Reflexes From the Superficial Peroneal Nerve During Walking in Stroke Subjects

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Zehr, E. P., K. Fujita, and R. B. Stein. Reflexes from the superficial peroneal nerve during walking in stroke subjects. *J. Neurophysiol.* 79: 848–858, 1998. The function of ipsilateral cutaneous reflexes was studied with short trains of stimuli presented pseudorandomly to the superficial peroneal nerve (SP; innervates the top of the foot) during treadmill walking in neurologically intact (NI) subjects and subjects who had had a stroke. Ankle and knee joint angles together with electromyograms (EMG) of tibialis anterior (TA), soleus (SOL), medial gastrocnemius (MG), vastus lateralis (VL), and biceps femoris (BF) muscles were recorded. Net reflex EMG and kinematic responses to stimulation were quantified in each of the 16 parts of the step cycle and responses compared between the stroke and NI subjects. Stimulation strongly suppressed extensor muscles throughout stance in the stroke subjects. TA muscle showed a significant suppression during swing phase that was correlated with reduced ankle dorsiflexion in both stroke and NI subjects. BF reflexes were facilitatory during parts of swing and VL reflexes were suppressive throughout stance in the stroke subjects. There was a significant correlation between BF facilitation and knee flexion during swing, which was stronger in NI subjects. We conclude that only part of the stumble correction to foot dorsum electrical stimulation observed in NI subjects is maintained after stroke, and that new, suppressive responses are seen.

INTRODUCTION

Investigations of reflexes after spinal cord injury or CNS insult often reveal hyperactive and generally excitatory responses (Stein et al. 1993). Reflexes arising from muscle afferents have been studied in spinal cord injured subjects during slow walking. Yang et al. (1991) showed that the phasic soleus (SOL) H-reflex modulation observed in neurologically intact subjects was impaired in spastic paretic subjects and that the reflex was greatly enhanced. These responses seemed to be related to the degree of motor impairment in a given patient. Likewise, Sinkjaer et al. (1995) found the SOL H-reflex to be modulated only by the level of background muscle activation and not by phasic locomotor activity in spastic multiple sclerosis subjects, indicating a deficient central modulatory mechanism. Sinkjaer et al. (1996) recently showed that modulation of stretch reflexes and ankle joint torque is also deficient in these subjects. They demonstrated that the reflex impairment, as well as alterations in intrinsic stiffness of the ankle joint, contributes to the locomotor deficit. El-Abd and Ibrahim (1994) studied cerebral somatosensory-evoked potentials (SEPs) elicited by stimulation of the motor nerve to SOL during gait in stroke subjects with spastic hemiplegia. They documented a deficiency in modulation of the SEPs on the affected side of most of their subjects.

Cutaneous reflexes are also affected by CNS insult. Jones and Yang (1994) studied cutaneous reflexes arising from stimulation of the tibial nerve in spinal cord injured patients and found an altered pattern of reflex modulation to that seen in uninjured subjects (Yang and Stein 1990; Zehr et al. 1997b). The patients showed very little inhibitory modulation and instead excitation predominated through. t the step cycle in tibialis anterior (TA) muscle. In SOL, there were some inhibitory responses during stance, but facilitation predominated during swing phase. Interestingly, Fung and Barbeau (1994) stimulated the medial plantar nerve (a mixed nerve and distal branch of the tibial nerve) in spastic paretic spinal cord injured subjects during walking. They observed that this cutaneomuscular stimulation could modulate the excitability of SOL H-reflex to a level that co d be similar to neurologically intact (NI) subjects (see Stein et al. 1993). To the best of our knowledge, cutaneous reflexes from other nerves have not been studied during gait in other patient groups.

The function of cutaneous reflexes during walking in humans has been uncertain. Recently, we showed that reflexes arising from the superficial peroneal nerve (SP), which innervates the dorsal surface of the foot, and the tibial nerve, which innervates the plantar foot surface, elicit functional electromyograph (EMG) and kinematic responses during treadmill walking (Zehr et al. 1997b). Electrical stimulation of the SP nerve during early swing was shown previously to give rise to a stumble correction in which TA muscle was suppressed and biceps femoris (BF) muscle facilitated (Van Wezel et al. 1997; Zehr et al. 1997b). These neural responses gave rise to kinematic changes in which ankle dorsiflexion and knee extension were reduced, such as would be predi ed in a stumble avoidance of an encountered object (Zehr et al. 1997b). These responses to electrical stimulation seemed similar to the stumble correction observed in cats after both electrical and mechanical stimulation (Forssberg 1979) and after perturbing humans during treadmill walking (Schillings et al. 1996). However the extent to which this stumbling corrective response may be maintained in pathological gait is not known.

The purpose of the present study was to evaluate the reflex responses to SP nerve stimulation in subjects who had had a stroke. The function of observed reflexes in the EMG were tested by correlation with kinematic changes in knee or ankle joints, conducted previously in neurologically intact subjects (Zehr et al. 1997b). Portions of these results were presented briefly in abstract form (Zehr et al. 1997a).

METHODS

Subjects and experimental protocol

Eleven NI subjects (9 males and 2 females), aged 26–56 yr, and eight stroke subjects (7 males and 1 female), aged 49–78 yr, participated in the experiments with informed, written consent. The average age was 62 (median = 59.5) in the stroke and 37.7 (median = 33) in the NI group. All patients had some extent of foot drop and many had some minor level of spasticity (~1 on the modified Ashworth scale). Two of the stroke subjects occasionally demonstrated periods of clonic activity in the lower limb musculature and five stroke subjects regularly used a cane for overground walking. The stroke subjects were 3–48 mo postinjury and were studied on the more affected side. All experiments were conducted under an approved protocol for human subjects at the University of Alberta. During each session, subjects walked on the treadmill at self-selected and comfortable speeds for periods of ~7 to 10 min at each speed. For the stroke subjects, the walking speeds were in the range of 0.71–1.6 km/h (median = 1.2 km/h) and for the NI subjects 1.5–3.5 km/h (median = 2 km/h). The majority of the stroke subjects held the treadmill handrail for support throughout a walking trial. Approximately 400–600 steps were collected for each speed (including stimulated and control unstimulated steps). A more detailed description of the experimental protocol and data analysis was published elsewhere (Zehr et al. 1997b).

Nerve stimulation

The SP nerve was stimulated by using either a GRASS SD9 (Grass Inst., Quincy, MA) isolated constant voltage stimulator or a custom-built constant current stimulator with trains of five pulses at 200 Hz, with a pulse width of 1.0 ms. No differences in responses were observed with either type of stimulation. The electrodes for the SP nerve were placed on the front of the leg just near the ankle joint, such that stimulation produced a strong radiating parasthesia over the foot dorsum. Flexible 1-cm disposable Ag-AgCL surface EMG electrodes (Electrotrace, Jason, Huntington Beach, CA) were used for cathodal stimulation. The threshold of stimulation in terms of the perceptual threshold (PT), defined as the lowest stimulation that was just detectable by the subject, and the radiating threshold (RT), defined as a clear radiating parasthesia, were determined in all subjects. Stimulation intensities were typically twice RT. The stimulator was driven by a pseudorandom pulse generator with a minimal repeat time equal to the step cycle time and a maximum that was approximately twice the step cycle time for each subject; thus a stimulus arrived approximately once every 3 steps. Accordingly, we collected many unstimulated steps and no step had more than 1 stimulus. Outputs from both the trigger pulse generator and the stimulator were sent to a 12 bit A/D converter and then into a 486 66-MHz microcomputer running Axotape (Axon Instruments) data acquisition software.

