On the reflex coactivation of ankle flexor and extensor muscles induced by a sudden drop of support surface during walking in humans

Kimitaka Nakazawa, Noritaka Kawashima, Masami Akai, and Hideo Yano
Motor Dysfunction Department, National Rehabilitation Center for the Disabled, Tokorozawa 359-8555, Japan

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Nakazawa, Kimitaka, Noritaka Kawashima, Masami Akai, and Hideo Yano. On the reflex coactivation of ankle flexor and extensor muscles induced by a sudden drop of support surface during walking in humans. J Appl Physiol 96: 604–611, 2004. First published October 3, 2003; 10.1152/japplphysiol.00670.2003.—Recent studies have revealed that the stretch reflex responses of both ankle flexor and extensor muscles are coaugmented in the early stance phase of human walking, suggesting that these coaugmented reflex responses contribute to secure foot stabilization around the heel strike. To test whether the reflex responses mediated by the stretch reflex pathway are actually induced in both the ankle flexor and extensor muscles when the supportive surface is suddenly destabilized, we investigated the electromyographic (EMG) responses induced after a sudden drop of the supportive surface at the early stance phase of human walking. While subjects walked on a walkway, the specially designed movable supportive surface was unexpectedly dropped 10 mm during the early stance phase. The results showed that short-latency reflex EMG responses after the impact of the drop (<50 ms) were consistently observed in both the ankle flexor and extensor muscles in the perturbed leg. Of particular interest was that a distinct response appeared in the tibialis anterior muscle, although this muscle showed little background EMG activity during the stance phase. These results indicated that the reflex activities in the ankle muscles certainly acted when the supportive surface was unexpectedly destabilized just after the heel strike during walking. These reflex responses were most probably mediated by the facilitated stretch reflex pathways of the ankle muscles at the early stance phase and were suggested to be relevant to secure stabilization around the ankle joint during human walking.

METHODS

Subjects. The subjects were 12 adults (24.6 ± 5.7 yr, 172.1 ± 6.7 cm, 61.0 ± 6.4 kg) who had no history of neurological disorder. The subjects gave their informed consent to the experimental procedures, which were conducted in accord with the Declaration of Helsinki of 1975 and approved by the ethics committee of the National Rehabilitation Center for the Disabled, Tokorozawa, Japan.

Experimental device. A specially designed movable platform (40 cm wide, 60 cm long, and 15 cm high) was used to drop the support surface (Fig. 1). This platform consisted of four electromagnets, which enabled a short drop of the support surface (height, 1 cm; duration, 100 ms) triggered by a computer signal. A 5-m-long walkway was built, and the platform was incorporated into the middle of the walkway. Furthermore, the platform was placed on a force plate (40 cm wide, 250 cm long; Kistler, Winterthur, Switzerland) so that ground reaction forces could be measured and used to trigger the drop of the support surface.

Experimental protocols. To measure muscular reactions to the drop of the supportive surface, perturbations were induced while the subjects were walking on the walkway.

During the experiment, the subjects walked on the walkway at a comfortable speed and cadence. Several practice runs were provided to enable the subjects to repeat the trial with a similar walking speed.
and cadence and to step onto the platform with their right foot. A
metronome was used to indicate the subject’s preferred cadence and to keep
the cadence constant. Stride time from the right foot touch down
onto the platform to the next right foot touch down was measured for
each trial with a stopwatch. Because the responses in EMGs are
evaluated as the differences from the EMG activity of the unperturbed
trials, we tried to keep the walking speed and cadence as similar as possible. As variation in the walking increases, the risk of both over-
or underestimation simultaneously increase.

Perturbed and unperturbed trials were mixed in a randomized order
to prevent the subjects from predicting the perturbation. In total, 5
perturbations were applied out of 45 trials for 8 subjects, and, for the
other 4 subjects, 12 perturbations were applied out of 70 trials. The
perturbation was applied in such a way that the support surface was
dropped when the vertical component of ground reaction forces
(VGRF) reached a predetermined trigger level during the initial
increasing phase of the VGRF. The intrasubject trigger level was the same (60% of body weight) throughout the experiment. As a reaction
to the perturbation, some subjects sometimes lifted both arms, a
motion that is called “parachute response.” Qualitatively, this re-
sponse disappeared as the subject became habituated to the perturbation.
Regardless of whether or not this response occurs, we accepted all
perturbed trial data.

