Muscle reflexes and synergies triggered by an unexpected support surface height during walking

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Abstract

An important phase in the step cycle is foot contact. When the moment of foot contact differs from the one expected, a fast response is needed. Such a mismatch can be caused by hitting a support surface earlier or later than expected. To investigate this, experiments were performed with healthy young adults who walked on a platform that was unexpectedly at a lowered (5cm) or at a level height. Glasses blocked the lower visual field. In the unexpectedly lowered trials, the absence of expected heel contact triggered responses in the ipsilateral anti-gravity (MGi, RFi) and contralateral flexor muscles (TAc, BFc) with latencies of 47-69 ms. Following the delayed heel contact, enhanced activity was found in the MGi, RFi and TAc muscles. This specific muscle synergy was presumably activated to arrest the forward propulsion of the body. In contrast, when the surface was unexpectedly at level height, the subjects expected to step down and the leg briefly yielded. A muscle synergy was activated at 46-81 ms that flexed the ipsilateral knee (TAi, BFi, RFi) and extended the contralateral one (MGc, BFc) in order to unload the perturbed leg and delay the contralateral swing phase. Both conditions triggered a fast functionally relevant muscle synergy due to a mismatch between the expected and actual sensory feedback at the moment of foot contact. The results are consistent with an internal model that compares the expected with the actual sensory feedback. The short latency of the response suggests a subcortical, possibly cerebellar pathway.
1. Introduction

When walking in a natural environment, ground conditions are constantly changing. One of the most successful models in motor control is based on the idea that we continuously compare our internal model of movement with the actually produced one and use the error signal to adjust the motor output (Wolpert and Miall 1996). Can we use such a model to understand how we ambulate over ground conditions that are constantly changing? An important part within the step cycle is the moment of touchdown since at that point one has to match the leg stiffness with the characteristics of the ground surface. If there is a mismatch, the central nervous system has to react.

The mismatch at the moment of touchdown can be caused by hitting the support surface earlier or later than expected. The involved processes have been studied most extensively during jumps or falls from various heights. The usual strategy is to activate support muscles prior to landing. Most stiffness at landing is caused by pro-active, centrally organized anticipatory muscle activation, which is timed to the expected instant of touch down (Santello 2005). The anticipatory muscle activation is also scaled to the expected stiffness of the landing surface (Kamibayashi and Muro 2005). If the touchdown is earlier than expected, the leg is not sufficiently stiff and the leg yields (Verdini et al. 2006). This brief yield of the leg appears to be related to reduced muscle activity of the quadriceps and tibialis anterior muscles before foot contact during walking. When hopping on an unexpectedly stiff surface, the ankle and knee become more flexed after landing because the extensor activations prior to landing were insufficient (Moritz and Farley 2004). After foot contact, however, leg muscle activations occur with latencies of 68-188 ms, thereby restoring leg stiffness.

If foot contact occurs later than expected, reflex activity after foot contact enhances. In drop falls through a false platform, the post-landing reflex component was increased when the interval
between the false and solid floor was at least 50ms (McDonagh and Duncan 2002). The short interval needed to induce the corrections indicated that the reactions might be mediated subcortically. Do similar mechanisms operate when an unexpected surface height is encountered during walking? When a cat steps in an unexpected hole, there is an absence of loading at the expected time. As a consequence, there is a short latency silencing of the extensor muscle activity (Gorassini et al. 1994). This supports the contention that load feedback provides reinforcing feedback during the stance phase of walking (Duysens and Pearson 1980; for review see Duysens et al. 2000). Grey et al. (2004) described results, which most closely resembled the cat data, with a decrease in extensor activation occurring shortly after the onset of unloading during midstance caused by an induced plantarflexion movement of the ankle. In humans, the importance of load feedback during gait has been emphasized by other authors as well (Dietz and Duysens 2000) but the corrective responses to an unexpected step down or sudden loss of ground support have only recently been examined.

Several studies have used a platform, which dropped down after foot contact. In a study by Nakazawa et al. (2004) a platform dropped 1 cm after the vertical component of the subject’s load had reached 60% of body weight. Marigold and Patla (2005) studied the response to a step on an unexpectedly compliant surface with a drop in ground support of ~ 2cm. Furthermore, Nieuwenhuijzen et al. (2006) have investigated steps on an inverting platform, which also lowered the ground support surface but in a medial-lateral direction only. All these studies found long-latency muscle responses (later than 100 ms) in a range of lower limb muscles that could stabilize the ankle. In contrast to the cat experiments, the individuals made contact with the support surface before and during the lowering of the surface, thereby providing afferent feedback. Furthermore, it is questionable whether a 1-2 cm drop in ground support is sufficient to compare to the cat ‘foot-in-
hole’ studies. Therefore, the following questions were asked in order to identify the responses of healthy subjects to an unexpected change in support surface height during walking:

(i) Does the absence of expected load feedback in an unexpected step-down trigger fast reflex activity?

(ii) Does stepping down unexpectedly result in enhanced post-landing muscle activity compared to stepping down expectedly?

(iii) Does an unexpectedly high support surface result in fast reflex activity to compensate for the premature onset of loading?