Electromyography

The skin was lightly abraded and cleansed with alcohol and disposable Electrotrace Ag-AgCL surface EMG electrodes were applied in bipolar configuration longitudinal to the predicted path of the muscle fibers (2 cm interelectrode distance) over the SOL, medial gastrocnemius (MG), TA, vastus lateralis (VL), and BF muscles. SOL electrodes were placed distal to the termination of the gastrocnemius muscles, whereas MG electrodes were placed over the medial head of the gastrocnemius. TA electrodes were placed over the largest girth of the tibialis anterior muscle. For VL, the distal electrode was placed ~4–6 cm proximal to the lateral margin of the patella and for BF placement was over the muscle belly at approximately one-third of the distance from the knee to the hip. The position of the stimulating electrodes was such that it typically produced some minor facilitation of the nearby extensor digitorum brevis muscle. We therefore monitored the peak-to-peak EMG of the M-wave from this muscle to check that its stimulation remained relatively constant throughout the cycle. Variation in stimulation across the step cycle was generally <10%. Ground electrodes for the EMG were placed over electrically neutral tissue, such as the knee, and EMG signals were preamplified and highpass filtered at 100 Hz. Then they were full-wave rectified, thus yielding components down to DC and low-pass filtered at 100 Hz. This filtering process provided a good linear envelope with little spread of the stimulus artifact (which could then be digitally removed off-line, see below) and is one that we have previously employed (Zehr et al. 1997b). The processed output was sent to a 12 bit A/D converter and then into a microcomputer sampling at 500 Hz.

Kinematics and step-cycle timing

Angular position of knee and ankle were recorded with custom-made potentiometric electrogoniometers placed over the joint and secured with plastic tape and fabric straps. Signals obtained from custom-made force sensors located in the insole of the subject's shoe were used to establish step cycle parameters (e.g., heel contact, toe-off). The electrogoniometers and force sensors could resolve angular changes >1° and forces >5 N, respectively. Angle and force signals were preamplified (the subject wore a small pouch and belt to hold the amplifiers) and then sent directly to the AXOTAPE computer system.

Data acquisition and analysis

The data were sampled continuously and stored on hard disk for off-line analysis. Custom written software programs were used to separate the step cycle into 16 separate parts, beginning with heel contact. The stimuli occurred randomly throughout the step cycle. All responses to stimuli occurring in the same part of the step cycle were averaged (n = ~10–20 in each part) together and aligned to stimulus delivery within that part of the step cycle. The values obtained for each of the 16 averages without stimulation were subtracted from the corresponding averages from stimulated steps during the same trial.

Background step cycle EMG profiles

To compare phasic levels and timing of muscle activation during walking between the NI and stroke subjects, unstimulated, control EMG profiles were obtained throughout the entire step cycle and a mean value was obtained for each part of the step cycle for each subject. These values were normalized to the maximum activation level occurring in the step cycle, thus yielding an EMG activation profile ranging from a minimum activation to a maximum of one for each subject. These values were then averaged across each part of the step cycle for each group of subjects. Significant differences between groups in these values would indicate a different muscle activation profile during gait that is independent of absolute activation level (which may vary widely across subjects and between groups).

Net reflex EMG analysis

Stimulus artifacts were digitally removed and then the EMGs were filtered with a 5-point digital moving average filter. The unstimulated control EMGs for each part of the step cycle were subtracted from the corresponding stimulated step cycle parts to yield subtracted evoked EMG traces. The evoked EMGs for each subject were analyzed for the net reflex effect by using the Average Cumulative Reflex

EMG after 150 ms ($ACRE_{150}$) (Zehr et al. 1995). This technique was employed because it provides a useful quantification of the net EMG reflex effect and provides an index for correlation to kinematic changes (Zehr et al. 1997b). Briefly, the analysis program calculated the subtracted, residual reflex EMG traces and digitally removed the stimulus artifact. The poststimulus data were then sequentially summed and any significant facilitation or suppression was identified as positive or negative deflections in the EMG record. The value obtained at 150 ms after stimulation was then divided by the time interval of integration to measure an overall reflex effect (i.e., negative values indicate overall suppression and positive values overall facilitation). As we were interested in reflex effects, a 150 ms poststimulus interval was chosen because it preceded any significant voluntary activation in lower leg muscles (see Zehr et al. 1995). The $ACRE_{150}$ values for each subject were normalized to the peak EMG value (averaged over a 40-ms time window) occurring during the entire step cycle for each muscle and expressed as percentages.

Kinematic analysis

Subtracted values for angular changes were used for analysis. The maximum change observed in these smoothed (n = 5 point filtering) signals over an interval ranging from 140 to 220 ms poststimulation was calculated. We have used this sliding latency previously (Zehr et al. 1997b), which was chosen to reflect the delays between an EMG response and the peak mechanical change in moving muscles. As described for the EMG, these values were then normalized to the maximum range of motion occurring within the step cycle and expressed as percentages.

Statistics

In all instances, analysis was conducted on averaged values for each subject from each part of the step cycle. Significant differences from zero for the net reflex effects were determined by calculation of t-ratios for each part of the step cycle and differences between groups of subjects were determined with t-tests. Linear least-means square regression analysis was used to evaluate correlation between EMG indices and changes in ankle and knee joint angle at each part of the step cycle. Descriptive statistics included means ± SE and statistical significance was set at $P \leq 0.05$. When available, datasets from different walking

