Differential Modulation of Spinal and Corticospinal Excitability During Drop Jumps

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Taube W, Leukel C, Schubert M, Gruber M, Rantalainen T, Gollhofer A. Differential modulation of spinal and corticospinal excitability during drop jumps. J Neurophysiol 99: 1243–1252, 2008. First published January 16, 2008; doi:10.1152/jn.01118.2007. 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 toward takeoff. 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, LLR2)-latency responses of the soleus muscle (SOL) after jumps from 31 cm height. MEPs and H-reflexes were expressed relative to the backcentric phase when compared with isolated concentric action. Consequently it was 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 preactivated 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 with isolated concentric action (Dietz et al. 1979; Gollhofer et al. 1992; Voigt et al. 1998). In hopping and during drop jumping, it was shown that the H-reflex excitability was very low at takeoff, still low in the flight phase, but increased before touch down. During the stance phase, H-reflexes remained facilitated but decreased again before takeoff (Dyhre-Poulsen et al. 1991; Moritani et al. 1990; Voigt et al. 1998). The increase of the Ia-transmission toward 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 (20–60 cm) compared with jumps from “great” heights (76–80 cm) (Komi and Gollhofer 1997; Taube et al. 2006). 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 predict the time of ground contact and thus the instance of muscular stretch (Dyhre-Poulsen and Lauresen 1984; Horita et al. 1996, 2002). Therefore it is assumed that supraspinal centers not only initiate jumping and landing movements but also preprogram 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) (Christensen et al. 2001; Petersen et al. 1998; Taube et al. 2006). 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 predict the time of ground contact and thus the instance of muscular stretch (Dyhre-Poulsen and Lauresen 1984; Jones and Watt 1971; McDonagh and Duncan 2002; Santello and McDonagh 1998). Therefore it can be speculated that during drop jump, preprogrammed 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 cortico-

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spinal excitability during the drop jump at distinct phases after ground contact (SLR after \(\sim 45\) ms, MLR \(\sim 70\) ms, LLR \(\sim 90\) ms, and LLR\(_2\) \(\sim 120\) 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**

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 \(\pm 1.5\) yr). 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

After a 5-min 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 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 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, MA). According to their latencies, the first three response peaks were termed SLR, MLR, and LLR. The fourth peak in the EMG was assumed to be a later component of the LLR and subsequently called LLR\(_2\). The onsets and peaks of the SLR, MLR, LLR, and LLR\(_2\) were determined on the basis of the background EMG activity and previously reported latencies and durations of SLR, MLR, LLR, and LLR\(_2\) (Grey et al. 2001; Kawashima et al. 2004; Petersen et al. 1998; Sinkjaer et al. 1999; Taube et al. 2006). Stimulation was timed so that the 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 LLR\(_2\) (see Fig. 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 LLR\(_2\); 2) drop jumps plus electrical stimulation (soleus H-reflexes) to assess spinal excitability at SLR, MLR, LLR, and LLR\(_2\); a) stimulation intensity adjusted to elicit H-reflexes of 20% of \(M_{max}\) during stance (“low intensity stimulation”) and b) stimulation intensity adjusted to elicit M-waves of 50% of \(M_{max}\) during stance (“high-intensity stimulation”); and 3) drop jumps + TMS to assess corticospinal excitability at SLR, MLR, LLR, and LLR\(_2\) peaks: stimulation intensity subthreshold to elicit MEPS during stance [0.9 motor threshold (MT); in accordance with Day et al. (1991)] and stimulation intensity of 1.2 MT to elicit MEPS during stance [suprathereshold stimulation at MLR, LLR, and LLR\(_2\); in accordance with Petersen et al. (1998)].

TMS- and peripheral nerve stimulation experiments were conducted on two separate days to avoid fatigue. At each test day, subjects performed \(\sim 120\) drop jumps. After every 10 jumps, subjects were instructed to rest for 2 min. After familiarization with the jumping procedure, 15 jumps without stimulation were performed to determine the latencies for the SLR, MLR, LLR, and LLR\(_2\). Based on these latencies and the latencies of the MEP and the H-reflex, the modulation of H-reflexes and MEPs were compared by analyzing root mean square values (RMS) 20 ms around each reflex peak (gray boxes).