To answer these questions, subjects stepped down and onto a level surface, both with and without awareness of the surface height. The conditions can be characterized by three important time periods; (1) prior to expected foot contact, (2) between expected and actual foot contact, (3) post-landing. In the case of an unexpected step-down, the subjects approach the surface without awareness of the surface condition (period 1). The absence of expected foot contact is likely to trigger an error signal prior to the actual foot contact (period 2), followed by a strong delayed foot contact, which may induce enhanced post-landing activity (period 3). To test this paradigm, the unexpected step-downs are studied under two contexts; first with respect to unexpected level walking, and second with respect to stepping down expectedly. In unexpected level walking, the subjects are not aware of the support surface condition. Therefore, the surface is approached similarly as in the unexpected step-down condition. As a consequence, the pre-programmed anticipatory muscle activity prior to the moment of the expected foot contact is the same. This allows us to determine the exact onset latency and muscle synergy triggered by the absence of the
expected load feedback in order to answer question (i). This comparison is analogue to the comparison used in the cat ‘foot-in-hole’ experiments.

In the second comparison the physical conditions of the task are the same (step-down), while the level of expectancy differs. This allows us to answer question (ii); whether post-landing muscle activity (period 3) is enhanced as a consequence of the unexpectedness of the step-down. To answer question (iii), unexpected level walking is compared with expected level walking. In unexpected level walking, subjects are expecting a step-down (period 1). Hence, their preparation of a step-down is interrupted by a foot contact that arrives earlier than expected. This allows us to study the effects of premature onset of loading on post-landing reflex activity (period 3).
2. Methods

2.1 Subjects

Twelve young adults (5 male) participated in the study (mean ± sd; age 24 ± 3 yrs, height 1.76 ± 0.12 m, body weight 67 ± 13 kg). None of the subjects had a neurological or musculoskeletal disease that could affect the behavioural responses. The experiment was performed in accordance with the Declaration of Helsinki and approved by the regional ethics committee.

2.2 Experimental set-up and Instrumentation

The experimental set-up is shown systematically in figure 1A. It consisted of a 2.6 m long wooden walkway ending on a gravity driven platform of 1 square meter, supported at each corner by two electromagnets (Commissaris et al. 2002). By releasing the electromagnets, the platform could lower the ground support surface by 5.0 cm. Subjects were instructed to walk 4 steps, starting with their right foot, and then come to a full stop with their feet placed next to each other. The third step, with the right foot, was the step on the platform. This step is referred to as ipsilateral (i). The step prior to the step on the platform, with the left foot, is referred to as contralateral (c, see fig. 1A). All subjects wore a safety harness attached to the ceiling. Furthermore, subjects wore glasses that blocked the lower part of their visual field to deprive them from visual feedback of the platform position. During the trials, subjects were instructed to look at a marker on a wall straight ahead, to control for head position. Step length and cadence were standardized by foot placement instructions and the use of a metronome. Subjects performed several practice trials to become familiar with the correct foot placement and cadence prior to data collection.

[Figure 1]
Surface electromyography (EMG) activity was recorded bilaterally from the tibialis anterior (TA), medial gastrocnemius (MG), rectus femoris (RF) and biceps femoris (BF) muscles. The skin of the subject was shaved and treated with scrub gel and alcohol to reduce the skin impedance to a value below 10 kOhm. Two electrodes (Conmed, children’s ECG, 2.0 cm diameter) were placed adjacent to each other (inter-electrode distance of 2.0 cm) on the centre of the muscle belly, in parallel with the direction of the muscle fibres. Signals were pre-amplified (x 10⁴- 10⁵), full-wave rectified, low (300 Hz), and high-pass (3 Hz) filtered. Electrogoniometers (home-made potentiometers) were placed on the lateral part of both knees and the right ankle to measure the joint trajectories. Heel and toe contact, was measured by footswitches (designed in collaboration with Algra Fotometaal b.v., Wormerveer, The Netherlands) that were placed on insoles in gymnastic shoes. Force plates (Dtaxe Weighing system, type 104A) were placed underneath the four corners of the platform. The steep increase in the force signal closest to the right foot followed the footswitch signal by on average 7.4 ms, presumably due to damping (fig 1B). When the footswitch data was missing, the force signal was used for determining the moment of actual foot contact (AHC), taking into account the 7.4 ms delay. In the case of an unexpected step-down, foot contact did not occur at the expected moment. The timing of expected heel contact (EHC) was measured by an infrared system, consisting of two horizontal transmitters and receivers, placed at level height (see dashed arrow, fig 1B). All signals were recorded from 1s prior to 2s after heel contact of the contralateral step, as was detected by a force-sensitive trigger that was embedded in the wooden walkway. All data was sampled with a frequency of 1kHz. Data was analog-to-digital converted and stored for further off-line analysis. An RSScan pressure plate that was placed on the platform, was
used to detect the area of first foot contact (RsScan International ®, Olen, Belgium; 0.4 x 0.5 m, 2 sensors/ cm², 500 Hz).