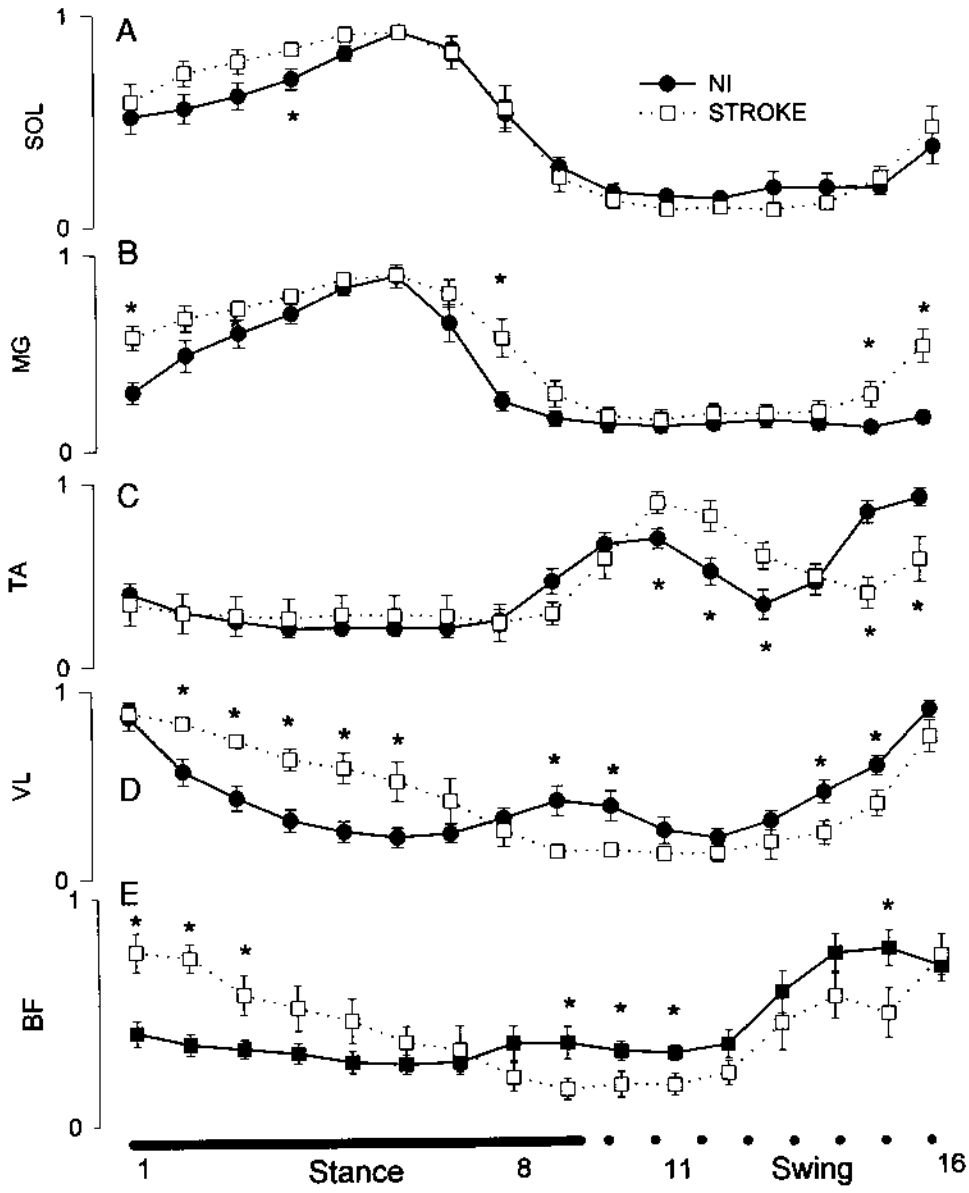

FIG. 1. EMG activity throughout step cycle for stroke (□) and neurologically intact (NI) subjects (● and ■). Values are normalized to peak muscle activation recorded during step cycle for each subject. Data points represent means ± SE. *, differences between subject groups at $P < 0.05$.

speeds for a given subject were pooled in the analysis. In this way, 13 data sets were analyzed for each group yielding 12 degrees of freedom for statistical analysis. For the NI subjects, 6 data sets at 2 km/h walking were included from our previous publication (Zehr et al. 1997b).

RESULTS

Locomotor cycle duration and EMG profiles

Although the two subject groups walked at different speeds, the relative timing of the step cycle was not different. Stance accounted for 66.2 ± 1.6% of the step cycle duration in stroke and 64.2 ± 1.6% in NI subjects (not significant).

Average values of the mean normalized EMG profiles for each part of the step cycle and for all five muscles studied in both groups of subjects were plotted in Fig. 1. NI subjects are represented by ● and stroke subjects by the □. At the bottom of the figure, (——) indicates those parts of the step cycle that fall in the stance phase and (· · ·) those that fall in the swing phase. Typically, the stance phase was parts 1–8 and the swing phase 10–15. The transitional portions of the step cycle, in which only part of the foot is in contact with the ground, occurred in part 9 (stance-to-swing) and part 16 (swing-to-stance). There were few differences in the relative activation of SOL and no differences when the maximum absolute val-

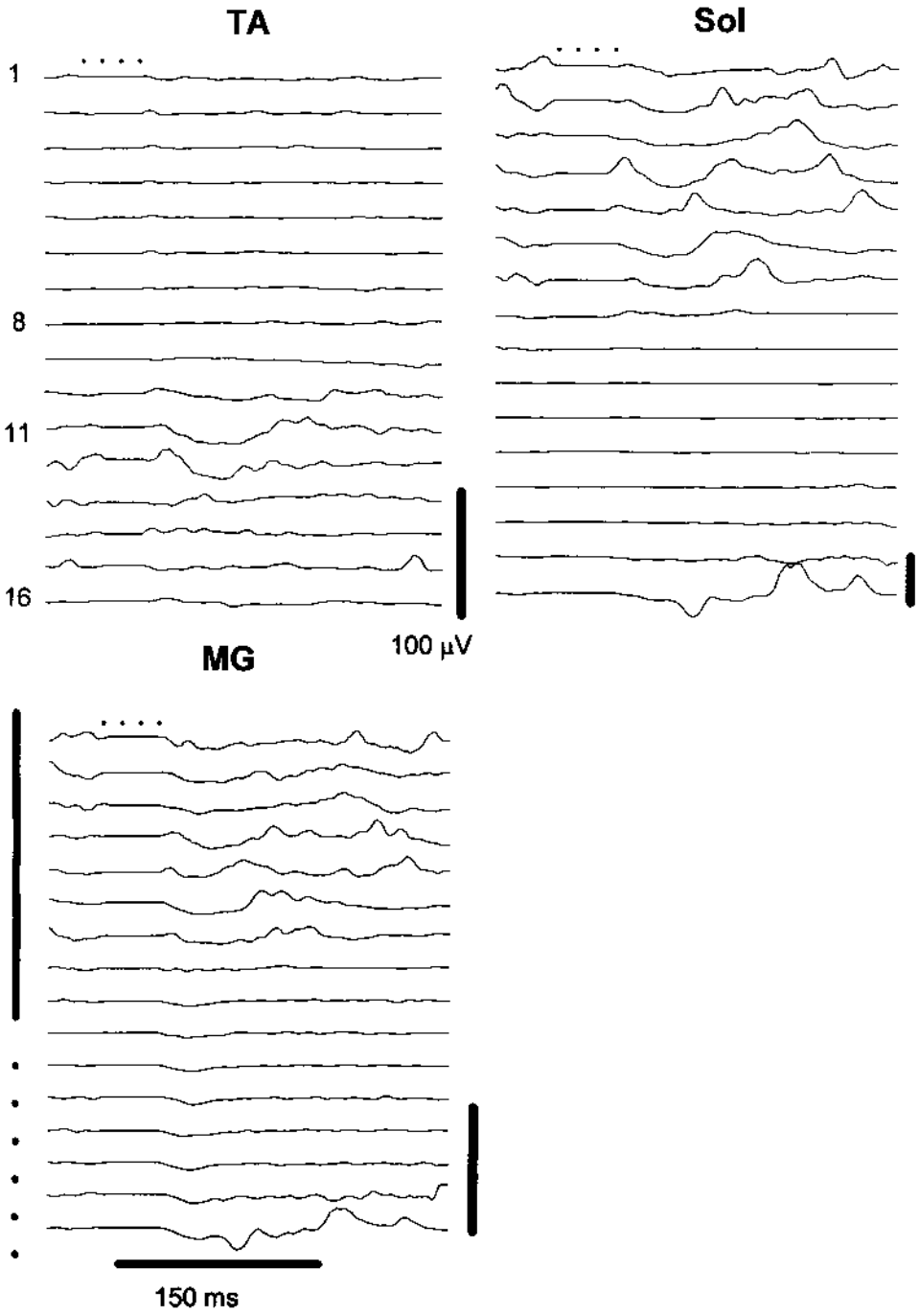

FIG. 2. Subtracted EMGs of tibialis anterior (TA, *top left*), soleus (SOL, *top right*), and medial gastrocnemius (MG, *bottom left*) muscles of a representative stroke subject (aged 56 yr) after superficial peroneal (SP) nerve stimulation. Stimulus artifact was suppressed in all traces and replaced with a flat line, atop which was placed a dashed line. Each trace has 50 ms before and 250 ms after stimulation. Numbers (*left*), specific portions of step cycle; (——) and (· · ·), approximate duration of stance and swing portions of step cycle.

ues were compared (45.6 vs. 39.0 μV for stroke and NI, respectively). Relative MG muscle activation was significantly greater at the transition from the end of swing to the beginning of stance (*, significant differences in Fig. 1) in stroke compared with NI subjects, but stroke subjects had reduced maximal MG activation (27.5 vs. 84.9 μV, $P < 0.01$).