Data recording. For all subjects, EMG activities from the right
lower leg muscles, the Sol, the lateral gastrocnemius, the medial
gastrocnemius (MG), and the TA were recorded. In 8 of the 12
subjects, the EMG activities of those 4 muscles in the left leg were
also recorded. Furthermore, the EMG activities from the rectus fem-
oris, the trapezius, the lateral deltoid, and the erector spinae (ES) were
recorded in both sides of the legs in four subjects.

Two surface electrodes (Ag-AgCl, 0.8-cm diameter) were applied
along the muscle fibers over the belly of each muscle with an
interelectrode distance of 0.5 cm. The EMG signal was detected by
using a bipolar differential amplifier with an upper and lower cutoff
frequency of 20–500 Hz.

Special care was taken to eliminate any artifacts in the EMG
recording. To ensure this, the electrodes were attached by using
two-sided adhesive tape after careful preparation of the skin. Very thin
elastic nylon bandages were then used to firmly hold both the
electrodes and the lead lines to the body; in this way, any artifacts due
to a small displacement of the electrodes and lines were prevented.
We further confirmed whether any artifacts were contaminating the
EMG signals by hitting the floor with the subject’s heel several times.

Right knee and ankle joint angles were measured by using electro-
goniometers (Biometrics, Newport, Gwent, UK). While the subjects
were walking on the walkway, the VGRF was measured by the force
plate. All electric signals were digitized with a sampling frequency of
1 kHz by an analog-to-digital converter and stored in the computer
memory for later analysis.

Data were recorded within the time interval from 500 ms before to
1,000 ms after the trigger signal to drop the supportive surface. The
trigger signal was generated when the VGRF on the movable platform
reached the predetermined force level after heel contact. For the
control trials (unperturbed trial), the same intervals were recorded, but
the supportive surface did not drop after the trigger.

EMG analysis. Figure 2 shows the process of EMG analysis used in
the present study. From the rectified EMG signals, mean values were
estimated for each 10-ms time window (Fig. 2A). The corres-
ponding EMG activity levels during the unperturbed trials were then
subtracted from the EMG activities of the perturbed trials (Fig. 2B).
To quantify the responses, the peak response amplitude, response
duration, and integrated EMG activity were calculated for each
perturbed trial. Both the beginning and end of the response were
defined as the times when EMG activities reached levels higher and
lower, respectively, than twice the standard errors of the background
activity. The latency of response was defined as the duration from
the impact of the drop to the EMG onset. The integrated EMG was
calculated between the beginning and end of the response.

Statistics. The EMG response magnitudes among the various muscles
were statistically compared by using a one-way ANOVA. When
statistical significance was encountered, Tukey’s post hoc test for
multiple comparisons was performed to determine which muscle
response was significantly different from the other by using $P < 0.05$

as the criterion of statistical significance. Data are presented as
means ± SE.

RESULTS

Changes in the locomotor pattern due to the perturbation. In
the present study, a new type of perturbation was applied during
the early stance phase of walking. As demonstrated in Fig. 3, the perturbations were applied during the initial rising
phase of the vertical ground reaction force, which corre-
sponded to the early stance phase of the walking cycle. In Fig.
4, the duration of the drop of the supportive surface was clearly
demonstrated as a transient decrease of the VGRF. Generally,
this perturbation did not induce a large instability that affected
the entire body during walking. This was likely due to the fact
that the drop was only 1 cm in height. The typical reaction to
the perturbation during walking was characterized by a pro-
longation of the stride time (1,124.0 ± 26 vs. 1,164.1 ± 21 ms,
unperturbed vs. perturbed walking) without any large angular
changes in the lower limb joints.