2.3 Protocol

The experimental definitions and protocol are schematically illustrated in figure 1C. The series of trials were divided in two categories; expected (E) and unexpected (U; see left side of Fig. 1C, <definitions>). The U trials were preceded by the warning that the platform might be lowered 5 cm during each of the following trials. Strictly speaking, the lowered platform trials were not 100% unexpected, since the subjects were aware of the possibility of a lowered platform. Nevertheless, this terminology was used to distinguish these from the second group, the expected trials (E). In the latter trials the subjects were told beforehand whether the platform was lowered or not. Hence we use the labels E and U in the sense of <aware> and <unaware>, but we prefer the expectancy label (E, U) because it better describes the anticipatory state of the subjects, which was the topic of the present study. Furthermore, the trials received a second label since they were also divided in two categories based on the physical condition of the landing surface. The level trials were termed level (L), the lowered platform trials were termed down (D). Hence, for example, an ED trial was a trial in which the subjects expected (E) the platform to be down (D).

Subjects started the protocol with 10 trials of expected level walking (EL, expected level; see top line in right side of Fig. 1C, <protocol>). Next, the platform was unexpectedly lowered 9 times (UD, unexpected down, hence UD 1, UD 2, etc.) in a series in which each UD was followed by 8 - 10 trials of level walking (UL, unexpected level, hence UL1, UL2, etc.). The exact number of UL trials varied to prevent the subjects from being able to predict the next UD (see second line in Fig. 1C, <protocol>). In the last part of the experiment, the subjects performed 10 expected step-
down trials (ED, expected down, see third line in Fig. 1C, <protocol>). All UL trials were intrinsically unexpected, since the subjects were always in anticipation of a possible down trial. However, the level of anticipation differed within the series of UL trials. The first UL trial after a UD (UL1) was characterized by a strong expectation of another step-down. This induced a brief yield of the leg at the moment of foot contact, as was characterised by a short unloading phase. Hence, the UL1 trials represent the level walking trials where subjects were most biased towards expecting a step-down. Therefore, they were grouped separately for further analysis on the concomitant EMG responses. In the subsequent UL trials, the subjects gradually adapted, which was characterised by a decrease in the amplitude and duration of the unloading phase. During the UL $\geq$ 7 trials the duration of the unloading phase was significantly decreased compared with the UL1 trials, whereas it was still increased compared with the EL trials. Apparently, the walking pattern of the subjects had stabilized and was no longer strongly biased towards expecting a step-down. However, this walking pattern was still slightly different from expected level walking. Because of these two reasons, the trials UL $\geq$ 7 were selected as control trials to compare with the UD condition (UD-UL).

2.4 Data analysis

The EMG data of all subjects were normalized with respect to the maximum EMG that was measured during a UL $\geq$ 7 trial. Maximum EMG was determined for each muscle, by calculating a moving average over a window of 30 ms. The responses of the UD trials were investigated with regard to two conditions, UL and ED. For the first comparison, UD- UL, the data were aligned such that the expected heel contact of the UD trials (EHC) coincided with the actual heel contact (AHC).
of the UL trials. For the second comparison, UD-ED, the data of both conditions were aligned at the moment of actual heel contact (AHC).

The amplitude of the difference in EMG activity was calculated by a subtraction method. For each subject, the average normalized EMG data of the UL and ED trials (for the first and second comparison, respectively) was subtracted from the average of the UD 2-9 trials. The subtracted data was divided in 29 bins of 10 ms, starting at EHC of the UD trials and AHC of the UL trials. The EHC of the UD trials was on average 90 ms prior to AHC. Afterwards, the muscle activity per bin was averaged for all subjects.

The onset latency of the EMG responses was determined by visual inspection. For each subject and muscle, the mean of the UD trials were plotted together with the mean ± 2 x SD of the UL condition. For graphic use only, the EMG trials were filtered (zero-lag, second order Butterworth filter, low-pass 50 Hz). Using a custom-made MATLAB program, onset latencies were determined by placing a cursor at the moment where the UD curve exceeded the mean ± 2 x standard deviation (SD) of the UL curve for a period of at least 30 ms. Afterwards, group averages of the onset latencies were calculated for all muscles.

As appeared from analysis of the landing strategies, subjects occasionally landed flatfooted or on their forefoot instead of with their heel first in the UD condition. Nevertheless, the heel landing was the most commonly used strategy (72.5%). Therefore, only heel landings were selected for further analysis.

2.5 Statistical analysis

To verify if the average EMG amplitudes of the 10 ms bins were significantly different from zero, multiple one-sample t-tests were performed. A one-way ANOVA was used to test whether the onset
latencies differed across muscles and whether the onset latencies of the first trial differed from the onset latencies of the subsequent trials. An alpha level of 0.05 was chosen for the level of significance.
3. Results

All subjects successfully performed the test protocol. In some trials, subjects unexpectedly stepped down, leading to a delay in the moment of foot contact. This will be elaborated on in section 3.1 and 3.2. At other times, the surface was unexpectedly at level height, leading to a premature onset of loading, which will be described in section 3.3. The average normalised EMG responses of a typical subject are illustrated in figure 2 for all experimental conditions. The most striking result was the large increase in muscle activity in the UD condition. The absence of foot contact at expected heel contact (EHC, dashed line) substantially increased the activation of several ipsilateral and contralateral muscles around actual heel contact (AHC, solid line). To determine the exact onset of this muscle activity, the UD condition was compared with the UL condition. In both of these conditions, the subjects were unaware of the height of the support surface. Therefore the support surface was approached similarly and the pre-perturbation EMG activity was the same.