Most importantly because it relates directly to the foot drop in the stroke subjects, the TA muscle activation profile during swing was quite different between stroke and NI subjects. Whereas NI subjects had peak TA activation at end swing (part 15) or the swing to stance transition (part 16), stroke subjects showed peak activation in early to mid swing and the end swing burst was significantly reduced. The absolute values of TA EMG were also lower ($P < 0.05$) in stroke subjects during late stance and swing. However, although reduced as compared with NI subjects 29.6 vs. 41.6 μV), the maximal activation level in TA was not significantly different between the two groups across the step cycle. In the upper leg muscles VL and BF, stroke subjects generally had highest muscle activation during stance and lowest muscle activation during swing in contrast to NI subjects. The absolute activation levels were lower in both VL (21.9 vs. 41.2 μV) and BF (24.3 vs. 32.4 μV) muscles in stroke subjects as compared with NI, but these differences were not significant.

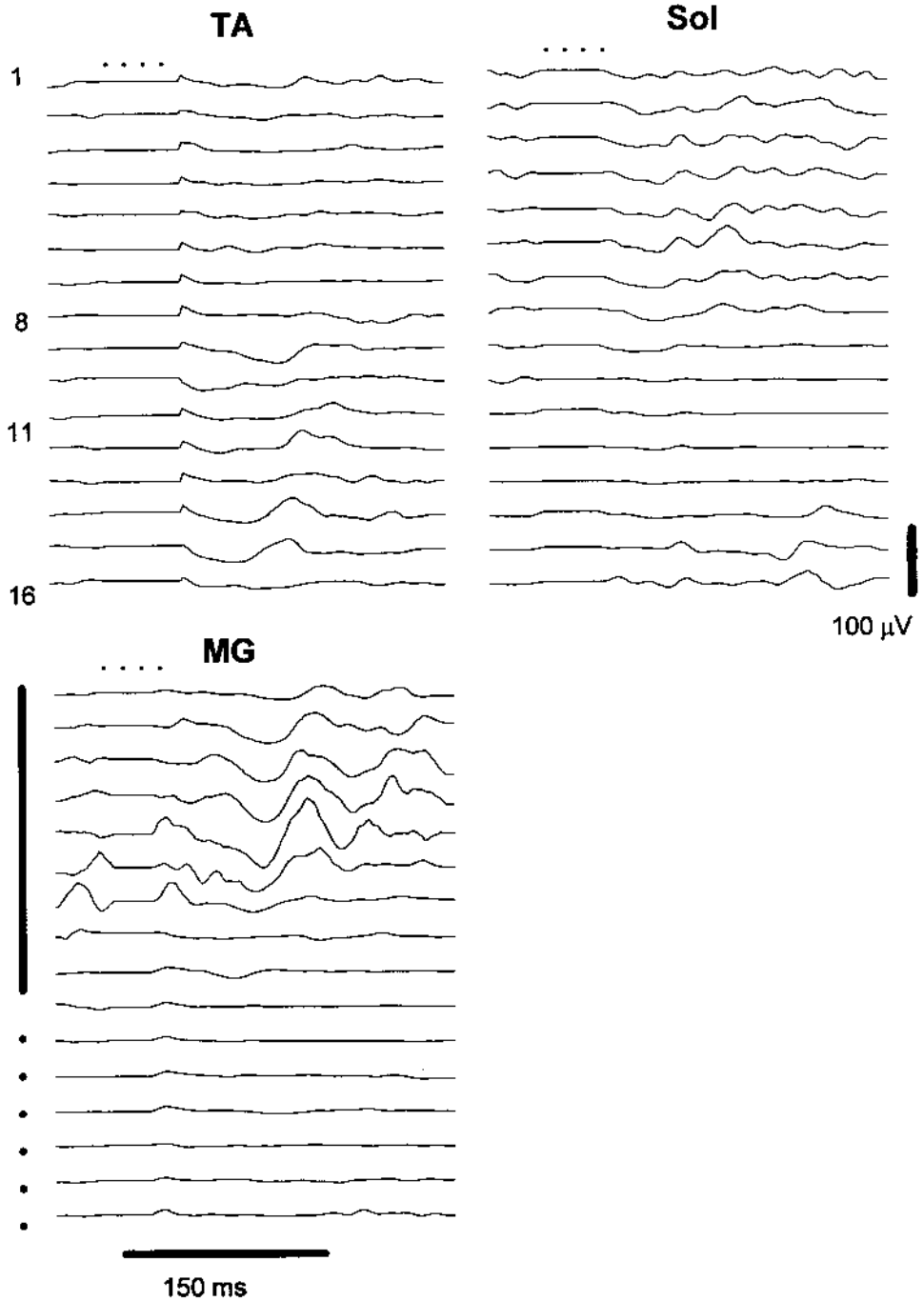

FIG. 3. Subtracted EMGs of lower leg muscles in a sample NI subject (aged 65 yr) after SP nerve stimulation. Format is same as Fig. 2.

Lower leg responses

The responses (after subtraction of the background EMG) to SP nerve stimulation in each of the 16 parts of the step cycle for TA, SOL, and MG muscles of one representative stroke subject (aged 56 yr) were plotted in Fig. 2. In this and all such figures, the solid vertical line indicates approximately the portions of the step cycle, which fall in stance and the dashed line those in swing. The horizontal dashed lines at the top of each muscle indicates approximately the stimulation period; the stimulation artifacts were removed for clarity in all figures. The solid calibration bar at the bottom right of each muscle indicates 100 μV. In Fig. 3, the same format was used to display data from one age-matched NI subject (55 yr). Note the complex pattern of responses especially at longer latency (~135 ms poststimulation) seen in the NI (particularly in MG) as compared with the stroke subject.

The overall response to stimulation ($ACRE_{150}$—see METHODS) was calculated for each subject and mean values for both groups of subjects are plotted in Fig. 4. Stroke subjects showed large suppressive responses throughout stance (Fig. 4, *top*) and no significant responses during swing in SOL muscle. In contrast, NI subjects had little net response except for some minor, but statistically significant (*, values that are significant at $P < 0.05$) facilitation during late swing (parts 13–15, Fig. 4*A*) in SOL. Both groups of subjects had significant suppressive responses in MG muscle during mid to late stance and NI subjects also showed some facilitation at end swing. The responses in MG muscle were significantly correlated to angle changes during stance in both groups ($r = 0.63$ and 0.55 for NI and stroke subjects, respectively).