EMG responses to the perturbation during walking. A typi-
cal example of EMG recordings from the lower leg muscles
during perturbed walking is demonstrated in Fig. 3 together
with the ensemble-averaged waves of the unperturbed walking.
The short latency, which is probably a reflex EMG response,
was consistently observed in the muscles of the perturbed side
(right stance leg). The latencies of the responses recorded in
those muscles are shown in Table 1. The onset of the EMG
responses in the ankle muscles after the impact that occurs as
a result of dropping the platform occurred within 100 ms,
which corresponded to ~140–160 ms after the onset of the
drop. These EMG responses lasted 60–100 ms from the onset of
the EMG burst, implying that a long-latency polysynaptic
reflex pathway was involved in those responses. In the TA, espe-
sially, the duration of the response tended to be longer
than in the other muscles, although the differences were not
statistically significant. The observed responses in the EMGs
were probably not induced by muscle stretches that occurred
when the platform was dropped, because there were no notice-
able changes in angular displacement to either the knee or

Fig. 1. Experimental setup. The support surface of the movable platform in the
middle of the walkway drops 10 mm in the early stance phase during walking.

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Fig. 2. A: example of the raw electromyograph (EMG) signal from the medial gastrocnemius muscle (left) and the integrated EMG (iEMG) for each 10-ms period (right). B: process to quantify the EMG response. Unperturbed walk EMG is subtracted from the perturbed walk EMG to obtain the net EMG responses. Peak EMG, iEMG, and response duration were all derived from the averages of the subtracted EMG. p, Perturbed trial.

Fig. 3. Typical example of the EMG responses in the 4 ankle muscles [soleus (Sol), lateral gastrocnemius (LG), medial gastrocnemius (MG), and tibialis anterior (TA)] in the right and left legs and the vertical component of ground reaction force (VGRF) recorded in 1 subject. Oblique lines indicate that the heel contact and all EMG and VGRF traces are aligned at this moment. UPT, unperturbed trial; PT, perturbed trial.
ankle joints during the dropping phase (Fig. 4). Rather, the impact that occurred as a result of dropping the platform seemed to trigger the response. This will be discussed in more detail in the DISCUSSION.

Figure 5 demonstrates a typical example of the quantified muscle responses to the perturbation as defined by the subtracted EMGs. It was clearly shown that the reflex EMG response consistently appeared in both the ankle extensor and flexor muscles in the perturbed leg during walking, whereas none of the muscles except the TA showed such a response in the contralateral (left) leg. As demonstrated in the MG and TA waveforms in this figure, the reflex EMG responses in the ankle extensor muscles were followed by negative EMG values. This was due to a short period of suppression after the induced EMG response. After the suppression period, greater EMG activities appeared again (not shown in the figure), which corresponded to the normal EMG activities seen in these muscles during the stance phase but with greater amplitude after the perturbation. In the TA muscle, which is normally silent during the midstance phase, the EMG response also appeared. Thus the reflex EMG responses were simultaneously induced in both the ankle flexor and extensor muscles in the perturbed leg.

The magnitude of EMG responses induced in each muscle by the perturbation is summarized in Fig. 6. In the figure, the peak EMGs are expressed in both absolute value and relative value to the background EMG magnitude for 100 ms before the impact that occurs as a result of dropping the platform. A markedly greater response was induced in MG in the perturbed leg, and a greater response, although slightly smaller than that in the MG, was also induced in the TA. Interestingly, a comparably sized reflex EMG response also appeared in the contralateral TA that was in the swing phase, whereas no reflex EMG response was observed in the extensor muscle group.

