

How Neurons Make Us Jump: The Neural Control of Stretch-Shortening Cycle Movements

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How can the human central nervous system (CNS) control complex jumping movements task- and context-specifically? This review highlights the complex interaction of multiple hierarchical levels of the CNS, which work together to enable stretch-shortening cycle contractions composed of activity resulting from feedforward (preprogrammed) and feedback (reflex) loops. **Key Words:** motor control, neural plasticity, jumping, feedforward, feedback

INTRODUCTION

By definition, the stretch-shortening cycle (SSC) describes a natural muscle function in which the preactivated muscle-tendon complex is lengthened in the eccentric phase followed by muscle-tendon shortening in the concentric phase. In bipedal and quadrupedal species, locomotion, as well as many other movements, such as hopping and throwing, is organized in a SSC. It was argued that the efficiency of the SSC is dependent on the ability to transfer energy from the preactivated and eccentrically stretched muscle-tendon complex to the concentric push-off phase (13). Therefore, the major advantage of the SSC compared with isolated concentric and eccentric muscle activation is considered to be the partial storage and subsequent release of kinetic energy leading to enhanced power and/or greater economy (7,33). The efficiency of the SSC therefore is dependent on the recoil properties of the tendomuscular system, which can be influenced by the central nervous system (CNS). First, the preprogrammed muscular activation prior to touchdown can be adapted presumably to provide an appropriate stiffness. In this respect, Arampatzis *et al.* (1) demonstrated that the change in leg stiffness was related to the level of preactivation (pre-*innervation*) but not its duration. Second, reflex activity after touchdown can be modulated task-specifically (17). Third, the preprogrammed muscular activity after touchdown, which

is not influenced primarily by reflex activity (35), might be adapted according to the task (for instance, drop height) and the training status (29).

This shows that the neural control of SSC is highly complex, as both feedforward (preprogrammed) and feedback (reflex) mechanisms have to be highly adaptive to ensure the balance between a system, which achieves maximum performance (power), and the risk of overload injuries. Especially during jumping, very high forces are exerted on the tendons so that the Achilles and patella tendons work in ranges relatively close to their point of failure. Thus, muscular activations during SSC movements have to be adjusted both task- and phase-specifically. For this purpose, multiple hierarchical levels of the CNS have to interact accurately to ensure the appropriate muscular activation. We present an integrated view of our research based on electrophysiological measurements and present these findings in the context of other closely related literature. We focus on the interaction of spinal and cortical levels and the combination of feedforward (preprogrammed) and feedback (reflex) controlled muscular activation.

SPINAL MECHANISMS CONTRIBUTING TO THE SSC

Do Spinal (Stretch) Reflexes Occur During SSC?

The human CNS responds instantaneously to stretches of a relaxed muscle. Muscle spindles detect changes in the muscle length and alter firing frequencies in Ia afferent fibers proportionate to the velocity of the change in length and to a smaller degree in relation to the amplitude (11). The increased activity of Ia afferents after muscle stretch depolarizes α -motoneurons at the spinal level, which elicit a stretch reflex called short-latency response (SLR). If the muscle is

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preactivated before stretching, not only an SLR can be observed but also medium-latency responses (MLR) and long-latency responses (LLR) (16,31). Based on these observations, it seems reasonable to assume that stretch reflexes also are elicited in the eccentric phase of SSC as one may argue that the mechanism of stretching the extensor muscles during touchdown is similar to other tasks like rotating the ankle joint. Otherwise, it may be proposed that the ankle rotation and the consequent muscle stretch during a voluntarily initiated drop jump is organized differently than after externally driven ankle rotations because of the capacity of the CNS to predict accurately the instance of ground contact (*e.g.*, (23)) and, thus, the onset of the muscle stretch. Theoretically, the CNS could inhibit muscular activation caused by afferent excitation precisely at the time of the SLR. As both lines of reasoning seem equally warranted, several experimental setups have been used to clarify whether muscular activity at the time of the SLR during SSC movements indeed is influenced by spinal stretch reflexes. Observations strengthening the hypothesis of an integrated stretch reflex were the following: a) the latency of the first muscular activation peak after ground contact corresponded to the latency of the SLR elicited by stretching of the relaxed muscle (7,19); b) the muscular activation peaks of the triceps surae increased with increasing stretch-velocity (14); c) the maximum electromyography (EMG) amplitude during the contact phase of running was two to three times higher than the activity during maximum voluntary contractions (7); d) the activation peak at the time of the SLR decreased during running after partial blockage of Ia afferents by ischemia (7); and e) vibration of the Achilles tendon, which is known to decrease primarily the efficacy of Ia afferent activity, led to a significant decrease of the SLR during running (5).

Recently, we provided further evidence, which strongly supported the assumption that stretch reflex generated muscular activity during SSC movements. In the first study, we introduced a new methodology to investigate stretch reflex responses by means of a pneumatic cuff surrounding the lower leg (18). Immediately after inflation of the cuff, a selective reduction of the SLR could be seen, which was elicited by a dorsiflexion of the foot in an ankle ergometer. Changes in the stretch velocity but not the stretch amplitude affected the size of the SLR pointing toward the Ia afferent pathway being primarily responsible for this response. As the effect was seen immediately after inflation of the cuff, the time was too short to cause ischemia. Therefore, it was postulated that inflation restricted the stretching of the muscles under the cuff so that most of the changes in length probably occurred in the series elastic structures of the muscle-tendon complex distal to the cuff. As a consequence, the muscle spindles embedded within the muscle may be less excited, resulting in a reduced SLR. When the cuff was applied during hopping, the muscular activity at the phase of the SLR also was reduced (18). Thus, it seems likely that the Ia afferent pathway is important to generate the SLR in hopping, whereas other structures like the Golgi tendon organs or cutaneous receptors probably are much less involved.