[Figure 2]

3.1 Does the absence of expected load feedback in an unexpected step-down trigger fast reflex activity?

The reflex activity as a consequence of stepping down unexpectedly was studied by comparing the UD with the UL condition. Illustrated in the upper left panel of figure 3 is the average MG\textsuperscript{i} activity in the UD and UL condition for one subject. At the moment of EHC, the subjects’ pre-programmed muscle activity was similar for both conditions. In contrast, a large increase in MG\textsuperscript{i} activity could be observed well before AHC. To further explore this difference, the EMG responses of both conditions were subtracted from each other and averaged for the whole population. The absence of
foot contact at EHC induced reflex activity in the ipsilateral antigravity muscles (MGi, RFi) and contralateral flexor muscles (TAc, BFc), well before AHC (see horizontal grey bars, Fig. 3). Simultaneously, there was a slight but significant decrease in the BFi activity. The decrease was the result of silencing of the BF activity that usually occurred soon after AHC, as was the case for the UL trials. The onset latencies ranged from 47-69 ms after EHC (see arrows in Fig. 3), which corresponds to 21-43 ms prior to AHC. The onset latencies did not differ significantly from each other. Nevertheless, a general pattern was observed. The MGi, BFi, TAc and BFc muscles showed the first responses at 47-57 ms after EHC, followed by the RFi at 69 ms after EHC. In most subjects, the RFc was activated as well, with an average onset of 69 ms after EHC. Due to a larger variability between the subjects, this response did not reach the level of significance for the whole group, except for the very late part of the response. The extra muscle activity continued for at least 200 ms in all ipsilateral muscles, and to a lesser extent in the contralateral muscles.

[Figure 3]

The first UD trial (UD 1) is likely to yield responses that differ fundamentally from those obtained afterwards, since the subjects have not yet experienced the sensation of an unexpected step-down. To examine this, the responses of the UD 1 trials were compared with the responses of the subsequent UD trials (UD 2-9). The results are shown in figure 4 for the ipsilateral MGi and contralateral TAc. As can be seen, the onset latency and duration of the muscle activations were similar, whereas the EMG amplitudes of the UD 1 trials exceeded those of the subsequent ones. This was found for the other muscles as well.
3.2 Does stepping down unexpectedly result in enhanced post-landing muscle activity compared to stepping down expectedly?

To study the effects of expectancy on post-landing muscle activity, the UD trials were compared with the ED trials, in which the physical condition of the task was the same (step-down), while the level of expectancy differed (U versus E). Three subjects had to be excluded from the ED condition because they accidentally had vision of the support surface by removing the blocking glasses. The UD vs ED comparison showed increased post-landing EMG activity, as is illustrated in figure 5. Increased activity was found in the extensor muscles on the ipsilateral side (MGi and RFi), as well as in the contralateral TAc and to a lesser extent BFc. This increased post-landing activity had durations of 70-180 ms (see Fig. 5) starting already at the moment of AHC. In contrast, post-landing EMG activity in the UD vs UL comparison (see Fig. 3) was observed in other muscles as well (TAi, BFi, RFc). This was presumably caused by the difference in the mechanical situation (step-down versus level walking).

Some other marked differences were observed between the two comparisons. Increased muscle activity prior to AHC was found in the MGi, RFi, TAc and BFc muscles (see Fig. 5). This increased activity might be caused by a difference in pre-programmed muscle activity or as a reflex response to passing EHC. Since the increased RFi activity prior to AHC started earlier in figure 5 compared to figure 3, part of the activity was probably caused by a difference in pre-programmed muscle activity. Furthermore, the decreased BFi activity, as observed in figure 3, was not present in figure 5, because now all data were aligned at AHC (while in Fig. 3 the AHC of the UL condition preceded the AHC of the UD condition).
Since the EMG of the UD and ED condition differed from each other, changes might also be found in the underlying kinesiology, as will be examined next. The kinesiology of the UD and ED condition differed from each other with respect to certain aspects (see Fig. 6, left column). In the UD trials, the ankle was slightly more plantar flexed before AHC, suggestive of a more flat-footed landing, which may have been caused by the increased MGi activity. After AHC, the ipsilateral ankle dorsiflexed more rapidly compared to the ED condition. Furthermore, the ipsilateral knee extended, instead of the knee flexion that was observed after AHC in the ED condition. This might be caused by the increased RFi activity before and after AHC. On the contralateral side, the knee rapidly flexed, probably due to the early activity of the contralateral TAc and BFc.

To determine whether the unexpectedness had consequences on the kinesiology of level walking as well, the UL condition was compared with the EL condition (see Fig. 6, right column). The ankle was slightly more plantar flexed in the UL condition just before heel contact, which indicates a more flatfooted walking pattern. Otherwise, the kinesiology of the UL and EL condition were largely comparable for most of the part of the step cycle. It has to be noted that the kinesiology of the UL condition represents the average of the UL ≥ 7 trials. Whether the results were different for the level walking trials that were most unexpected (UL 1) is investigated next.
3.3 Does an unexpectedly high support surface result in fast reflex activity to compensate for the premature onset of loading?

