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Deficits Underlying Impaired Visually Triggered Step Adjustments in Mildly Affected Stroke Patients

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Abstract

Background. The ability to make step adjustments while walking is often impaired following a stroke, but the basic sensorimotor control deficits responsible have not been established. *Objective*. To identify these deficits in Patients who have recovered from stroke leaving only mild lower limb impairment. *Methods*. Ten stroke and 10 age-matched control patients stepped onto an illuminated rectangle. In 40% of trials it jumped 140 mm either medially or laterally when the stepping foot left the ground, thus provoking a mid-step adjustment. In a separate block, patients performed the same task but with the body supported by a frame to eliminate balance responses. *Results*. Irrespective of support condition stroke patients produced short-latency foot trajectory adjustments compatible with a fast-acting, possibly subcortical, visuomotor process. However, the latency was slightly but significantly longer for the contralesional leg (148 ms) than the ipsilesional leg (141 ms) and longer than for controls (129 ms). Stroke patients' foot adjustments were executed slower and undershot the target more than controls. These deficits were most pronounced in the medial direction when the body was unsupported. The pattern of undershooting was the same for ipsilesional and contralesional legs. *Conclusions*. Mildly impaired stroke patients have deficits in initiating and executing visually triggered step adjustments but more profound difficulties with balance control during the adjustment, which caused them to suppress mid-step adjustments of foot placement in the medial direction where balance demands were greatest. Paradoxically, such suppression outside the laboratory may also threaten balance if it leads to unsafe foot placement or obstacle collision.

Keywords

stroke, gait, motor activity, balance, visuomotor

Introduction

Safe and independent ambulation in daily life is often compromised in persons who have sustained a stroke. They have a 3- to 10-fold increased risk of falling, mostly during walking activities, with balance and gait disorders being the most important risk factors.[1-6] When walking over uneven or cluttered terrain, one should be able to quickly and accurately adjust the ongoing step, for instance, to avoid an obstacle or to steer the foot toward a desirable landing area, while maintaining balance. Healthy persons are able to initiate such adjustments in response to new visual information at remarkably short latency (~120 ms) and to alter their foot placement successfully according to the environmental demands.[7-9] After a stroke, studies have shown that the ability to make step adjustments is often impaired,[10-15] but the exact underlying deficits and their interplay remain poorly understood. In general, step adjustments could fail either because the stepping leg is adjusted too late or because the movement itself is inadequate in some way. The movement is potentially quite complex and could be compromised for a number of reasons. There may be problems with planning a suitable new foot placement, an inability to move the foot with accuracy and speed to a new intended position, or difficulties implementing the associated movements that are required to maintain balance as the foot placement changes. Knowledge of the component deficits is fundamental for

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the development of targeted interventions to improve ambulatory abilities after stroke.

Here we aimed to identify whether deficient step adjustments after a stroke are due mainly to slowness of movement initiation, problems of movement execution, or an inability to integrate balance responses into the movement. To achieve this, we used a jumping-target acquisition stepping task in which a floor-mounted target suddenly and unpredictably jumped to a new position while the stepping foot was in the air, thus requiring a mid-step adjustment. The task was also performed with an external frame that supported the body to eliminate the need to make balance responses during the step adjustments. By contrasting step adjustments made with and without the supporting frame we hoped to ascertain whether the balance control system plays a role in step adjustment impairments after stroke. We studied a group of patients who had made a good recovery from stroke and who showed only mildly impaired lower limb function. They were chosen because we wished to identify which aspect of the step adjustment process is the most vulnerable following a stroke and, therefore, the most likely to contribute to poor walking ability in natural environments.

Figure 1. External frame used to support the subject when making foot movements in the supported condition.

Methods

Participants

Ten stroke patients (8 males; age: mean 50, SD 11.7, range 32-63 years) and 10 healthy control patients (4 males; age: mean 54, SD 15.0, range 32-69 years) participated with written informed consent and approval by the local ethics committee. Stroke patients were outpatients of the National Hospital for Neurology and Neurosurgery and were required to have suffered a supratentorial stroke (due to either hemorrhage or infarction) at least 6 months prior to the study and to have a Functional Ambulation Categories score of 5 (ability to walk independently).The exclusion criteria were hemineglect, hemianopia, or impaired understanding of the instructions. The healthy controls were selected if they were without neurological or orthopedic disease and were of a similar age ($P > .05$) to the stroke patients recruited. All patients were right footed (ie, the leg they use to kick a ball) prior to the stroke.

Clinical Measures

Muscle strength of hip flexors, knee extensors, and ankle dorsiflexors were scored using the Motricity Index[16] (maximum = 100%). Selectivity was scored using an abbreviated version of the lower extremity part of the Fugl-Meyer assessment,[17] not including the heel-shin items (maximum = 100%).