The most recent and convincing argument that spinal reflexes contribute to SSC movements was derived from the observation of a time-locked occurrence of the SLR with respect to the instant of ground contact (35). In this study,

we altered the time of ground contact during hopping by changing the height of the landing surface while subjects were airborne. We hypothesized that if a stretch reflex indeed contributes to the early EMG burst, then advancing or delaying the touchdown without the subject's knowledge should advance similarly or delay the SLR burst. This was indeed the case when touchdown was advanced or delayed by shifting the height of a programmable platform up or down between two hops, and this resulted in a correspondent shift of the SLR (Fig. 1). These results are in line with observations from landing, where the EMG burst that appears shortly after landing disappeared when the subjects fell through a false floor, which confirms that the burst results from a feedback loop, *i.e.*, from a stretch reflex (8).

In summary, there is good evidence for spinal stretch reflex activity at the time of the SLR during SSC movements. The most likely source of this reflex activity is the excitation of primary muscle spindle endings. However, there is a discussion going on how the muscle spindles are activated: some authors propose muscle fascicle stretches being the relevant stimuli, whereas others argue that mechanical vibrations in response to the ground contact trigger the response (for literature, see (5)). Furthermore, it is likely that during functional movements several other pathways may influence the SLR including the Golgi tendon organ Ib afferents, cutaneous receptors, and mechanoreceptors in other muscles, as well as preprogrammed input from supraspinal centers (for further details, please see (5) as well as the section "Supraspinal mechanisms contributing to the SSC").

Functional Role of the Stretch Reflex in SSC Movements

As previously mentioned, the efficiency of the SSC is dependent on the energy transfer from the preactivated and eccentrically stretched muscle-tendon complex to the concentric push-off phase. An appropriate stiffness regulation is considered to be one constitutive factor for a successful transfer (13). It is assumed widely that stretch reflexes may have a role in adjusting the leg stiffness. More specifically, reflex contributions induced by stretching of the antigravity muscles during touchdown (eccentric phase) were proposed to enhance muscular stiffness and therefore increase the performance during the concentric phase when compared with isolated concentric action (7,33). This reflex-induced enhancement of performance may be even more relevant in submaximal SSC contractions as it was observed that the SLR after rapid stretch of the isometrically contracted muscle is largest when the intrinsic muscle stiffness is low. Thus, the reflex may prevent muscle yielding in conditions where the muscle is not (pre-) activated strongly. In line with this assumption, vibration-induced reductions of the SLR were shown to increase muscle yielding while running with low-to-moderate speeds (7–12 km·h⁻¹) but not at a faster speed (15 km·h⁻¹) (5).

The importance of stretch reflex responses to modulate the leg stiffness first was highlighted in animal studies demonstrating that muscle stiffness is dependent on an intact reflex system and is reduced in the a-reflexive state. Furthermore, animal studies indicated that reflex responses can change the form of the mechanical force response of the muscle from one dominated by viscosity to one dominated by elasticity (21).