In the UL 1 trials, immediately following a UD trial, the subjects clearly anticipated another step down since they consistently showed an increased load-rate during the first 30 ms of foot contact, followed by a brief yield in the vertical ground reaction force (vGRF). This is illustrated at the top of figure 7 for a typical subject, in which the average of the UL 1 trials was compared with the average of the expected level walking trials (EL).

Prior to touchdown, the ankle was in a more plantar flexed position, which is typical for subjects preparing to land on a lowered surface. The unexpected early foot contact resulted in a high load-rate. Since the stiffness of the leg was not appropriately sized, the subject was not able to take up the high load and the leg briefly collapsed as is shown by the yield in the vGRF curve (see shaded area in Fig. 7, 48-78 ms after foot contact). At the onset of the unloading phase, the knee and ankle started to rapidly (dorsi)flex. Simultaneously, reflex activity was generated in several ipsilateral and contralateral leg muscles such as for example the BFi and BFc. The clear bursts ended simultaneously with the end of the unloading phase. At 100 ms after touchdown, a delay in the contralateral knee flexion was observed, which coincided with late extensor bursts in the TAc and RFc. The delayed flexion resulted in a 26 ms delay in the moment of contralateral toe-off (cTO), as is indicated by the dashed vertical lines in figure 7. Next, the data were subtracted from each other and averaged for the whole population (see Fig. 8).
The kinematic data showed increased ipsilateral ankle plantar flexion prior to touchdown, corresponding with a decrease in TAi activity. During the yield of the leg at 40-78 ms after AHC, the ipsilateral ankle and knee (dorsi)flexed (see shaded area in Fig. 8). Simultaneously, the TAi, RFi, and BFi muscle generated extra EMG activity with latencies of 46-65 ms after AHC. Significant onset latencies were detected in 11 of the 12 subjects for the ipsilateral upper leg muscles RFi and BFi. The bins showed a significant increase in EMG activity with a duration of 60 ms and 80 ms respectively. The burst onset of the TAi muscle was less consistent and was detected in 7 of the 12 subjects. Because of the larger variability between the subjects, only the later part of the response of the TAi was significant. On the contralateral side, clear facilitatory responses were seen in the MGc and BFc with latencies of 66 and 81 ms, resulting in a delayed knee flexion and a 35 ms delay in the moment of cTO. These bursts were detected in, respectively, 8 and 6 subjects and were therefore less consistent than the muscle bursts on the ipsilateral side. The RFc seemed involved in the contralateral activation as well. However, the average amplitude of the response was not significantly different from zero. Overall, the responses to an unexpected level surface (UL 1) were more subtle than those observed after an unexpected step-down (UD).
4. Discussion

Landing on a surface that is either lower or higher than expected triggers, respectively, a fast braking reaction or a brief yield of the leg.

4.1 Unexpected step-down triggers fast reflexive braking reaction

In the case of a lower than expected surface the main finding was that EMG responses in various leg muscles occurred very soon after passing the level surface (47-69 ms) and well before the actual heel contact (21-43 ms). The muscle synergy involved a brief braking reaction, which was achieved through short-latency activations in a number of muscles. On the ipsilateral side the MGi was the first muscle activated followed closely by the RFi. Simultaneously the ongoing activity of the BFi was suppressed. This induced an ankle plantar flexion and knee extension. On the contralateral side the TAc and BFc were activated, resulting in a large knee flexion. The latencies were barely longer than the one observed in the ipsilateral MGi, indicating that the response had a bilateral nature. The observed synergy showed a remarkable resemblance with the one described by Hase and Stein (1998) with latencies of 150-200ms, in an experiment in which subjects were required to walk and then stop abruptly after they received a non-nociceptive electrical stimulation of the peroneal nerve above the ankle (resemblance schematically illustrated in fig. 9A and B). In the present study it is shown that a similar synergy can also be elicited as a natural response to an unexpected absence of ground support surface as soon as 47 ms after passing the level surface (EHC).

[Figure 9]
4.2 Origin of the fast braking reaction

The question arises as to which pathways may have been responsible for delivering the error signal to the CNS that triggered the fast braking reaction. The trigger for this response must have been the absence of foot contact at the point of expected landing. This implies that there are very fast pathways signaling the absence of expected foot contact. Fast responses during perturbations of walking might be attributable to monosynaptic stretch reflexes. However, the most obvious responses in the present study are seen in the MGi and TAc. These muscles were not lengthened but shortened due to the delayed heel contact and ankle plantarflexion on the ipsilateral side, and forward propulsion of the body on the contralateral side. This makes involvement of stretch reflexes in the braking reaction not plausible.