In TA muscle, stroke and control subjects showed large and significant suppression in the early part of swing phase (parts 10 and 11 in Fig. 4*C*). In the stroke subjects stimulation produced about a 30% suppression of TA activity in early swing, though a smaller than normal response in late swing (all values are measured relative to the peak activity observed during the step cycle without stimulation). Along with the effects in EMG, both groups also

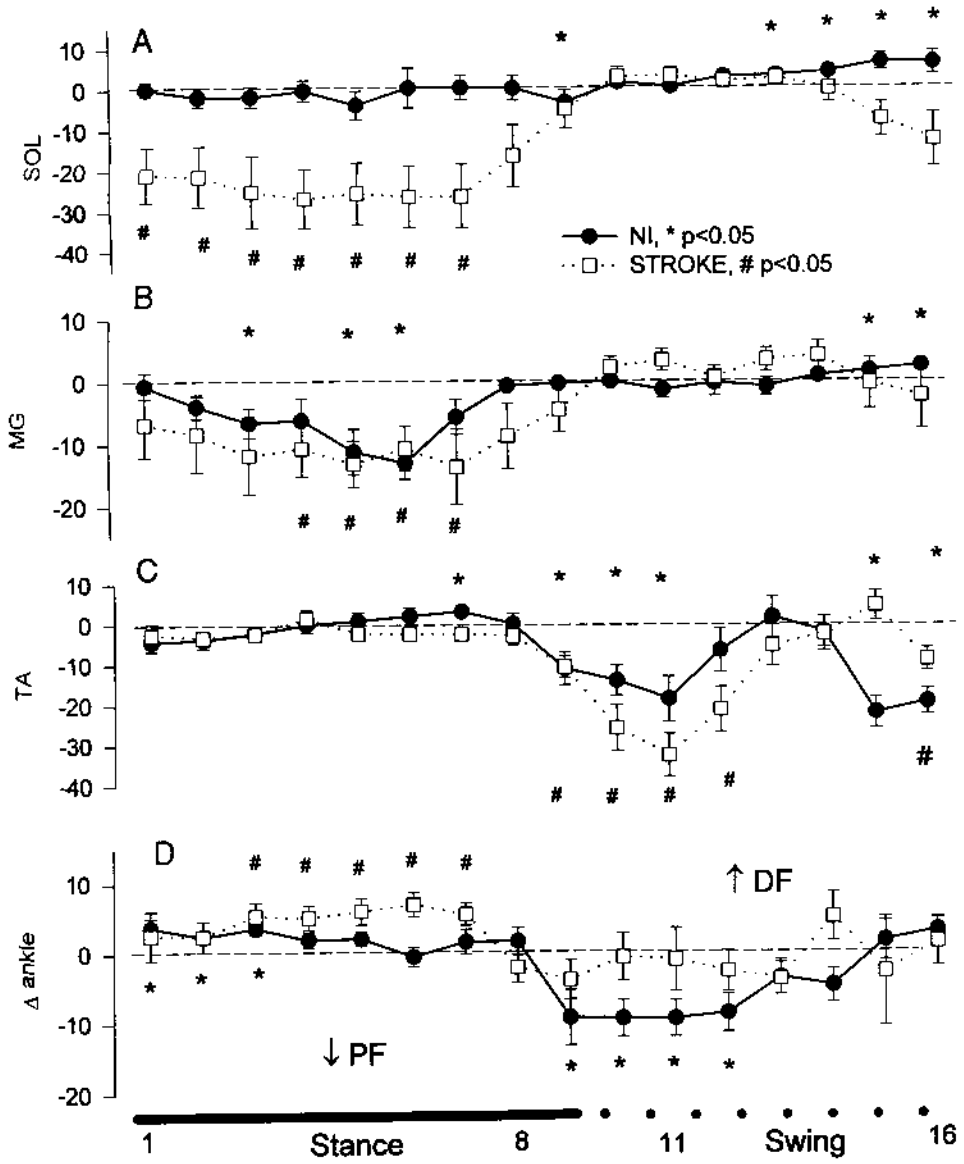

FIG. 4. Mean values for average cumulative reflex ($ACRE_{150}$, as percentages of maximum locomotor EMG) for lower leg muscles and ankle angle changes in both groups. * (NI, ●) and # (stroke subjects, □), statistically significant effects at $P < 0.05$. DF, dorsiflexion; PF, plantarflexion.

had associated changes in ankle joint angle because of stimulation (Fig. 4*D*). The stroke subjects showed significantly increased ankle dorsiflexion throughout stance (labeled as a change in the plantarflexion (PF) direction in Fig. 4*D*), whereas NI subjects showed only some minor increase in ankle dorsiflexion during early stance. The major response in NI subjects was a reduction in ankle dorsiflexion during the stance to swing transition and early to mid swing (parts 10 to 12), which was much less prominent in stroke subjects. TA suppression was correlated with the reduced ankle dorsiflexion in only one part (early swing, $r = 0.62$) of the step cycle for the stroke but in one-third of the step cycle for NI subjects ($r = 0.60$–0.75). Plotted in Fig. 5, *top*, are the correlations between ankle angle and TA effect for early swing for stroke (part 11; □) and NI subjects (part 10; ●). Data from late swing (part 14) are plotted at the bottom of Fig. 5. Note that only NI subjects show significant correlation at this part of the step cycle.

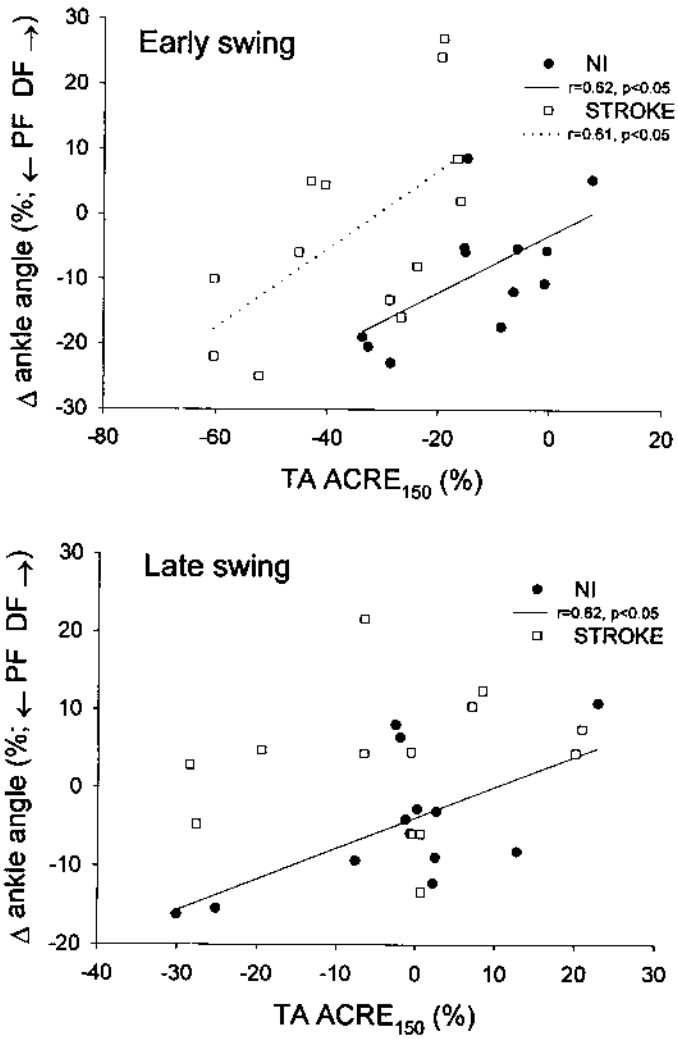

FIG. 5. Data from lower leg of NI and stroke subjects for normalized $ACRE_{150}$ and kinematic changes during swing. *Top*: data for early swing (□ and ●, stroke and NI subjects, respectively). *Bottom*: late swing. Significant regression is indicated by solid (NI) and dashed (Stroke) lines. Note that correlation between TA effect and ankle angle was seen only in NI subjects during late swing.