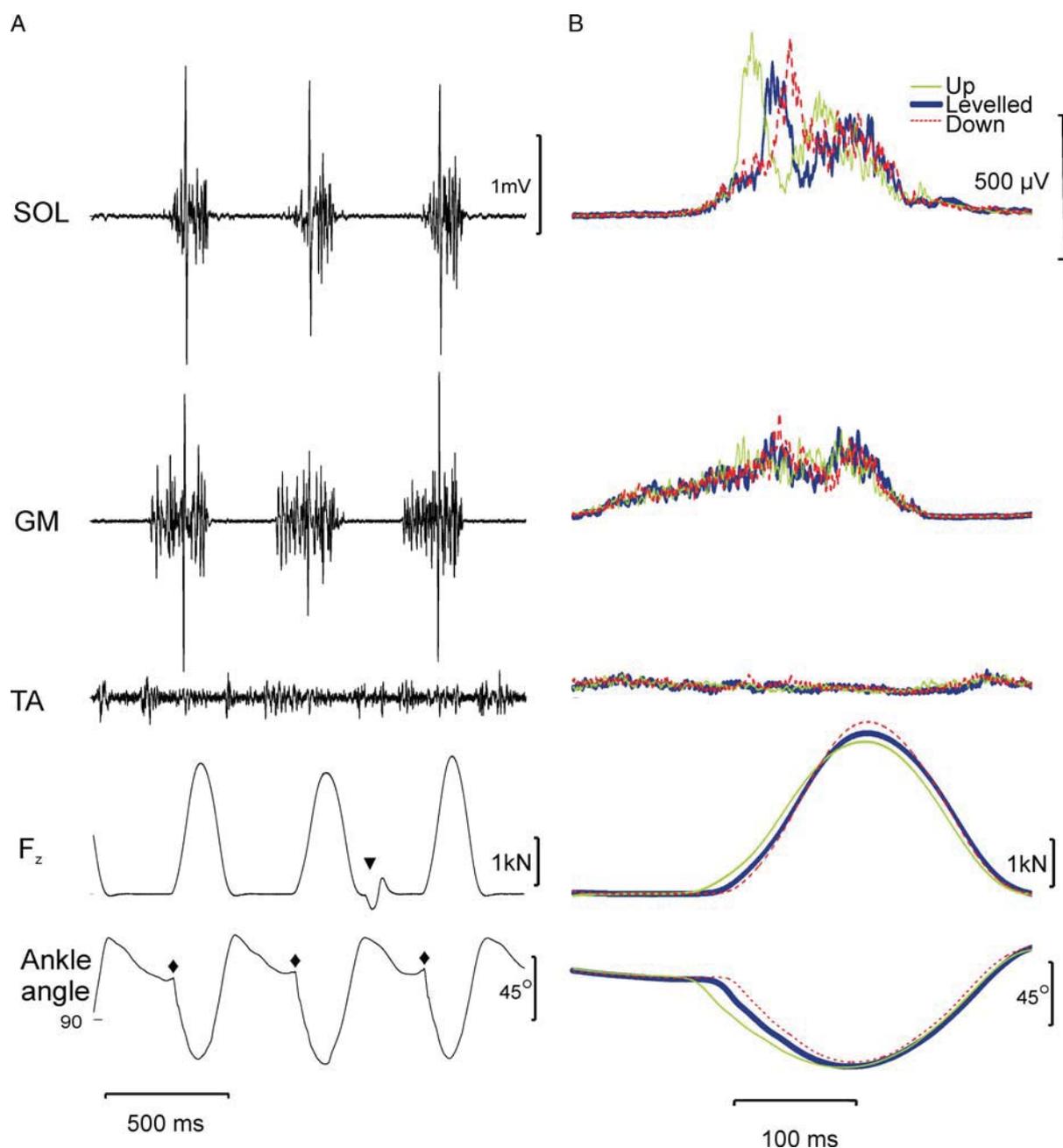


Figure 1. Evidence of spinal stretch reflex activity during hopping on a movable platform. A. Raw electromyography (EMG) and kinematics from three hops indicate an artifact in the force signal because of acceleration and deceleration of the platform when moving up or down and indicate the point where the ankle angle shows a maximal dorsiflexion acceleration. B. Ensemble average of 25 sweeps for the conditions "Up," "Level," and "Down." "Up," "Level," and "Down" refer to the positions of the platform. During the period the subject was in the air, the platform either stayed in the leveled position (Level) or moved 2.5 cm up or down in a randomized fashion. In the control trials, the platform made a lateral movement but returned to the level position before the subject touched down so that in all three conditions the same sound was made, and no audible cues were given about the position of the platform. All averaged trials are aligned to the crossing of the light barrier, which was positioned 3 cm above the moveable force platform. Note that the platform in the "Up" position causes a shift in the signals for ankle angle, ground reaction force, and the short-latency EMG burst in the soleus muscle ahead in time, whereas the "Down" position delays these signals in time. The shift in the short-latency response dependent on the position of the landing surface indicates that this burst is caused by peripheral feedback because of the impact at touchdown. In contrast, the initial rise in EMG activity (before the sharp peak of the short-latency response) is comparable in all three conditions. Thus, it can be assumed that this rise is not caused by a feedback process but is rather preprogrammed (feedforward control). (Reprinted from (35). Copyright © 2010 The Physiological Society. Used with permission.)

Thus, reflex responses seem to ensure and to preserve the system's elasticity (21). However, although it seems, based on these observations, beyond controversy that the mechanical stiffness of the leg can be influenced by spinal stretch reflexes, it remains a topic of debate how the neuromuscular system has to be adjusted to create optimal mechanical properties

for energy storage and utilization during SSC (a thorough discussion of the mechanical properties determining the performance in SSC movements can be found in (4)).

Based on cross-sectional studies, it seems that the stretch reflex is adjusted according to the falling height by the CNS: when subjects were asked to perform drop jumps from different

drop heights, the muscular activity at the SLR was lower in drop jumps from excessive (80 cm) than from low (30 cm) heights (14) (Fig. 2). In subsequent experiments of our group, the size of the H-reflex at the time of the SLR was related inversely to the drop height (17,19). Furthermore, we could demonstrate in submaximally performed drop jumps that the decrease in H-reflex amplitude with increasing drop height was correlated with a reduction in the ankle joint stiffness (29). This correlation supports and strengthens the long-held assumption (7,10,13,33) that spinal reflex gating contributes to the modulation of the ankle joint stiffness during SSC movements. It may be hypothesized that from a functional point of view, reduction of the Ia afferent input (17,19) and the muscular activity (13,14) at the time of the SLR could serve as a preventative mechanism to compensate for the higher loads associated with greater drop heights (Fig. 2). Diminished reflexes were thought to reduce the stiffness and, therefore, the peak stress of the tendomuscular system (10). Furthermore, based on the results of Lin and Rymer (21), who demonstrated that tendomuscular elasticity is dependent on intact reflex responses, it can be hypothesized that the mechanical force response of the muscles progressively changes from an elastic to a more viscous state when augmenting the drop height because of a reduction in reflex contribution. Such gradual switches in reflex contribution influencing the muscle properties should be most pronounced when subjects are asked to land instead of rebounding from the ground. Indeed, it was demonstrated that subjects drastically reduced their H-reflexes in the landing condition (10,17) where a viscous system was required.

Interestingly, the neuromuscular system adjusts the muscular activity not only based on the drop height but also depending on the characteristics of the landing surface. It was

demonstrated that the stretch reflex response is diminished when rebounding from soft elastic surfaces (24). Moritz and Farley (24) further observed that apart from the stretch component, the overall muscular activity was higher on soft surfaces than on solid surfaces despite similar joint moments and mechanical leg work. Additionally, the leg kinematics changed from the normal pattern known from solid surfaces where, during the contact phase, the legs first are flexed and extended subsequently to a reversed pattern. Therefore, the authors assumed that a higher overall muscular activation might be needed to compensate for the loss of the normal extensor muscle stretch-shortening cycle, that is, to compensate for the loss of the stretch reflex contribution.