Second, the reaction might be caused by the absence of the load-reinforcing reflex. For loading it is known from cat work that there are extensor reinforcing reflexes that appear preferentially during locomotion (Duysens and Pearson 1980; Duysens et al. 2000). When cats step in an unexpected hole, it was shown that there is a short latency (35 ms) decrease in the ongoing activity of the ankle extensor muscle LGM (Gorassini et al. 1994). Later experiments by Hiebert et and Pearson (1999) showed that it was possible to compensate for the EMG silence by stimulating extensor nerves, thereby providing evidence for the contention that the EMG silence was due to absence of proprioceptive input from these extensor muscles. In the present study, a suppression of the extensor muscles as a consequence of the absence of foot contact was not found. However, the present experiment differed from the cat studies in several important aspects. First, there was no extensor activity around heel contact in humans during normal walking, which makes it impossible to demonstrate EMG silencing after passing the expected heel contact. Grey et al. (2004) were able to demonstrate short latency extensor muscle silencing during a rapid plantar flexion movement in
humans, but this was done during mid-stance when the extensor muscles were active. A second important difference is that cats are quadrupeds and therefore balance is much less threatened than in the biped human. Moreover, a different corrective response is required. The cats stepped on a treadmill and hence had to continue stepping after the perturbation, which required a substantial activation of the flexor muscles. In contrast, the present subjects stopped walking briefly after stepping on the lowered surface. Altogether, the absence of the extensor-reinforcing reflex is not likely to have caused the fast braking reaction that was observed.

Next, it might be argued that the load receptors on the contralateral side (the standing leg) experienced an increased load at the transition of stance to swing. Because of the forward propulsion and downward movement of the body and the delay of the ipsilateral foot contact and thus double support phase, the load underneath the forefoot of the contralateral leg increased and was lengthened with respect to a normal phase transition. Moreover, onset latencies of the ipsilateral and contralateral leg did not differ significantly from each other, indicating that the response might have originated from the contralateral leg as well.

Another contributor to the corrective response might have been the vestibular system, detecting the increased forward rotation of the body and the drop in surface height. It is unlikely however that the fastest responses were vestibularly induced, since the vestibulo-spinal tract requires at least 60 ms to reach the peripheral muscles (Britton et al. 1993). Nevertheless, the vestibular system might have played a role in the later parts of the corrective response.

4.3 Braking reaction as a result of an internal model comparison

In the present study, the CNS expected strong sensory feedback at the moment of predicted foot contact. The absence of the expected feedback acted as a very powerful stimulus to initiate the fast
braking reaction that was observed. The current findings show great resemblance with the paradigms of several studies of reaching, grasping and aiming (Wolpert and Miall 1996; Blakemore et al. 1998). These studies are in favor of a theoretical model in which there is a continuous comparison between the expected feedback and the real-time sensory feedback. An efference copy of the motor command is used to predict the sensory consequences the ongoing action. This is compared with the actual sensory feedback of the movement. A discrepancy between the two will lead to a fast adjustment of the motor command.

Several suggestions are made as to where this comparison is made: at a spinal, cerebellar or transcortical level. In cat, the responses to the absence of expected load suggested spinal involvement. To determine the spinal contribution to the corrective response, Hiebert et al. (1994) studied the responses to stepping in an unexpected hole in spinal cat. The responses were delayed and of smaller amplitude, indicating that spinal pathways could generate at least part of the response, but that supraspinal input was necessary for a fast onset and an appropriately scaled response. The ability to generate complex reactions as a response to postural perturbations and loading was also shown in cats with a high level of decerebration (so-called "premammilary cats", Nichols et al. 1987), indicating that the presence of the brainstem or cerebellum in addition to the spinal pathways was sufficient to evoke these types of responses. In human infants, who are generally believed to have an immature corticospinal tract, and therefore reduced supraspinal input, widespread task and phase-dependent reactions were found as well (Lam et al. 2003). Following stroke that damages the supraspinal structures, it was found that extensor muscles were still modulated based on loading (Marigold et al. 2004). Moreover, in humans with a spinal cord lesion it was shown that the load compensating mechanisms are still operational (Dietz et al. 2002; Dietz and Duysens 2000). These studies indicate that a large proportion of the responses to postural
perturbations in humans might be stored at a spinal level. Could supraspinal structures play a role as well?

The cerebellum is though to be an important structure involved in comparing the sensory reaﬀerence with the predicted sensory feedback (Wolpert et al. 1998). Animal and human studies have shown that cerebellar integrity is needed to respond in time and accurately to unexpected perturbations of arm movements (Shimansky et al. 2004, Timmann et al. 2007). However, the fast latencies that were observed in the current study (47-69 ms) are too short for transcerebellar loops. Cerebellar cooling in monkeys has shown a decrease in the M2 amplitude (Hore and Villis 1984), which occurs at an average latency of 69ms in human (Peterson et al 1998). The presently observed latencies are also too short for transcortical loops. Evidence from SEP registration and MEP stimulation demonstrated that the transcortical pathway for the TA muscle is at least 70 ms (Nielsen et al. 1997).

In the current study, the subjects were able to predict the moment at which the perturbation occurred (always at right heel contact). Therefore, the cerebellum might have primed the spinal structures of the possibility of an upcoming perturbation, as previously suggested by Shimansky et al. (2004) for a different paradigm. This abolishes the need for a transcerebellar loop, thereby reducing the onset latency of the corrective response to spinal levels. The fast braking reaction as observed in the present study, had the same onset latencies during the first as compared to the subsequent trials. This suggests that the spinal structures were already correctly primed before the subjects had experienced the nature of the perturbation. Since the moment of heel contact is a very important phase within the step cycle, the cerebellum might always pre-program the spinal structures prior to the moment of foot contact with the expected sensory feedback. As a consequence, a fast modification can be generated if necessary. The actual sensory feedback is
likely to be responsible for shaping the compensatory synergies, such that they result in appropriate responses to the given perturbation.