Thus it was clearly demonstrated that a significant amount of reflex EMG response was induced in the TA during the early stance phase (the perturbed leg) and the swing phase (the unperturbed leg) of walking. By contrast, in the ankle extensor muscles, the reflex EMG responses were induced only during

Table 1. Group EMG response latency in each ankle muscle recorded after the impact that took place when the movable platform was dropped during walking

<table>
<thead>
<tr>
<th></th>
<th>Sol</th>
<th>LG</th>
<th>MG</th>
<th>TA</th>
</tr>
</thead>
<tbody>
<tr>
<td>12</td>
<td>41.67±1.51</td>
<td>41.18±2.07</td>
<td>41.27±1.88</td>
<td>50.70±3.07</td>
</tr>
</tbody>
</table>

Values are means ± SE given in ms; n, no. of subjects. EMG, electromyograph; Sol, soleus; LG, lateral gastrocnemius; MG, medial gastrocnemius; TA, tibialis anterior.
the early stance phase. No responses were observed in these muscles during the swing phase.

Adaptation of the EMG responses to the repetition of the perturbation. The observed short-latency EMG responses showed a general tendency to be smaller in size when the number of perturbations applied was increased. Figure 7 demonstrates that variations of the EMG responses are a function of successive applications of perturbation. In the figure, normalized values to each subject’s first responses are shown, and a total of 140 plots are included in each panel. The left-side Sol, lateral gastrocnemius, and MG were not analyzed, because no responses occurred in those muscles. This tendency toward habituation was observed in all of the lower leg muscles in which short-latency responses were induced. Statistically significant (P < 0.05) coefficients of correlation were found in the relation of the stimulus number and the normalized peak-response values in those muscles with the exception of the right-side TA.

**Fig. 5.** Typical example of the subtracted EMGs (single perturbed trial minus averaged control, see Fig. 2) obtained during walking. The numbers indicate the order of perturbed trials.

**Fig. 6.** Comparisons of the peak EMG [absolute (abs) and relative values (%BGA = percentage of background EMG level)] and response duration among 4 muscles in each leg. Values are means ± SE. Note that the results of “stance leg” are the group means ± SE of 12 subjects, and those of “swing leg” are of 8 subjects. *P < 0.05.
Fig. 7. Reduction of the short-latency EMG responses as a function of stimulus repetition. Data obtained from all subjects (n = 12) are plotted together and normalized with the first-response magnitude in each subject. From the left leg, only TA data (lTA) are shown because very few responses were observed in the ankle extensors. rTA, right TA. *P < 0.05.

EMG responses in the other muscles. As mentioned in METHODS, the EMG activities of the quadriceps and upper arm, neck, and trunk muscles were recorded in 4 of the 12 subjects. A sample recording is shown in Fig. 8, and the response latencies and the peak EMG response values are summarized in Table 2. In the perturbed-side rectus femoris, a short-latency response with similar latencies to those in the ankle muscles was observed (40.67 ± 8.33 ms), although the response was generally small and variable. In the ES, trapezius, and lateral deltoid muscles, reflex-like EMG responses were observed, mostly in the unperturbed (left) side. However, in some subjects, the responses were seen in the ES on both sides. Qualitatively, the responses in these muscles were much more variable than those in the lower leg muscles. In the present study, we did not perform further analysis for these muscles because our focus was on the responses of the lower leg muscles.

DISCUSSION

This study was, as far as we know, the first attempt to investigate lower leg muscle reactions when the body is destabilized as a result of dropping a supportive surface during human walking. In cats, however, Hiebert et al. (11, 12) and Gorassini et al. (9) have reported the first clear observations for kinematic and muscle responses of cats when ground support is unexpectedly lost just before foot contact during walking. They demonstrated that, when there is a loss of ground support, the limb is rapidly lifted and replaced in an attempt to seek support. In the present study, we did not observe similar corrective reactions. This may have been because the supportive surface in our experiment was only dropped a short distance. It would be interesting to see to what extent the lower limb activations would be modulated by the loss of a supportive surface as humans walk and to compare the findings with those observed in cats; however, we did not design the present study for such a purpose. Future studies and equipment improvements could make it possible to compare the corrective responses of humans and cats to the loss of a supportive surface during walking.