The preceding paragraphs illustrated that the Ia afferent transmission and the muscular activity at the time of the SLR are modulated task- and context-specifically. However, although there is a clear decrease in the muscular activity at the time of the SLR from drop jumps from low drop heights to drop jumps from high drop heights and finally to landings, it is difficult or even impossible to conclude that the elastic properties of the muscles are best when the reflexes are largest, and thus, the stiffness is highest. For instance, although the leg stiffness was shown to be higher in experienced jumpers (French elite long and triple jumpers) compared with untrained subjects of the same age, the elite jumpers demonstrated a strong but negative correlation between the maximal height reached during hopping and the corresponding leg stiffness (27). Similarly, Laffaye and coworkers (15) reported that elite handball, basketball, and volleyball players as well as high jumpers and novice jumpers decreased their leg stiffness when they augmented their rebound height in a one-leg jump task. Furthermore, it was demonstrated during drop jumps that the greatest power production, but not the

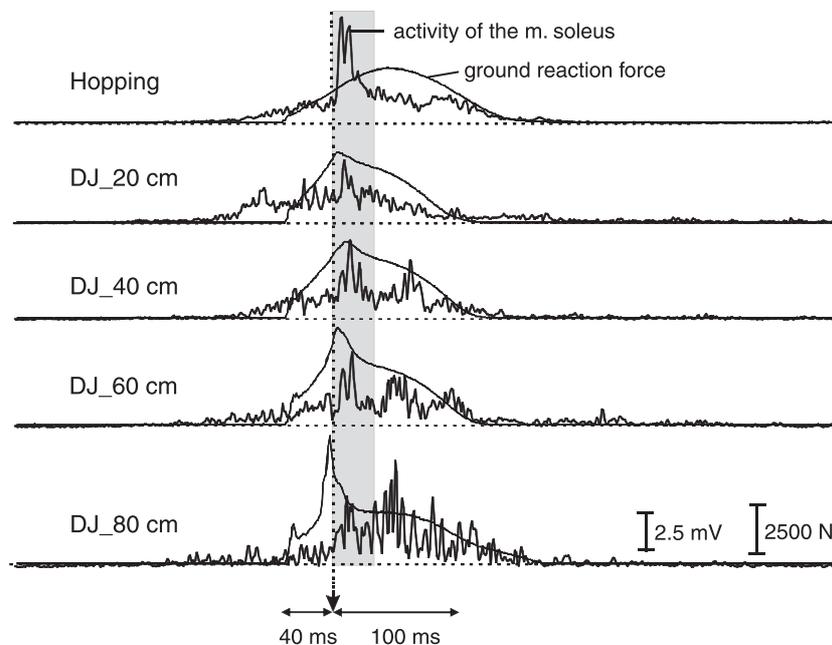


Figure 2. Changes in electromyography (EMG) pattern due to modulation of drop height. Rectified and averaged EMG pattern of the soleus muscle and vertical ground reaction force in various stretch-shortening cycle drop jumps with both legs. The figure illustrates the modulation in the pattern and in the force record with increasing stretch load (drop height). From top: both-leg hopping in place; DJ_20–DJ_80 cm, drop jumps from a drop height of 20–80 cm. It can be seen that the short-latency response (first peak in the EMG after the dotted vertical line) is rather increasing from 20 to 60 cm drop height but is reduced drastically when jumping from 80 cm. [Adapted from (14). Copyright © 1997 Human Kinetics. Used with permission.]

maximal rebound height, was related to a highly stiff system (2). Recently, Cronin and coworkers (5) showed that vibration of the Achilles tendon led to a significant decrease in the size of the SLR during running, and this decrease was accompanied by a reduction in ankle joint stiffness (evidenced as ankle yielding) at low to intermediate running speeds (7–12 km·h⁻¹). However, at 15 km·h⁻¹, vibration still reduced the SLR but had no effect on the stiffness (5). These observations demonstrate that it is in all likelihood wrong to assume that maximal performance is correlated with maximal stiffness (2,15,27) and to suggest that the stiffness is related directly to the magnitude of the SLR peak (5). Both adjustment of stiffness and changes of the SLR, therefore, may follow an optimum function with a u-shape rather than being linear.

In line with those assumptions, longitudinal training studies also emphasized that training-related increases in rebound height may not be primarily associated with changes in the lower leg stiffness but may be more strongly dependent on the compliance of the tendomuscular system (*e.g.*, (29)). In our most recent training study, SSC training from different drop heights resulted in augmented rebound heights, which were accompanied by reduced leg/ankle stiffness and a greater countermovement (knee flexion during the contact phase), but no changes in the muscular activity at the time of the SLR (29). Therefore, based on our observations and according to Rabita *et al.* (27), it may be proposed that to control SSC movements, the CNS is challenged to find the right balance between a powerful stiff system (2) and a more compliant system (29), which probably is better suited to store elastic energy. As a consequence, stretch reflex contributions probably have to be gated specifically to adjust the tendomuscular stiffness and, thus, to meet the criteria of context (*e.g.*, duration of ground contact, rebound height) and environment (*e.g.*, low versus high drop height, surface characteristics). In conclusion, the appropriate neuromuscular activation at the time of the SLR in SSC movements therefore is influenced most likely by multiple factors and can be detected only by studies combining neurophysiological with biomechanical measurements so that interrelations of neuronal control and tendomuscular properties can be identified. Furthermore, the muscular activity at the SLR seems important to adjust the tendomuscular stiffness, but it is certainly not the only determinant.

Modulation of Ia Afferent Transmission During Different Phases of the SSC

Studies investigating the Ia afferent transmission by means of peripheral nerve stimulation during hopping and drop jumping revealed a phase-dependent modulation of the H-reflex: the H-reflex excitability was high during the contact phase but decreased just before push-off and remained low during the flight phase (10,30,33). The functional significance of enhanced Ia-afferent transmission in the early contact phase may be that impulses from the Ia-afferents may enhance motoneuron activation on top of the ongoing EMG activity. Furthermore, it has been argued that afferent feedback can be used to produce a peak impulse by synchronizing the α -motoneurons in the already active soleus muscle at the time of ground contact (10). The progressive decline of the H-reflex amplitude during the contact phase implies that muscular activity in the later contact phase may be

less dependent on Ia-afferent input as in the early phase, suggesting that other sources of neural activity are becoming more prominent. However, it has to be mentioned that in all studies using peripheral nerve stimulation during jumping, factors other than changes in the Ia-transmission may have influenced the size of the H-reflex: muscle length is known to affect the H-reflex size as well as activity in the upper leg muscles like the biceps femoris or the rectus femoris (for references, see (30)). Furthermore, cutaneous afferent input can alter the excitability of the H-reflex. As jumping and hopping are highly dynamic movements involving many muscles and sensory information from numerous different sources, it is not possible to determine the main mechanisms, which are responsible for the H-reflex modulation during the SSC. However, irrespective of the exact underlying mechanism, these studies suggest that spinal reflex excitability assessed via the Ia-afferents is relatively high at touchdown and reduced toward take off.