Upper leg responses

Figures 6 and 7 show data from the upper leg of the same stroke and age-matched NI subjects as presented in Figs. 2 and 3. Note that the stroke subject has a predominant net suppressive response in both muscles, which is in clear contrast to the facilitation seen in the NI subject.

Mean values for VL and BF muscles and changes in knee angle were plotted for both groups of subjects in Fig. 8. As with the lower leg extensor muscles SOL and MG, VL muscle was significantly suppressed in the stroke subjects during most of stance (parts 1–6) and at late swing and the swing to stance transition (parts 15 and 16, Fig. 8*A*). In contrast, VL muscle was significantly facilitated throughout stance (parts 2, 4, 6, and 8, Fig. 8*A*) and mid-to-late swing (parts 12, 14, and 15) in the NI subjects. With regard to BF muscle, stroke subjects had significant facilitation only at the stance to swing transition (part 9, Fig. 8*B*) and during early swing (parts 10–12), whereas NI subjects showed significant facilitation throughout the step cycle, reaching a peak at the stance to swing transition. In concert with the suppression in VL muscle during early and midstance, knee extension was reduced during stance (parts 4, 6 and 7, Fig. 8*C*) in the stroke subjects. Knee extension was also reduced in NI subjects in concert with BF facilitation, but the prominent effect in these subjects was increased flexion during swing.

In the stroke subjects, there was significant correlation between reflexes in both BF and VL muscles and knee angle changes during stance (data from several parts of stance are plotted in Fig. 9; largest $r = 0.85$ and 0.86 for BF and VL, respectively), but this was essentially absent in the NI subjects. NI subjects had significantly increased knee flexion during late swing, which was correlated to the net effect in BF muscle ($r = 0.66$, $P < 0.05$).

DISCUSSION

Taken together the responses in the stroke subjects show some similarity to the NI subjects during swing and are similar to those reported by us previously (Zehr et al. 1997b). However the stroke subjects show several interesting differences. Firstly the stroke subjects showed suppression during stance in all extensor muscles studied (SOL, MG, and VL), which were associated with and correlated to changes in knee and ankle kinematics. Secondly, during swing they showed a similar suppression of TA muscle as in the NI subjects, yet the angular changes were much smaller and significant correlation between the kinematic and EMG indices occurred at only one portion of the step cycle. Thirdly, stroke subjects, had significant correlation between knee joint changes and upper leg reflexes during stance, unlike the NI subjects.

Do stroke subjects have a stumble correction to foot dorsum stimulation?

Previously we described significant correlation between TA and BF reflex effects and ankle and knee joint angle after SP nerve electrical stimulation in young, neurologically intact subjects (Zehr et al. 1997b). We suggested that these responses represented a portion of a stumble correction to activation of the cutaneous field overlying the foot dorsum.

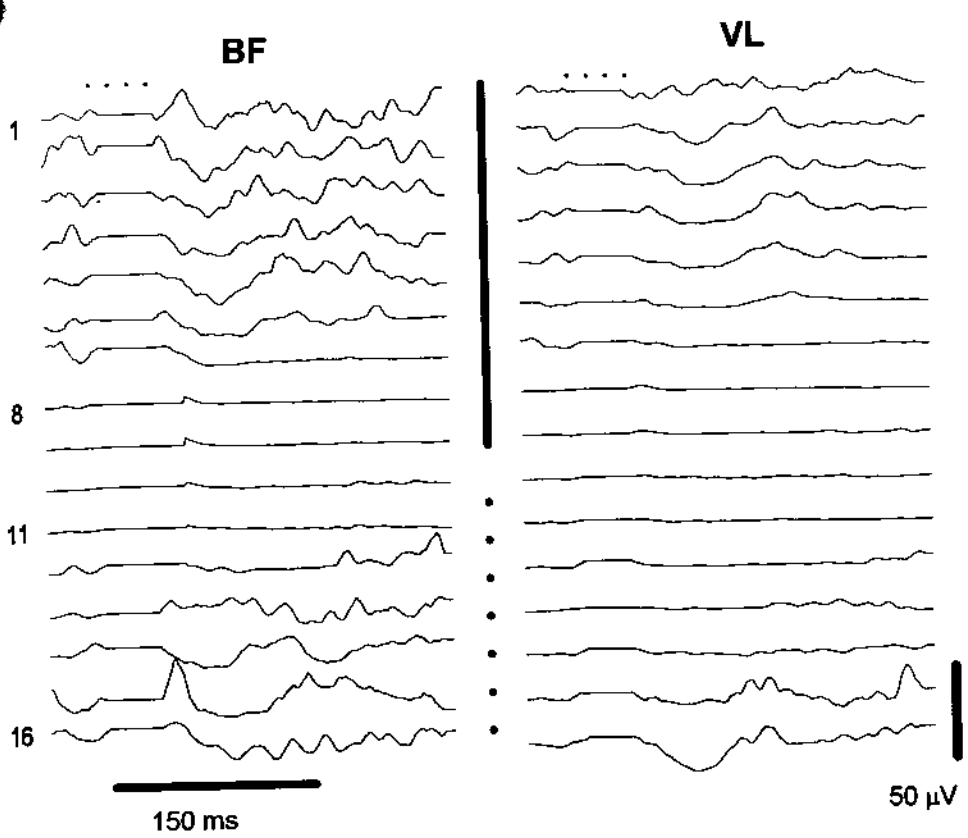

FIG. 6. Sample data from representative stroke subject; EMG responses in BF (*left*) and VL (*right*) muscles. Same subject and format as in Fig. 2.