**FIG. 1.** Electromyographic (EMG) pattern of the soleus (SOL) muscle during drop jump of 1 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 LLR\(_2\)). Peripheral nerve stimulation and transcranial magnetic stimulation (TMS) were triggered so that the H-reflexes and the motor-evoked potentials (MEPs) coincided with the peaks of SLR, MLR, LLR, and LLR\(_2\), respectively. Stimulation intensity of the peripheral nerve stimulation and of the TMS was kept constant at SLR, MLR, LLR, and LLR\(_2\). 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 (gray boxes).
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 afterward based on the trigger signal, which had been derived from the first increase in ground reaction force (ground contact). Additionally, five 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, five 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 ($M_{max}$). 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 (Pinniger et al. 2001).

**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, center to center 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 ($\times 1,000$), band-pass-filtered (30–1,000 Hz) and sampled at 4 kHz.

**H-reflex recording**

H-reflexes were elicited with an electrical stimulator (DS 7, Digitimer) 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 $10 \times 5$-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 an 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 $M_{max}$ (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 $M_{max}$ to obtain a sensitive measure for changes in the stimulation intensity (Pinniger et al. 2001).

**TMS**

TMS was applied over the left motor cortex using a Magstim 200 (Magstim, Dyfed, UK) with a 90-mm circular coil. The stimulus waveform was monophasic and had a pulse width of 200 $\mu$s. For each subject, the initial coil position was chosen so that the center of the coil was situated $\sim 0.5$ 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, whereas 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. MT of the SOL was determined during stance. MT was defined as the intensity at which an MEP reached 150 $\mu$V in 3 of 5 trials (Kujirai et al. 1993). The threshold was set to 150 $\mu$V as some subjects showed relatively high tonic muscular activation during stance (around 100 $\mu$V). Stimulation intensity during jumps was adjusted to 0.9 MT (subthreshold trials). At MLR, LLR, and LLR2, corticospinal excitability was additionally assessed with 1.2 MT (suprathreshold trials; see general experimental procedure in preceding text). 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, Garmsich-Partenkirchen, Germany), which provided a flexible adjustment to head-sizes from 53 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 horizontally 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 jump ($0–1$ mm). For measurements during jumping, the “helmet construction” therefore provided several advantages compared with the halo vest used in the same lab before, which was introduced by Schubert et al. (1997): 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 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%, 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 $M_{max}$ 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 LLR2. The RMS values were chosen according to Kiers et al. (1995). 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 (Simonsen et al. 1995; Voigt et al. 1998). 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 LLR2.

To ensure comparable stimulation intensity of the peripheral nerve stimulation, the intensity was adjusted to elicit M-waves of 50% of $M_{max}$ (M-wave$_{50\%}$) during stance. Subjects who showed significant deviations of their M-wave$_{50\%}$ amplitudes among SLR, MLR, LLR, and LLR2 were excluded from any analysis. For this purpose, the median of the M-wave$_{50\%}$ amplitudes of SLR, MLR, LLR, and LLR2
was calculated. Subjects were excluded if the mean of one of their M-wave_{50%} deviated more than ±12.5% of M_{max} (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-wave_{50%} \{formula: \[(HR – bEMG)/M-wave_{50%}\] * 100}. This analysis revealed also a progressive modulation see Fig. 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).

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 with the EMG responses without stimulation when expressed with respect to the background EMG activity (ANOVA “EMG activity” P = 0.002; Fig. 2A). This facilitation progressively decreased at MLR, LLR, and LLR_2 (ANOVA “stimulation point” P = 0.01; Fig. 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 Fig. 2B) showed a very similar modulation (H-reflex modulation see Fig. 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).

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 TMS in METHODS), MLR and LLR when compared with the background EMG [ANOVA EMG activity, not significant (NS)]. At LLR_2, TMS significantly facilitated the SOL response (Figs. 2C and 4; 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 vs. 122 ± 4%). However, this difference was not significant as only 7 of 10 subjects revealed greater MEPs with enhanced stimulation intensity (ANOVA intensity, NS; stimulation point*intensity, NS).