SUPRASPINAL MECHANISMS CONTRIBUTING TO THE SSC

Motor Cortex and Corticospinal System

The preceding paragraphs elaborate on the contribution of spinal reflex responses to the muscular activity during SSC movements. The following section concentrates on evidence supporting the concept of involvement of supraspinal structures in the SSC. In contrast to sudden and unexpected perturbations, drop jump and hopping allow supraspinal centers to predict accurately the time of ground contact and thus the instant of muscular stretch (*e.g.*, (23)). Therefore, it could be speculated that during the SSC, preprogrammed activation of supraspinal structures may contribute to the muscular activity at any time during the movement. Previous studies assumed that such a centrally preprogrammed muscular activity was important for the preactivation, the reflex modulation, and the stiffness regulation during SSC and landing movements in humans and animals (for references, see (30)). In other words, it was proposed that supraspinal centers do not only initiate jumping and landing movements but also preprogram at least part of the muscular activation pattern after touchdown. However, the source of this activation is not well understood. So far, only two studies investigated the corticospinal activity during drop jumping and hopping by means of transcranial magnetic stimulation (TMS) (30,35). In the first of our TMS studies, modulation of the magnetically elicited motor evoked potential (MEP) was assessed during different phases after ground contact in drop jump (30). The MEPs were small and nonaugmented shortly after ground contact (at the times of the SLR, MLR, and LLR) but were facilitated significantly after approximately 120 ms (LLR2; the activity 120 ms after touchdown was labeled “LLR2” in this study). As this modulation was reciprocal to the modulation of the H-reflex, which was high at SLR and then progressively declined toward the push-off phase, we argued that it is conceivable that corticomotoneurons enhanced their excitability at the time of the LLR2. At the same time, we supposed that the cortical influence was minor in the early contact phase and speculated that the early contact phase may rather be dominated by spinal reflex activity.

It has to be mentioned at this stage that, although in many studies changes in the MEP are compared with changes in the H-reflex to deduce alterations in cortical excitability, such a methodological approach is limited in its validity. This is due to the fact that the MEP is dependent on the excitability of corticomotoneurons, spinal motoneurons, and spinal interneurons (for references, see (31)). Therefore, additional measurements are needed to monitor the responses of the motoneuron pool to separate the spinal from the cortical effects. Although the H-reflex often is used as an “indication” for alterations in the motoneuronal excitability, a drawback of this technique is that the H-reflex is not dependent solely on the excitation level of the motoneuron pool but also is influenced by presynaptic inhibition and homosynaptic postactivation depression. Furthermore, comparison of the MEP and the H-reflex cannot assess excitability changes at the interneuronal level. Apart from these limitations, responses after peripheral nerve stimulation might not always originate from activation of the same population of motor neurons and might be affected differently by changes in the motoneuron excitability than responses mediated via the corticospinal tract. Thus, our first study using TMS during SSC movements (30) illustrated that the corticospinal excitability is enhanced toward push-off, but it could not provide information about the cortical involvement, that is, whether the motor cortex itself contributed to the muscular activation. To assess motor cortical contribution, another TMS method is needed: Davey *et al.* (6) were the first to demonstrate that a single transcranial magnetic stimulus below the threshold to elicit an MEP can produce a suppression in the EMG of a voluntary contracted muscle without prior facilitation. Several control experiments suggested that this TMS-evoked EMG suppression is due to the activation of intracortical inhibitory interneurons, which reduce the output from the motor cortex (6). Thus, whenever motor cortical output exists during movement, subthreshold magnetic stimuli should reduce this output resulting in a decreased muscular activation. Most interestingly, with respect to SSC movements, was whether there is contribution of the motor cortex to the muscular activity at the time of the SLR. As described previously in this review (please see the section “Do spinal (stretch) reflexes occur during SSC?”), the SLR during hopping is influenced by a stretch reflex response (35). Dyhre-Poulsen *et al.* (10) suggested that this stretch reflex response was set on top of the voluntary EMG activity. To test this assumption and to reveal motor cortical contribution at the time of the SLR, we applied low-intensity magnetic stimuli to the motor cortex during hopping (35). The SLR was reduced significantly in response to subthreshold magnetic stimulation, indicating that the SLR in hopping indeed is not only composed of activity resulting from sensory feedback but also influenced by a descending drive from the motor cortex (Fig. 1).

Together with the results of our previous study using TMS (30), showing an increased corticospinal excitability toward push-off despite a progressive reduction in spinal excitability, it may be assumed that motor cortical contribution is present throughout the entire SSC movement, that is, from the initiation of the jump throughout the time of ground contact (SLR, MLR, and LLR) until push-off.