On the neural pathway mediating the reflex EMG responses. The results of the present study demonstrate that, during walking, a short drop of the supportive surface induces reflex EMG responses simultaneously in both the antagonistic ankle flexor and extensor muscles. It was obvious that the reflex EMG responses were not induced by muscle stretches that occurred during the dropping phase and at the end of the drop, because no significant joint angle changes occurred between the onset of the drop and the onset of the EMG response, compared with the results of the unperturbed trials. In addition, the observed simultaneous occurrence of reflex EMG responses in both the ankle extensor and flexor muscles strongly suggests that the response was not simply induced by muscle stretches. The question then arises as to the neural source of the reflex EMG responses in the present study. The latencies of reflex EMG responses resulting from the impact of the drop (collision of the supportive surface with the ground) were in quite a similar range to that of stretch reflex responses during treadmill walking [42 ± 3.2 ms (Ref. 22) vs. 41.7 ± 1.5 ms (present study)]. This suggests that the reflex EMG response observed in our study mediated the spinal stretch reflex pathway, although the muscle stretch was probably not the neural source, as mentioned above. Recently, Schillings et al. (19, 20) demonstrated that mechanical stimuli other than muscle stretches could induce a short-latency response mediated by the spinal stretch reflex pathway. They demonstrated that a short-latency response, almost identical to the response latency in our trials, could be induced by the collision of the foot with an obstacle during the swing phase in treadmill walking. They suggested that this reflex EMG response was induced by small muscle vibrations due to the collision of the foot with an obstacle; that is, a sudden jar caused by a collision of the foot with an obstacle might excite Ia afferents in the muscles throughout the ipsilateral limb. The same neural mechanism could also be applied to explain our results. The impact of the drop causes an abrupt vibration of bones, tendons, and musculature in the ipsilateral limb. The vibration then induces an Ia afferent volley that elicits a short-latency response.

Another possible mechanism to explain the observed response might be the startle response, which arises from the brain stem. The observed reduction of responses with the repetition of perturbation (Fig. 7) supports this idea. However, the startle response could not alone explain the short-latency
EMG responses. First, the latencies of the EMG responses in the neck or trunk muscles were not necessarily shorter than those in the distal ankle muscles (see Tables 1 and 2), although the latency would increase as a function of distance from the brain stem if those were startle responses. Second, the latencies of the auditory evoked startle responses in TA and Sol during human walking (18) are longer \[57 \pm 2 \text{ ms, respectively}\] than those observed in the present study \[50.7 \pm 3.1 \text{ ms in the TA and 41.7 \pm 1.5 \text{ ms in the Sol}}\). However, we still cannot ignore the possibility that the startle response may constitute a part of the short-latency responses. Furthermore, there is also a possibility that a part of the response in the extensor muscles is due to a vestibulospinal discharge induced by the downward acceleration \((10, 23)\), although this mechanism cannot alone account for all responses, especially those in the flexor muscles. Further studies will be necessary before this issue can be fully clarified.

**Phase-dependent changes of the reflex EMG amplitude.** In the walking trials, strong reflex EMG responses were elicited in the triceps surae muscles of the perturbed leg (in the stance phase), whereas no reflex EMG responses were elicited in the contralateral leg (in the swing phase). It is well known that the H reflex elicited in the Sol muscle is strongly suppressed during the early swing phase \((3, 14)\). The results of our study are in agreement with those of previous observations. Augmented strength of the reciprocal inhibition, specifically during the swing phase, was demonstrated to suppress the spinal motoneuronal excitability of the ankle extensor muscles \((14)\). In contrast, the reflex EMG response, which is usually active in the swing phase, was elicited even in the contralateral TA in the present study. Because there was no difference in the latencies of the responses in either side of the TAs, it was likely that vibration of the ipsilateral limb induced the response in the contralateral TA as well.