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 (Fig. 3). The same stimulation intensity applied during jumping further enhanced MEPs in the TA (ANOVA EMG activity, P = 0.021; Figs. 2D and 3). Thereby absolute MEP amplitude was greatest at SLR and progressively decreased at MLR, LLR, and LLR_2 (Fig. 3). Taking background EMG activity into account, comparable facilitation occurred at SLR, MLR, LLR, and LLR_2 (see Fig. 2D; ANOVA stimulation point, NS). An increase in the stimulus intensity enhanced MEPs in the TA to a similar extent at MLR, LLR, and LLR_2 (see Fig. 2D; ANOVA stimulus intensity, P = 0.038; stimulus intensity*stimulation point, NS; stimulation point, NS).

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 Fig. 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 vs. LLR_2, P = 0.028).

Table 1. Background electromyogram of SOL and TA in mV at SLR, MLR, LLR, and LLR_2 in H-reflex and TMS trials (RMS in a time interval of 20 ms around each peak; see Fig. 1)

<table>
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<tr>
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<th>SLR</th>
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<th>MLR</th>
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<td></td>
<td>HR</td>
<td>TMS</td>
<td>Significance</td>
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<td>Significance</td>
<td>HR</td>
<td>TMS</td>
<td>Significance</td>
<td></td>
</tr>
<tr>
<td>SOL</td>
<td>0.24±0.03</td>
<td>0.26±0.04</td>
<td>NS</td>
<td>0.32±0.05</td>
<td>0.32±0.03</td>
<td>NS</td>
<td>0.32±0.05</td>
<td>0.36±0.04</td>
<td>NS</td>
<td>0.20±0.03</td>
<td>0.21±0.04</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>TA</td>
<td>0.11±0.02</td>
<td>0.12±0.02</td>
<td>NS</td>
<td>0.12±0.01</td>
<td>0.11±0.02</td>
<td>NS</td>
<td>0.10±0.01</td>
<td>0.09±0.02</td>
<td>NS</td>
<td>0.08±0.02</td>
<td>0.08±0.01</td>
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Values are means ± SE; HR, background electromyogram (bEMG) in trials with peripheral nerve stimulation; TMS, bEMG in trials with transcranial magnetic stimulation; NS, not significant; SLR, MLR, LLR, LLR_2, short-, medium-, and long-latency response; SOL, soleus muscle; TA, tibialis anterior.

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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. 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 ± SE. In the additional protocol, slopes of linear regression were compared using the Z-test for parallelism (Kleinbaum and Kupper 1978).

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**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 Figs 4 and 5A and 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 (60–100% 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 Fig. 5B and 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 LLR$_2$) 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 jumps. 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 (Burke et al. 1989; Funase et al. 1999; Lavoie et al. 1995; Maertens de Noordhout et al. 1992; Morita et al. 2000; Schieppati 1987; Ugawa et al. 1995). 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, Lösch et al. (1996) reported a similar modulation in three of eight subjects at high contraction levels: H-reflexes increased ≤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 that may cause motoneurons to discharge more than once. At the same time, dispersed volleys can lead to phase cancellation of action potentials that in turn reduce the size of the MEP (Kawashima et al. 2004). In contrast, the H-reflex is elicited by a single synchronized volley, and in this respect resembles the cervicomedullary MEP (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 (Martin et al. 2006).
authors concluded that alterations in the contraction strength lead to changes in the excitability of the motoneuron pool that 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 takeoff and stayed low during the flight phase (Dyhre-Poulsen et al. 1991; Moritani et al. 1990; Voigt et al. 1998). Our results support these earlier observations, also showing facilitated H-reflexes in the early stance phase (SLR, MLR) that then decrease toward takeoff (LLR, LLR2). Thereby a similar modulation was seen with contraction levels between 60 and 100% of the maximal voluntary contraction. 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 (Dyhre-Poulsen et al. 1991). In this way, the stretch reflex response most likely contributes to the enhanced efficiency of the stretch-shortening cycle compared with concentric action (Dietz et al. 1979; Golldofer et al. 1992). The progressive decline of the H-reflex amplitude relative to the background EMG at MLR, LLR, and LLR2 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. (2001), 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 takeoff, which is preprogrammed 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 (Norlund et al. 2002; Patikas et al. 2004) as well as activity in upper leg muscles like the biceps femoris or the rectus femoris (Izumi et al. 2001; Knikou and Conway 2002; Morita et al. 1998). Furthermore, cutaneous afferent input can also alter the excitability of the H-reflex (Knikou 2007). 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) that 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 toward takeoff (LLR2).