Future studies, with unexpected perturbations during different phases of the step cycle can shed light on the question whether these fast latencies can be evoked during other phases of the step cycle as well. Moreover, the exact role of the cerebellum in this reaction might be clarified by testing the current paradigm with patients suffering from cerebellar damage.

4.4 Stepping down unexpectedly results in enhanced post-landing muscle activity

The post-landing muscle activity of stepping down unexpectedly was higher compared to stepping down expectedly. Apparently, the absence of expected foot contact in the unexpected step down triggered enhanced muscle activity, partly similar to what was observed in passing a false floor during jumping (McDonagh and Duncan 2002). The latter study found enhanced activation of the antigravity muscles (MG, RF and BF) soon after impact (35-80ms). In the current study, enhanced post-landing activity was found in the ipsilateral MG and RF as well. However, the increased activity did not show sharp onsets at an equivalent latency. This discrepancy may have been caused by several factors. First, landing during walking is much smoother than after a jump. Furthermore, the priority in jumping is to damp the impact, while in the current setting maintaining balance and rapidly terminate the forward propulsion of the body is more important. Therefore, the post-landing reflex activity may have been concealed by the fast braking reaction that was already generated shortly after EHC.

4.5 Unexpected level walking triggers fast flexor response
In the unexpected level walking condition, the support surface was higher than expected. At the moment of foot contact, the limb was not yet prepared for impact as was shown by the increased plantar flexion and diminished activation of the TAi. As a consequence, the leg yielded, resulting in a brief unloading period at 40-78ms after touch down. The unloading period was similar to the one observed by Verdini et al. (2006), who found it to be related with reduced muscle activity prior to touch down as well.

The yield of the leg coincided with reflex responses at 46-81 ms after foot contact. Apparently, the unexpected early foot contact acted as a very powerful stimulus to trigger fast reflexive responses. The muscle synergy consisted of an early activation of the ipsilateral BF muscle (46 ms), followed by activations of the TAi, RFi and MGc (63-66 ms) and BFc (81 ms). The onset latencies were slightly faster than those observed after hopping on an unexpectedly hard surface (68-188ms; Moritz and Farley 2004)). The reflexes in TAi, RFi and BF might be a result of stretch. Although not all of these muscles were stretched as a result of the early impact, it has been shown in catching that stretch can induce simultaneous reactions in the antagonists as well as the agonists, probably in order to stabilize the joints (Lacquaniti and Maioli 1987). Alternatively, the reflex responses may have been induced by the cutaneous receptors of the foot sole. The muscle synergy of both the ipsilateral and contralateral leg resembled muscle activations that were found during stumbling (Eng et al. 1994; Schillings et al. 2000, compare Fig. 9C and Fig. 9D). The ipsilateral responses generated an ankle and knee flexion, while the activity of the contralateral muscles (MGc, BFc) generated a knee extension that delayed the moment of contralateral toe-off. The reflexive bursts, particularly of the BF muscles, ended simultaneously with the end of the unloading phase, indicating that active reloading of the leg resumed immediately after the flexion response had ended.
5. Conclusion

A difference between expected and actual loading at foot contact can trigger fast functionally relevant corrective muscle synergies, both on an unexpectedly high and low support surface. The muscle synergies and kinematic responses depend strongly on the perturbation and are almost the exact opposite of each other, which can be seen when comparing figure 9A with 9C. However, the onset latencies are very similar. This finding is in favour of the presence of an internal model, continuously comparing the expected with the actual sensory feedback. The resulting error signal is able to trigger fast responses, which are appropriately coordinated to ensure stability in the light of very different perturbations.

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Figure 1: Methods; (A) Experimental set-up. A 2.6m walkway was built, which embedded a platform that could be lowered 5 cm before foot contact. The step on the platform is referred to as ipsilateral (i), the pre-platform step as contralateral (c). (B) Illustrated is the moment of expected heel contact (EHC) as measured by an interruption in the infrared signal (IR). The moment of actual heel contact (AHC) was measured by footswitch signals, placed on the insoles of the subjects’ shoes. Simultaneously, force transducers under the platform measured vertical ground reaction force. (C) Schematic overview of the experimental definitions and protocol.

Figure 2: Average EMG amplitude of subject GE for the medial gastrocnemius (MG), tibialis anterior (TA), rectus femoris (RF) and biceps femoris (BF) muscles. Illustrated are the conditions expected level (EL), unexpected level (UL ≥ 7), expected step-down (ED) and unexpected step-down (UD 2-9). Each trace represents the average ± two times the standard deviation of the EMG of 10 trials (9 trials for the UD condition). The solid line indicates the moment of actual heel contact (AHC), the dotted line the moment of expected heel contact (EHC). The moment of EHC preceded AHC with on average 90 ms.