Subcortical Brain Regions

Our knowledge about the specific role of subcortical brain areas in relation to SSC movements is not well advanced, and the importance of these areas has to be deduced rather than based on direct investigation. One possibility for an experimental approach is the consideration of motor deficits in studies with subcortical brain lesions. Patients with cerebellar damage demonstrate impaired jumping abilities. This deficit may be related to their limited postural control. However, recent work suggests that especially predictive, that is, feedforward, control is affected when the cerebellum is damaged, which might be especially relevant for the control of fast and ballistic movements (3). In cerebellar patients, as well as in monkeys where the cerebellum was blocked partly by cooling, execution of fast wrist and finger movements was impaired (*e.g.*, (32)). Thus, it may be speculated that the cerebellum plays an important role in controlling dynamic, time-critical movements like the SSC. However, direct evidence linking the cerebellum to the control of SSC movements is rare. In mice, the formation of the cerebellum seems to be associated with their jumping behavior (for reference, see (30)). In humans, evidence is restricted to motor imagery studies: mental imagery of high jumping during functional magnetic resonance imaging was demonstrated to produce the highest activity in motor regions such as the supplementary motor area, the premotor cortex, and the cerebellum (25). The same athletes who were tested with functional magnetic resonance imaging carried out mental high jump training and could improve their performance in contrast to athletes who continued to perform physical training exclusively (26). One explanation of why motor imagery can enhance actual motor performance is that motor imagery and motor action engage overlapping brain systems. Therefore, it might be assumed that the activity in the cerebellum during the imagined performance of high jumping is related closely to the brain activity during the actual motor task. However, it is not clear so far in which way the cerebellum is involved in coordinating muscular activity during the final SSC, that is, the takeoff before the bar clearance. The results rather emphasize that the complex movement of high jumping from the start of the runway until the landing involves cerebellar activity.

Even less information is available about other subcortical structures, such as the basal ganglia or the brainstem, with respect to their contribution to the control of SSC movements. It would be most unlikely that they were not involved in controlling posture and muscular activity during the SSC.

Interaction of Feedforward and Feedback Control During the SSC

Based on previous observations and the results of our most recent studies, we conceptualized a theoretical framework for the interaction of feedforward and feedback mechanisms during SSC movements (Fig. 3). In this article, the term *feedforward control* (or *predictive control*) refers to the portion of the movement that is planned in advance and is not altered by online peripheral feedback (3). In contrast, feedback or reactive control involves in-flight integration of peripheral feedback into the current movement to provide online reinforcement and/or correction (3). Most of our natural