Modulation of the MEP

To test for cortical contribution, Day et al. (1991) 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. (1998) 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 (Taube et al. 2006).

In the present study, MEPS were superimposed on muscular activation peaks following touch down 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 ~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 (Petersen et al. 1998). Consequently, excitability changes assessed by TMS alone can hardly be attributed to specific structures of the CNS (Nielsen and Petersen 1995). Thus the facilitation of MEPS at LLR2 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 (Dyhr-Poulsen et al. 1991; Moritani et al. 1990), it seems unlikely that changes at the spinal level were responsible for enhanced efficiency of the TMS at LLR2. Although presynaptic mechanisms may have caused the reduced H-reflex excitability at LLR2, this would not explain facilitated MEPS at this time. Similarly, the on-average slightly lower background EMG activity at LLR2 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 LLR2, 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 hillock) and the corticospinal fibers are considered to be free from presynaptic inhibition (Jackson et al. 2006; Nielsen and Petersen 1994), changes in the MEP size that cannot 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 LLR2 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 LLR2, which occurred after ~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 (Petersen et al. 1998) and from 90 ms onwards with a peak at 110 ms following stretch applied during walking (Christensen et al. 2001). Similarly, contribution of transcortical pathways in the SOL muscle during posterior translation of the support platform was evident after 86–100 ms (Taube et al. 2006). 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 LLR2. 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 (Dyhr-Poulsen and Laursen 1984; McDonagh and Duncan 2002; Santello and McDonagh 1998). 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 with standing. This, however, was not the case for any of the stimulation points (see Fig. 3). A similar facilitation was observed at MLR, LLR, and LLR2 when expressed relative to the background EMG activity (see Fig. 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 nonfacilitated 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 threefold increase in the muscular activity (Cioni et al. 1985; Marsh et al. 1981). This is thought to rely on the synchronized activation of all motorneurons with nerve stimulation. In contrast, during voluntary contractions nonsynchronous discharges lead to amplitude cancellation (Farina et al. 2004). In the cat soleus muscle, it was shown that amplitude cancellation in the surface EMG can reach >60% at maximal activation (Day and Hulliger 2001). Based on these observations, it may be assumed that the muscular response of the SOL muscle ~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 CNS is/are responsible for generating the muscular response around 90 ms in jumping? Second, what functional benefit may emerge from the noninvolvement 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 (Chofflon et al. 1982; Miller and Brooks 1981; Tatton et al. 1975; Taylor et al. 1995). 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 (van Doornik et al. 2004). 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 (Flament and Hore 1986; Hore et al. 1991). The cerebellum also proved to be important for precise timing of the finger opening in throwing (Timmann et al. 1999, 2001). In mice, the formation of the cerebellum seems to be associated with their jumping behavior (Grimm et al. 1980).

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 in the preceding text neither investigated the LLR response of the SOL muscle nor did they evaluate cerebellar dysfunction in this muscle. Consequently, their results cannot directly be transferred to the SOL and to drop jumps in particular. However, it is remarkable that at LLR, the H-reflex excitability is low, there is no effect of the TMS onto the EMG, but 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 in the preceding text and the observation that jumping abilities are impaired in cerebellar patients (O’Hare and Khalid 2002), 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. Because the susceptibility of corticomotoneurons was not enhanced until the LLR2 (~120 ms), cortical influence via the corticospinal tract is supposed to be minor in the early stance phase. The nonfacilitation of the MEP at the LLR during drop jump in contrast to the facilitation of the LLR obtained after postural displacement (Taube et al. 2006) may point toward task-specific gating of transcortical reflex loops.

GRANTS

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