**Intermuscular differences in the reflex EMG amplitude.** As mentioned in RESULTS, large intermuscular differences were observed in the reflex EMG response amplitude. Among the three ankle extensor muscles, the reflex EMG response of the MG was specifically increased during walking, suggesting that the excitability of this pathway was selectively facilitated in the ankle extensor muscles, at least during the early part of the stance phase. It has been reported by Duysens et al. \((7)\) that the cutaneous reflex response during the stance phase of walking is selectively augmented in the MG. They suggested that the enhanced response was related to the particular functional role of the MG during this phase; that is, an exorotation moment during the stance phase would be generated, mainly by the MG \((7)\). A similar dissociated activation in the triceps surae muscle has been observed in several motor tasks, such as in a voluntary lengthening contraction \((16, 17)\), swimming in rats \((13)\), and water walking \((15)\). The present study revealed that the stretch reflex response during the stance phase was selectively enhanced in the MG, although the underlying neural mechanism is unknown. Functionally, however, the coactivation of the MG and TA might be the first defensive response to abrupt and unexpected changes in a supportive surface.

**Table 2. Latency and peak amplitude of the short-latency responses recorded in the thigh and trunk muscles**

<table>
<thead>
<tr>
<th></th>
<th>RF</th>
<th>Tr</th>
<th>Del</th>
<th>ES</th>
<th>LRF</th>
<th>LTr</th>
<th>LDel</th>
<th>LES</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Latency, ms</strong></td>
<td>4.067±0.81</td>
<td>25.67±0.37</td>
<td>53.33±11.47</td>
<td>70.5±8.71</td>
<td>28.5±13.9</td>
<td>27.67±4.97</td>
<td>37.67±7.17</td>
<td>57.0±7.20</td>
</tr>
<tr>
<td><strong>Peak, mV</strong></td>
<td>0.095±0.033</td>
<td>0.159±0.117</td>
<td>0.374±0.141</td>
<td>0.321±0.081</td>
<td>0.060±0.019</td>
<td>0.423±0.196</td>
<td>0.817±0.387</td>
<td>0.215±0.079</td>
</tr>
</tbody>
</table>

Values are means ± SE; n, no. of subjects. RF, rectus femoris; Tr, trapezius; Del, lateral deltoid; ES, erector spina; LRF, left RF; LTr, left Tr; LDel, left Del; LES, left ES.
In the present study, small EMG responses were observed in the contralateral ankle extensor muscles and greater responses in both sides of the TA. Generally, there is little EMG activity in the TA during the stance phase of walking. It is noteworthy that a markedly large reflex EMG response was evoked in the TA by a drop in the support surface during the early stance in this study. These results were consistent with the following observations. Capaday et al. (2) showed that a significantly large motor-evoked potential was induced in the inactive TA by transcranial magnetic stimulation during the stance phase in large motor-evoked potential was induced in the inactive TA.

Functional implications. To date, no study has shown that the disturbance of the support surface during the stance phase induced reflex responses in both the ankle extensor and flexor muscles, although several H-reflex or stretch reflex studies have demonstrated separately that the reflex responses in those muscles are increased during this phase. Generally, coactivation of agonist and antagonist muscles is thought to increase joint stiffness and, hence, joint stability (8). With an effective series of experiments, Christensen et al. (6) suggested that stretch reflexes are primarily involved in securing the stability of the supporting limb in the stance phase. They further emphasized that “the most critical period is soon after the heel strike when the subject is about to move the weight of the body from one leg to the other, because, at this time, it is still unknown how stable the ground is and whether it can support the weight of the subject.” We actually applied a specific perturbation to disturb the stabilization of the subject’s body during the stance phase and showed that reflex responses were most certainly elicited in both the ankle flexor and extensor muscles. Thus our results strongly support the hypothesis that the stretch reflex excitability in the ankle muscles is regulated to stabilize the ankle joint during the early stance phase.

In summary, our results showed that a sudden drop of a supportive surface during walking induces a reflex response in both the ankle extensor and flexor muscles. This is the first observation indicating that spinal or supraspinal stretch reflex pathways are coaugmented in both the ankle extensor and flexor muscles during the early stance phase of human walking. The coaugmented responsibility of the antagonistic ankle muscles would play a role in securing stabilization around the ankle joint.

GRANTS

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