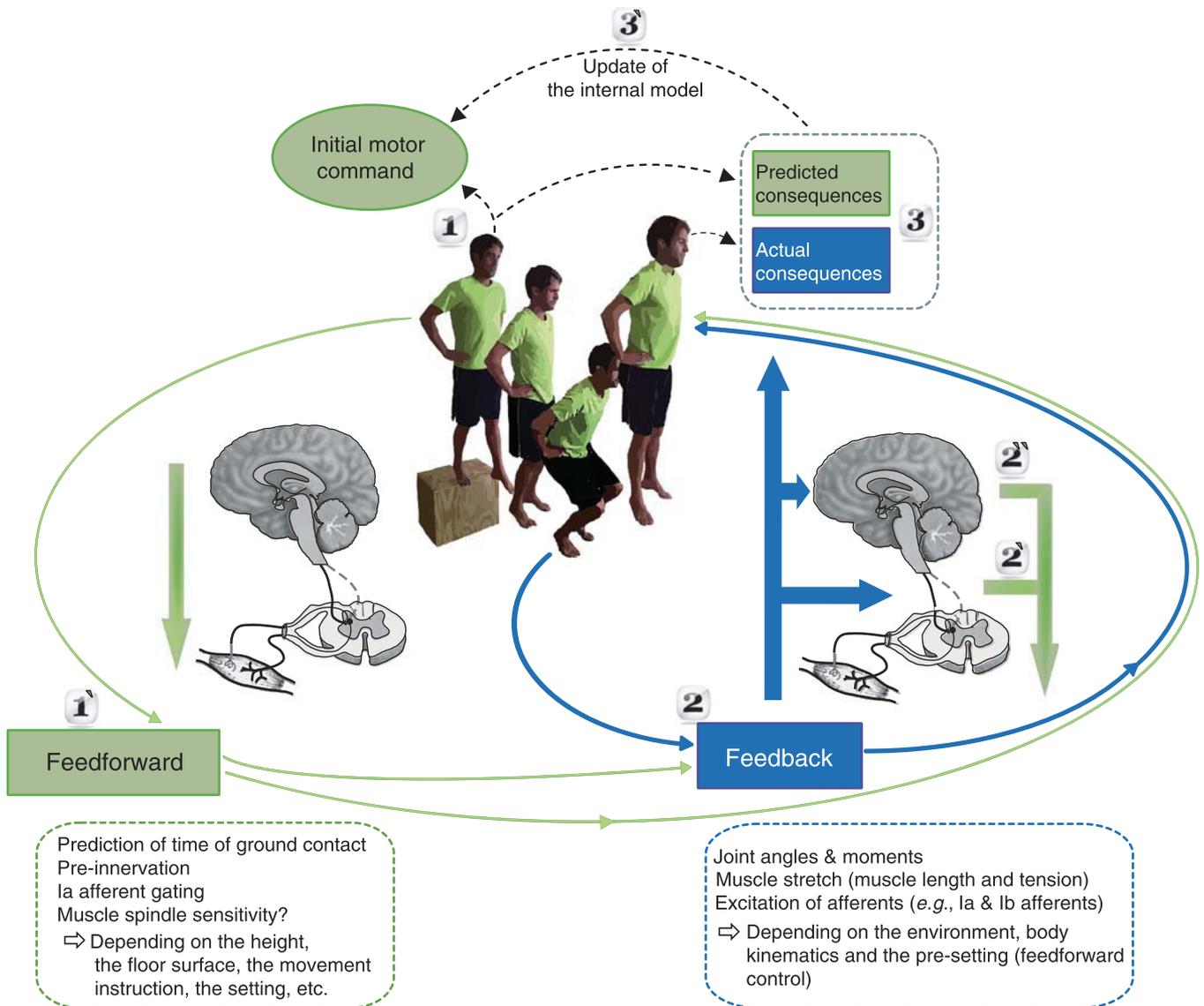


Figure 3. Interaction of feedforward and feedback control during stretch-shortening cycle movement. 1) Initial motor command to initiate the movement and to adjust the system in accordance to the expected environmental setting (1'): The feedforward or predictive motor control refers to the portion of the movement that is planned in advance and is not altered by online peripheral feedback. In case of the drop jump, the instant of ground contact can be estimated, and factors like floor surface, aim of the movement (for instance, "to rebound as fast as possible" or "to rebound as high as possible"), and the stability of the environment (e.g., opponent) can be given consideration. Dependent on the situation (20), the CNS will adjust its activity, like for instance, the amount and duration of preactivation or the Ia afferent gating. 2) At touchdown, peripheral feedback will be generated and can be integrated into the current movement to provide online reinforcement (e.g., activity of the short-latency stretch reflex (2') on top of a supraspinally preprogrammed baseline activity; (35)) and/or correction (for instance, if the CNS miscalculated the instant of touchdown or the properties of the landing surface; (22)). The feedback loop can involve spinal structures (2') or can be traveling via supraspinal centers (2'). 3) The predicted and the actual consequences of the movement are compared. If they are not in agreement, the internal model has to be updated (3'). This may be the case when the biomechanics of the limb or task have been changed. Consequently, the internal model has to adjust the motor output to the new setting by altering the feedforward command and modifying the gating of afferent integration (e.g., the level of presynaptic inhibition at the spinal level). It has been shown that for the update of the internal model during a series of jumps, information about one single miscalculated jump is sufficient to recalibrate appropriately the internal jump model (22). Most probably, subjects use the error between the predicted and the actual consequences (sensory feedback) for recalibration.

movements involve interaction of feedforward and feedback control (Fig. 3).

Feedforward Control

The feedforward component in walking could be shown by demonstrating after-effects after a period of training in a new environment. Similarly, repeated jumping on an elastic surface leads to after-effects when subjects are tested on solid ground afterward (22). These examples indicate that, for a

given task, the motor output has to be aligned and calibrated newly to meet the requirements of the modified environmental conditions. Therefore, it is likely that the CNS possesses an internal model of the dynamics of the limbs and the body to compute the necessary motor output for a desired task in a given setting. When the biomechanical properties of the limb or the requirements of the task are changed, movement errors will occur (28), which are needed for updating the internal model to adjust the motor output to the new setting

(12). The need for feedforward control basically is a consequence of the time delays of sensorimotor loops that limit the rapidity with which the motor system can respond to sensory events (34). Thus, particularly fast movements depend on preprogrammed muscular activity. This also is the case in SSC movements and can be demonstrated easily in blindfolded subjects who perform drop jumps from a certain drop height. When the drop height remains unchanged, the movement pattern after few attempts resembles the one with open eyes. However, when the instant of ground contact is earlier than expected (e.g., advising the subject to jump from a height of 60 cm but offering only 20 cm), subjects are not able to perform a drop jump any more because of a wrongly timed muscular activation (Schmitt S, Baur H, Mayer F, Gollhofer A, unpublished observations, 2006).

The feedforward component also was demonstrated in landing movements of monkeys because the onset of the preactivation EMG pattern was uninfluenced by turning off the light during the fall and by opening a collapsible platform delaying the time of ground contact (9). In a similar approach during human hopping, we have illustrated that lifting and lowering of the landing surface shifted the stretch reflex component forward and backward, respectively (35). However, the initial rise in the soleus background EMG activity always occurred at the same time independent of the position of the landing surface indicating a preprogrammed feedforward control (Fig. 1). It was shown in several studies that the CNS accurately predicts the time of ground contact when jumping down from an elevated platform with open eyes (e.g., (23)). During hopping, the prediction of the time of ground contact seems to be similarly precise, but it may be speculated that the visual information may not be as important as during landings or drop jumps because of the repetitive character of this movement.

In summary, a considerable part of the muscular activity seen in SSC movements seems to be preprogrammed. Thereby, the feedforward control in all likelihood not only affects supraspinal motor commands but also predetermines the integration of afferent feedback. This may be assumed based on adapted stretch and H-reflex activities in drop jumps in response to modulations of the drop height (17,19). In particular, reflex responses were shown to be reduced when subjects were asked to jump from high drop heights (over 60 cm). If the CNS had not decreased the spinal excitability and/or the susceptibility of the fusimotor system, the faster stretch velocities going along with increases in drop height should have resulted in an increased reflex activity (11). As this was not the case, we speculated that the CNS preprograms the level of reflex inhibition based on the drop height to avoid hyperexcitability of the spinal reflex loop, which otherwise might have resulted in extensive muscle-tendon stiffness, potentially causing overload injuries (19). The most likely mechanism to inhibit the spinal reflex circuit is presynaptic inhibition (17,19), where the release of transmitter at the synaptic cleft is reduced. Thus, in cases where the increase in drop-height leads to spinal reflex inhibition, the excitation of the Ia afferents may not be transmitted fully to the postsynaptic neuron (the α -motoneuron). This means that the presynaptic transmitter release is reduced without affecting the postsynaptic side, which still is susceptible to

other inputs. During SSC movements, preprogrammed modulation of presynaptic inhibition may therefore allow the adequate adjustment of spinal reflexes without affecting the input of supraspinal sites to the α -motoneuron pool.

A nice example of where the CNS has misleadingly preprogrammed the neuromuscular activity in SSC movements could be seen in subjects who got accustomed to jumping on an elastic surface and were asked afterward to perform the same kind of jump on solid ground (22). The after-effects included an increase in leg stiffness, decrease in jump height, and perceptual misestimation of the jump height. The authors proposed that the after-effects were due to an erroneous internal model acquired on the elastic surface.

