LLR and LLR₂ in H-reflex and TMS trials (RMS in a time interval of 20 ms around each peak; see figure 1)

<table>
<thead>
<tr>
<th></th>
<th>SLR</th>
<th></th>
<th></th>
<th>MLR</th>
<th></th>
<th></th>
<th>LLR</th>
<th></th>
<th></th>
<th>LLR₂</th>
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<tbody>
<tr>
<td></td>
<td>HR</td>
<td>TMS</td>
<td>Sig.</td>
<td>HR</td>
<td>TMS</td>
<td>Sig.</td>
<td>HR</td>
<td>TMS</td>
<td>Sig.</td>
<td>HR</td>
<td>TMS</td>
</tr>
<tr>
<td>SOL</td>
<td>0.24 ± 0.03</td>
<td>0.26 ± 0.04</td>
<td>n.s.</td>
<td>0.32 ± 0.05</td>
<td>0.32 ± 0.03</td>
<td>n.s.</td>
<td>0.32 ± 0.05</td>
<td>0.36 ± 0.04</td>
<td>n.s.</td>
<td>0.20 ± 0.03</td>
<td>0.21 ± 0.04</td>
</tr>
<tr>
<td>TA</td>
<td>0.11 ± 0.02</td>
<td>0.12 ± 0.02</td>
<td>n.s.</td>
<td>0.12 ± 0.01</td>
<td>0.11 ± 0.02</td>
<td>n.s.</td>
<td>0.10 ± 0.01</td>
<td>0.09 ± 0.02</td>
<td>n.s.</td>
<td>0.08 ± 0.02</td>
<td>0.08 ± 0.01</td>
</tr>
</tbody>
</table>

Values are means ± SEM; HR = bEMG in trials with peripheral nerve stimulation; TMS = bEMG in trials with TMS; Sig. = significance level; n.s. = not significant
Figure 1

Graph showing various reflexes and responses:
- MEP
- H-reflex
- Touch down

Time [ms]
0 50 100 150 200

0.2 mV

1000 N

Touch down
Ground reaction force
Background EMG m. soleus
Peripheral Nerve Stimulation
Transcranial Magnetic Stimulation

Background EMG m. soleus
Figure 3

RMS-EMG of the TA [mV]

- **MEP TA**
- ■ Background EMG TA

Stimulation Point: SLR, MLR, LLR, LLR2, STANCE

**Statistical significance:**
- *p < 0.05
- **p < 0.01
- #p < 0.1