That is, if the foot dorsum were contacted during swing a reduction of ankle dorsiflexion and knee extension would occur to prevent stumbling, which was replicated here in our expanded sample of NI subjects and is corroborated by recent results from Van Wezel et al. (1997). It should be noted that contact with an actual object would result in the activation of other receptors and reflexes, most notably the stretch reflex, in addition to cutaneous reflexes. After SP

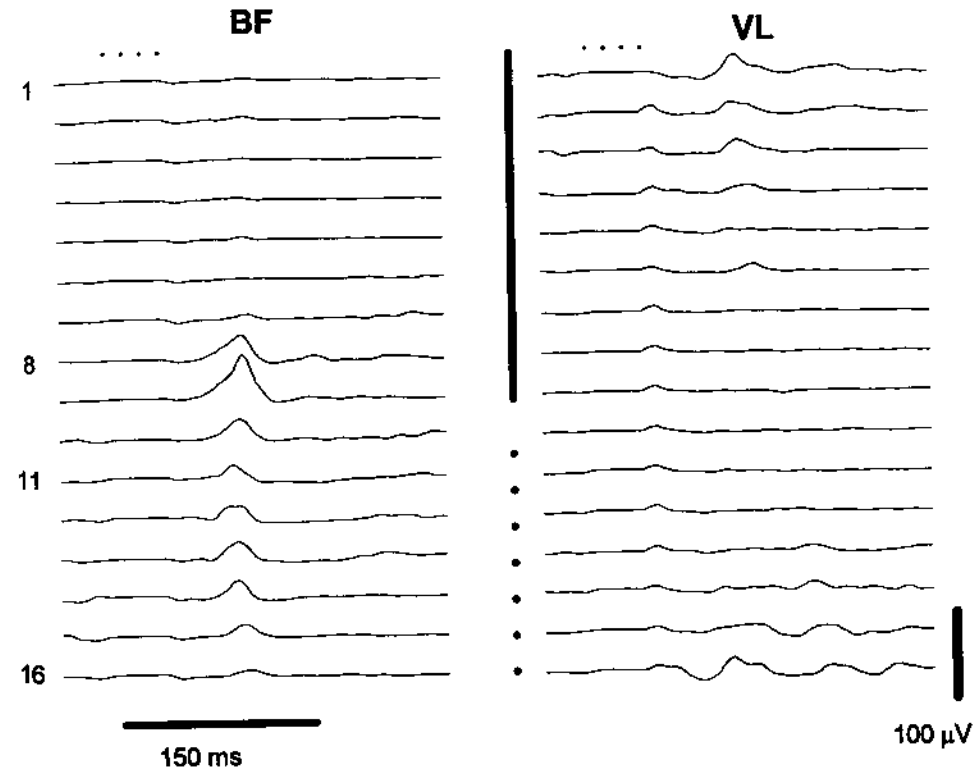

FIG. 7. Sample data from representative NI subject; EMG responses in BF (*left*) and VL (*right*) muscles. Same subject and format as in Fig. 3.

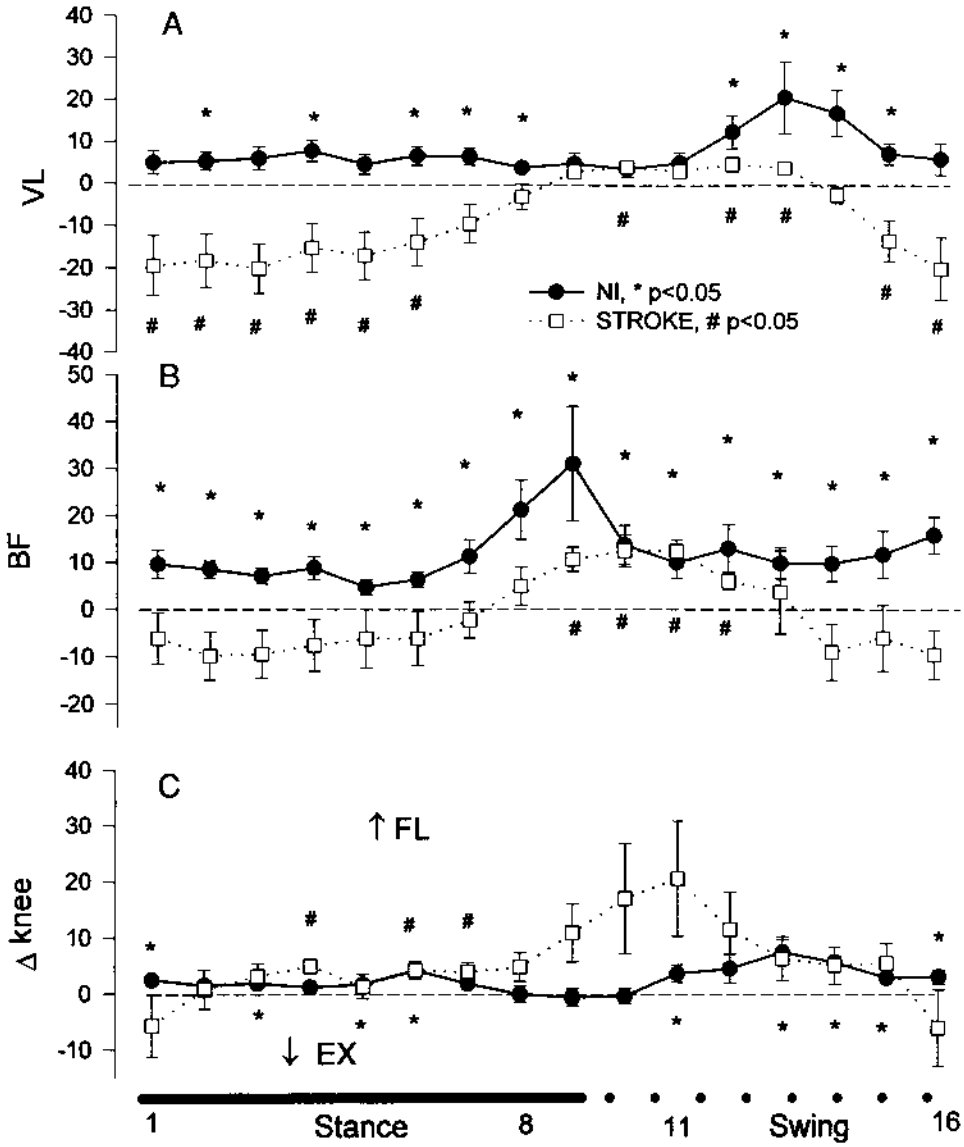

FIG. 8. Mean values for $ACRE_{150}$ (as percentages of maximum locomotor EMG) for upper leg muscles and knee angle changes in both groups. Format is same as Fig. 2. FL, knee flexion; EX, knee extension.

nerve electrical stimulation, the stroke subjects showed similar TA suppressive responses (early and late swing, Fig. 2*C*) and BF facilitation (early swing, Fig. 4*B*) to NI subjects. However significant mechanical changes did not occur simultaneously at the ankle (Fig. 4*D*) and knee (Fig. 8*C*) in the stroke subjects, except for a few parts during swing (see correlation in Fig. 5). Hence, only a portion of the previously described stumble corrective response to cutaneous electrical stimulation (Van Wezel et al. 1997; Zehr et al. 1997b) persists after stroke. This is reflected in the weaker correlation between EMG indices and kinematics during swing.

Why do stroke subjects not also have kinematic changes as large as NI subjects associated with the EMG effects? One explanation lies in a potential confounding effect of walking speed, which was significantly different in the two groups. The EMG reflex effects have a fixed duration and delay regardless of movement speed. By using TA and ankle angle as examples, a net suppression of ongoing locomotor activity might have a lesser effect at a slower rather than a faster walking speed. Our NI subjects found it unnatural and very difficult to walk at the slower speeds typically found in stroke subjects. The best we could do was to have them walk at speeds ranging from 1.5 to 4 km/h and extrapolate to the slower speeds found in stroke subjects. The same correlations were observed in the NI subjects regardless of speed. Hence the differences between NI and stroke subjects in the expression of the stumble correction is not likely to be the result of differences in walking speed between the two groups.