A further study highlighted that the parameterization of the internal model is dependent not only on previous experience but also on the setting of the task (20) (Fig. 4). In the first condition of this study, we instructed the subjects to perform drop jumps from 50 cm ("no switch condition"). In the second condition, subjects also performed drop jumps from 50 cm, but when a tone was presented prior to ground contact, they had to switch from jumping to landing ("potentially switch condition"). We were most interested in

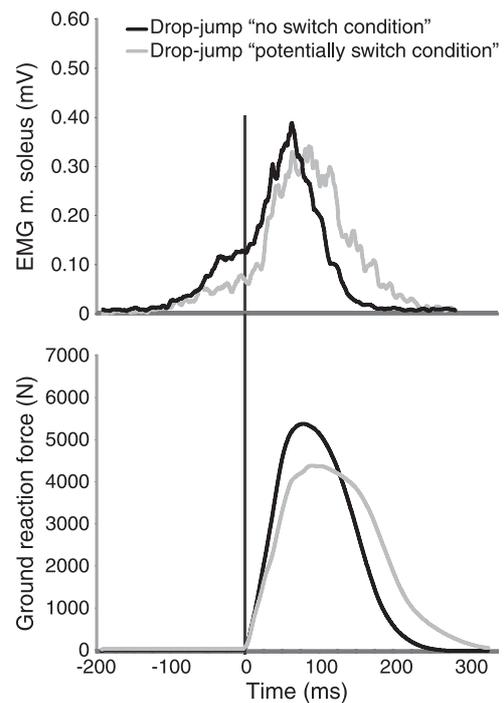


Figure 4. The parametrization of the internal model is dependent on the setting of the task. Shown are grand mean values of the m. soleus electromyography (EMG) and the ground reaction force for two conditions. In the first condition (termed *no switch condition*), subjects performed drop jumps from 50 cm and knew that there will be no sign (auditory cue) indicating that they had to change their movement (i.e., to land). In the second condition, called *potentially switch condition*, the subjects performed the instructed drop jump, and no switch of the movement had to be performed either. However, subjects were aware that an auditory cue could be presented in this condition. Zero on the x-axis refers to ground contact. Data truncated at 340 ms following ground contact. It can be seen that the muscular activation and the resulting ground reaction forces clearly were modulated depending on the performed condition despite biomechanical identical conditions. [Adapted from (20). Copyright © 2011 Elsevier. Used with permission.]

the two conditions where the subject did exactly the same but with different settings, that is, when subjects performed drop jumps from 50 cm and potentially had to switch the movement from a drop jump to a landing (“potentially switch condition”) and, in the other case, when subjects performed the drop jump but could be absolutely sure that no switch signal would occur (“no switch condition”). As the two tasks were identical biomechanically, every change in the movement execution in the “potentially switch condition” must have been caused by a change in the preprogrammed (feedforward) motor command. The results demonstrated that the feedforward control indeed changed so that the muscular activity was kind of between the muscular activity observed during drop jumps in the “no switch condition” and the muscular activity in pure landings (“no switch landing condition”). The changes in the feedforward control were evidenced by a reduced muscular activity of the extensor muscles at the time of the SLR and augmentation of activity toward push-off (Fig. 4). Such a strategy may have allowed a more flexible task execution because the decision whether to apply a drop jump or to land could be “postponed” as the muscular activity shortly before takeoff decided whether subjects rebounded or landed. When rebounding was required, subjects displayed a strong activation toward takeoff, whereas landing was accompanied by a suppression of this activity (20). This study demonstrates that depending on the setting (“no switch condition” versus “potentially switch condition”), the same task may be preprogrammed differently to accomplish the requirements of the situation. At the same time, our study proposes that the integration of afferent feedback also may change depending on the setting. The H-reflexes recorded at the time of the SLR were reduced as soon as the subjects had to potentially alter their movement. Although we could not clarify whether this reduction was due to post or pre-synaptic mechanisms, it nevertheless demonstrates that modulation of the feedforward control affects the integration of spinal reflexes.

In summary, centrally preprogrammed muscular activity can be considered as being extremely important for the organization of SSC movement, especially for the control of the pre-activation, the reflex modulation, and thus, the stiffness regulation (e.g., (19,35)). However, as already mentioned in the section “Spinal Mechanisms Contributing to the SSC,” feedback mechanisms also are integrated into SSC movements (e.g., (35)).

Feedback control

Although feedback mechanisms have the disadvantage of being time lagged, they do allow the system to provide specific responses to certain sensory events. The sensory consequences may be either expected — like the impact of touchdown and the resulting muscle stretch — or unexpected because of changes in the environment or errors in the feedforward program for example. Therefore, it may be speculated that the benefit of using a feedback system during SSC movements relies on the precise timing of the muscular activity through certain sensorimotor loops. For instance, the activation of the monosynaptic stretch reflex circuit leads to contraction of the triceps surae complex some 35–45 ms after touchdown (30).

The delay may vary from subject to subject but, within a subject, is as precise as a few milliseconds. This time-locked reflex response may ensure an appropriate source of activation because of the synchronous activation of the motoneuron pool to reinforce tendomuscular stiffness. Moreover, in cases where the internal model wrongly predicts the time of ground contact, a feedback mechanism has the advantage to generate the muscular activity with reference to the instant of ground contact. When the miscomputation takes place during a series of jumps or during cyclic SSC tasks like hopping or running, the detection of the movement error will be used to update the internal model to adjust the motor output for the next jump. It was shown that, for the updating, only information about one single miscalculated jump is necessary (22). Most likely, subjects use the error between the predicted and the actual sensory feedback to recalibrate their internal jump model (Fig. 3).

CONCLUSIONS AND PROSPECTS

This review illustrates the complex nature of SSC movements, in which the CNS has to coordinate and adjust the contribution of anticipated (feedforward controlled) and reflectory (feedback controlled) neuromuscular activity to provide an appropriate (not maximal) tendomuscular stiffness. To accomplish this task, cortical, subcortical, and spinal levels have to closely interact.

Acknowledgments

This study was supported by the Bundesamt für Sport (BASPO; Switzerland). We are aware that many more authors have worked in the field of (neural control of) SSC movements, but we could not cite their work because of reference limitations.

The authors declare no conflict of interest.

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