Figure 3: Unexpected down (UD) versus unexpected level walking (UL). The top of the figure shows the average MGi amplitude of the unexpected down (UD 2-9) and unexpected level (UL ≥ 7) condition of subject GE (N=1). The other panels show the average response amplitudes of the UD 2-9 condition when the average of the UL ≥ 7 condition was subtracted out (thus UD-UL). Each bin represents the group average (N=12) of the response for a period of 10ms; error bars represent the standard deviation. Traces start at the moment of expected heel contact (EHC) of UD, which was aligned with the actual foot contact of UL (AHC UL). The dotted line illustrates the moment of
actual heel contact (AHC) of UD. A positive deflection implies enhanced UD muscle activity; a negative deflection implies a decreased UD activity as a consequence of muscle silencing. Bin amplitudes with a statistically significant difference from zero (p< 0.05) are marked by the grey bars on the x-axis. The onset latencies of the responses (L) are indicated by arrows. Onset latencies were determined with respect to the moment of EHC by a visual detection method. MG = medial gastocnemius, TA = tibialis anterior, RF = rectus femoris, BF = biceps femoris, i = ipsilateral, c = contralateral, L= onset latency, N= number of subjects.

Figure 4: First trial responses. Typical muscle response profiles of the average UD 1 (thin line) and UD 2-9 trials (thick line) when the ensemble average of the UL ≥ 7 trials was subtracted out (thus UD-UL), as calculated for all subjects (N=12). Traces start at the moment of expected heel contact (EHC) of UD, which was aligned with the actual foot contact of UL (AHC UL). The dotted line illustrates the moment of actual heel contact (AHC) for the UD condition. Each bin represents the group average (N=12) of the muscle amplitude for a period of 10ms; error bars represent the standard deviation. Asterisks (*) represent UD 2-9 bins that have a statistically significant difference from zero (p< 0.05), rounds (°) represent the UD 1 bins that have a statistically significant difference from zero, N= number of subjects.

Figure 5: Unexpected down (UD) versus expected down (ED). The upper panel shows the average muscle amplitudes for the UD and ED trials of subject GE (N=1). The other panels show the responses of the UD 2-9 trials when the average of the ED trials were subtracted out (thus UD-ED). The data of both conditions were aligned at the moment of actual heel contact (AHC). Each bin represents the group average (N=9) of the amplitude for a period of 10ms; error bars represent the
standard deviation. A positive deflection implies enhanced UD muscle activity; a negative deflection implies a decreased UD activity as a consequence of muscle silencing. Bin amplitudes that have a statistically significant difference from zero (p< 0.05) are marked by the grey bars on the x-axis. MG = medial gastocnemius, TA = tibialis anterior, RF = rectus femoris, BF = biceps femoris, i = ipsilateral, c = contralateral, N= number of subjects.

Figure 6: Joint angle curves of the ankle (AN) and knee (KN). Left column; unexpected down (UD) versus expected down (ED) (N=9). Right column; unexpected level (UL) versus expected level (EL) (N=12). The panels show the mean ± the standard error of the EL and ED condition (grey line). Superimposed is the average of the UL and UD condition (black line). i=ipsilateral, c=contralateral. For each subject, the UL condition was based on the average of the UL ≥ 7 trials. The UD condition was based on the average of the UD 2-9 trials.

Figure 7: Single subject characteristics of unexpected level walking (UL1). Illustrated in the left column are the vGRF, joint angle curves and muscle activation patterns of subject CA. The data were aligned at actual heel contact (AHC), indicated by the solid vertical line. The period that was defined as the start and end of the unloading phase is indicated by the shaded vertical area. The grey curve illustrates the average of the control trials of level walking (EL), the black curve illustrates the average of the first trials of level walking after an unexpected step down (UL1). The curves represent the average ± two times the standard deviation of 9 UL1 trials and 10 EL trials. i = ipsilateral, c= contralateral, AK = ankle, KN = knee, MG = medial gastocnemius, TA = tibialis anterior, RF = rectus femoris, BF = biceps femoris, TO = toe-off.
Figure 8: Population characteristics of unexpected level walking (UL 1). Population averages of the subtracted kinematic and EMG data are shown. The data were aligned at actual heel contact (AHC), indicated by the solid vertical line. The average of the EL trials was subtracted from the UL 1 trials. The amplitude of the subtracted signal was averaged across bins of 10 ms. Afterwards it was averaged for all subjects (N=12). Error bars represent standard deviation. The arrows represent the onset latency (L), which indicates the average moment of significant burst onset. i = ipsilateral, c= contralateral, AK = ankle, KN = knee, MG = medial gastrocnemius, TA = tibialis anterior, RF = rectus femoris, BF = biceps femoris, L=onset latency, N= number of subjects, TO = toe-off.

Figure 9: Schematic overview of the responses to an unexpected lowered surface (A) and an unexpected level surface (C). Results are compared with the rapid stopping reaction (B), as adapted from Hase& Stein (1998; figure 4,6 and 8), and tripping (D), as adapted from Eng et al. (1994; figure 4 and 5). i = ipsilateral, c = contralateral, MG = medial gastocnemius, TA = tibialis anterior, RF = rectus femoris, BF = biceps femoris, SOL = soleus, VL = vastus lateralis. Arrows indicate the direction of joint movement.
### Figure 1

#### Definitions

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#### Protocol

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Figure 2
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Figure 9

A  Unexpected lowered surface

B  Rapid stopping

C  Unexpected level surface

D  Tripping

Figure 9