Another explanation might be the age differences in the subject groups. However an examination of the data of 2 age-matched subjects as was shown for the lower leg (Figs. 2 and 3) and upper leg (Figs. 6 and 7) reflexes reveals this to be unlikely. The patterns observed in the older NI subject are quite similar to that seen in the young NI subject (aged 26 yr) shown in Zehr et al. (1997b) (Fig. 2, lower leg, Fig. 4, upper leg). On balance, we are confident that the differences seen between the NI and stroke subjects result from the pathological changes arising from the stroke.

A more probable explanation may arise from the mechani-

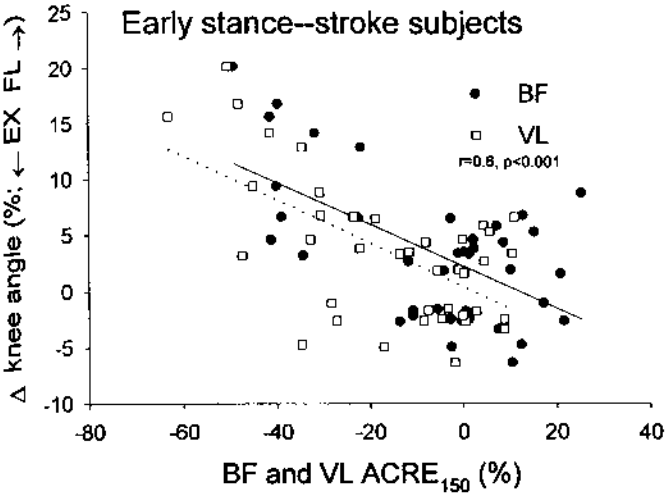

FIG. 9. Correlation between biceps femoris (BF) and vastus lateralis (VL) and knee angle for stroke subjects during early stance. □ and ●, BF and VL muscles, respectively. (——) and (· · ·): significant regression lines. Note that this correlation was not a prominent feature in NI subjects. Format is same as Fig. 5.

cal properties of the muscles and joint stiffness of the stroke subjects. Any correlation between EMG and kinematics will be affected by the inherent passive stiffness of the joint. If the passive stiffness is quite low, a large EMG reflex would be more highly correlated to a large kinematic change and vice versa. Sinkjaer and Magnussen (1994) showed that the passive stiffness in ankle plantarflexors was increased in hemiparetic subjects compared with healthy controls during isometric contraction. Recently, Sinkjaer et al. (1996) showed that passive stiffness was also significantly higher during locomotion in spastic multiple sclerosis subjects. Although we have not performed this analysis on our stroke subjects, the intrinsic and passive stiffness at the knee and ankle joint may well explain the lack of a kinematic stumble correction despite the neural responses in the stroke subjects.

Stroke subjects show prominent effects during stance

As described in RESULTS, the stroke subjects showed significant suppressive responses in all three extensor muscles studied (SOL, MG, and VL). These responses were associated with (see Figs. 4*D* and 8*C*) reductions in plantarflexion and knee extension. Changes in the upper leg were significantly correlated to both VL and BF and knee responses (see Fig. 9). These results are in contrast to the NI subjects in whom SOL was relatively unaffected and VL was in fact facilitated rather than suppressed during stance. It is difficult to argue for a functional role of these observed responses in stroke subjects during stance. It may indicate impaired reflex function during stance. The generalized suppression of extensor activity at a time when the ipsilateral limb is being loaded could be dangerous to the patient and lead to limb collapse and a fall. Just why the stimulation leads to such profound EMG and mechanical effects during stance (in contrast to the results in NI subjects) is not clear at present, but may be important in rehabilitation of these patients (see *Implications for rehabilitation*).

Cutaneous reflex modulation after stroke

Interestingly, although we previously showed a difference in triceps surae responses to SP stimulation (MG suppressed, lateral gastrocnemius, and SOL relatively unaffected) during stance (Zehr et al. 1997a), this was not found in the stroke subjects where suppression predominated in all extensor muscles during stance. Jones and Yang (1994) noted that tibial nerve cutaneous stimulation also caused suppressive responses in SOL during stance in spinal cord injured subjects, contrary to the modulation observed during gait in NI subjects. Further while we showed profound suppression of TA during swing after SP stimulation, Jones and Yang (1994) reported generally excitatory responses with no reflex reversal or modulation during swing. This is in contrast to NI subjects, where there is a reflex reversal from TA facilitation during early swing to suppression at end swing (Yang and Stein 1990) with kinematic correlates (Duysens et al. 1992; Zehr et al. 1997a).

If neurological impairment and spasticity from supraspinal lesions acts through a decreased descending control over interneuronal circuits within the spinal cord (Delwaide and Olivier 1987), it's not surprising that cutaneous modulation is also impaired or abnormal in stroke subjects. DeSerres et al. (1995) suggested that the reflex reversal and modulation seen in TA muscle after tibial nerve stimulation most probably results from the control of parallel excitatory and inhibitory pathways to TA motor units. This type of modulation was described in the cat by Degtyarenko et al. (1996), who suggested that segmental interneurons (whose excitability would be partially regulated by supraspinal input) play a dominant role.

Interestingly, Nielsen et al. (1997) recently showed that there may be a transcortical pathway from cutaneous afferents to TA motoneurons. They suggested that longer latency reflexes in TA evoked by sural and SP nerve stimulation are partly mediated by a transcortical connection. Although we did not make an extensive study of the latencies of the responses here (see METHODS), in Figs. 2 and 6 the longer latency (and generally facilitatory) responses are smaller in the stroke versus the NI subjects. This could lead to the large, net suppressive responses observed in the present paper. This interpretation is also consistent with previous studies showing reduced polysynaptic components in the stretch reflexes of spastic hemiparetic subjects subjected to treadmill acceleration during stance (Berger et al. 1984, 1988).

Implications for rehabilitation

The significant suppressive responses elicited in lower leg EMG after SP nerve stimulation may be used to modulate exaggerated reflexes such as those arising from spasticity (such as suggested by Fung and Barbeau 1994 for stimulation of the plantar foot surface and H-reflex modulation). Stimulation of the SP nerve during swing might reduce excessive stretch reflexes that impair normal dorsiflexion in spastic gait. However, two points must be kept in mind. Firstly, the stimulation of SP nerve could conceivably contribute to foot drop by suppression of residual TA activity during early swing, although foot drop was not noted in the kinematics of the studied stroke subjects.

Secondly, stimulation during stance had such large effects that consistent mechanical changes were observed in the stroke subjects in contrast to the NI subjects reported here and previously (Zehr et al. 1997b). However too much stimulation might compromise stability during stance, as mentioned above. It may be possible to phasically induce reflex changes when needed (see Fung and Barbeau 1994) to counter excessive excitation of TA (Jones and Yang 1994) or to prevent clonus at the beginning of stance (Yang et al. 1991).

In summary, stroke subjects appear to retain the neural component (as measured in the EMG), while the mechanical linkage in the stumble corrective response is weakened. Stroke subjects also have strong responses during stance in the extensor muscles, which are mirrored in leg kinematics. These responses may have possible therapeutic applications but might result in unwanted destabilization of balance and posture. Therapeutic application in these or more compromised subject populations will require further study.

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