Experimental Setup and Protocol

Patients were tested while standing and wearing a safety harness. The stepping task was executed with the subject either unsupported or supported by a floor-mounted frame that contained padded bars that was fitted between the upper arms and the trunk (Figure 1). A small current ran through the participant that completed a circuit with the floor. The circuit was broken when a foot was lifted, which provided precise foot-off and foot-down times. Six target rectangles (14×21 cm), made from electroluminescent material whose luminance could be controlled electronically (Pacel Electronics, Poole, UK), were on the floor. Two were 16 cm directly in front of each foot, and the others were 14 cm lateral and medial to these.

A beep signaled trial onset. After a delay of 3 seconds, one of the targets directly in front of the feet was randomly selected and illuminated to inform the subject which foot to lead with and where to step. For the unsupported condition, patients were instructed to take a step forward to place their leading foot on the target and their trailing foot approximately alongside. For the supported condition, they were instructed to place their leading foot on the target and keep the other foot where it was. To regularize step duration, before each support condition patients underwent a training session in which a beep was sounded 450 ms after the foot

Table 1. Clinical Details

Subject	Age	Months Poststroke	Gender	Lesion Location	Type of Lesion	FM	MI
1	59	26	Male	R corona radiata, internal capsule, basal ganglia	Infarction	89.3	75
2	61	9	Male	R striatocapsular	Infarction	96.4	83
3	60	104	Male	R posterior MCA	Infarction	92.9	83
4	55	24	Male	R internal capsule	Hemorrhage	100	83
5	34	22	Male	L corona radiata, basal ganglia, temporal lobe	Infarction	100	100
6	40	28	Female	L corona radiata, internal capsule, basal ganglia	Infarction	96.4	100
7	32	34	Male	R internal capsule, temporal lobe, basal ganglia	Infarction	89.3	75
8	54	46	Male	L corona radiata	Infarction	89.3	83
9	63	16	Male	L internal capsule	Infarction	100	100
10	43	53	Female	R corona radiata, basal ganglia, sylvian fissure	Infarction	85.7	69

Abbreviations: FM, Fugl-Meyer lower extremity; MI, Motricity Index; MCA, middle cerebral artery.

left the floor. The subject was asked to make his/her foot strike coincide with the beep.

The experimental session was performed without step-duration feedback. In 40% of randomly selected trials the initial target was extinguished at the time of foot-off, and either the lateral target (20%) or the medial target (20%) was illuminated giving the appearance of a target jump. Patients were instructed to place their foot on the illuminated target when a jump occurred. Patients performed 40 steps with each leg for each support condition. Trials for the 2 support conditions were blocked with the unsupported block preceding the supported block, making a total of 160 steps. The unsupported block was performed first because it was considered to be the more difficult condition, and there was a concern that the stroke patients might fatigue more than control patients over the course of the experiment. A poorer performance in the more difficult unsupported condition might then be interpreted as a fatigue effect rather than being due to the extra balance demands associated with the unsupported condition. Rest periods were provided after a maximum of 40 trials.

Infrared light–emitting markers were attached to the big toe and the heel. Marker trajectories were sampled in 3 dimensions at 200 Hz using a Coda motion-capture system (MPX30 and CX1 units, Charnwood Dynamics, Leicestershire, UK). Each subject's footprint was chalk marked and digitized by tracing around it with an infrared marker at the end of the experiment. The footprint was coregistered with the positions of the 2 markers on the feet, thus enabling the footprint to be reconstructed wherever the foot landed during the recorded trials.

Data Analysis

The position of the footprint centroid was measured at the end of each trial. The error in foot placement was defined as the mediolateral distance of the foot centroid from the target centroid with positive values indicating undershoots.

Onset latencies of stepping adjustments were derived from the foot acceleration profiles. Mediolateral displacement of the toe marker was low-pass filtered (15 Hz) with a zero phase-shift filter before differentiating twice to derive acceleration. Onset latencies were defined as the time between foot-off and deviation of the mean mediolateral foot acceleration trajectory in jump trials with respect to mean control trials. Two experimenters, who were blinded to the patients' identities and group, independently judged the deviation onsets by eye from superimposed traces on a computer screen. The values across all latency measurements from the 2 raters were significantly correlated (Pearson correlation = .749; $P < .01$) and had a mean and SD difference of 4.65 and 15.95 ms, respectively. The latency measurement was taken as the mean of both raters' values.

The average speed of adjustment was calculated as the observed adjustment (14 cm jump minus foot placement error) divided by the time between adjustment onset and subsequent foot contact.

Foot-placement errors, latencies, and correction speeds were analyzed using a mixed between-subject and within-subject 4-factor analysis of variance (SPSS version 15.0). The between-subject factor was group (stroke/controls) and the within-subject factors were leg (affected/nonaffected), jump direction (medial/lateral), and support (with/without). For statistical purposes, we randomly chose the right or left leg of control patients (same proportion as stroke patients) to be the "affected" side. Leg effects in the stroke patients were analyzed by conducting additional analyses for the stroke group separately, including the same within-patients factors. The α level was set at .05.

Results

Individual stroke subject details are shown in Table 1.

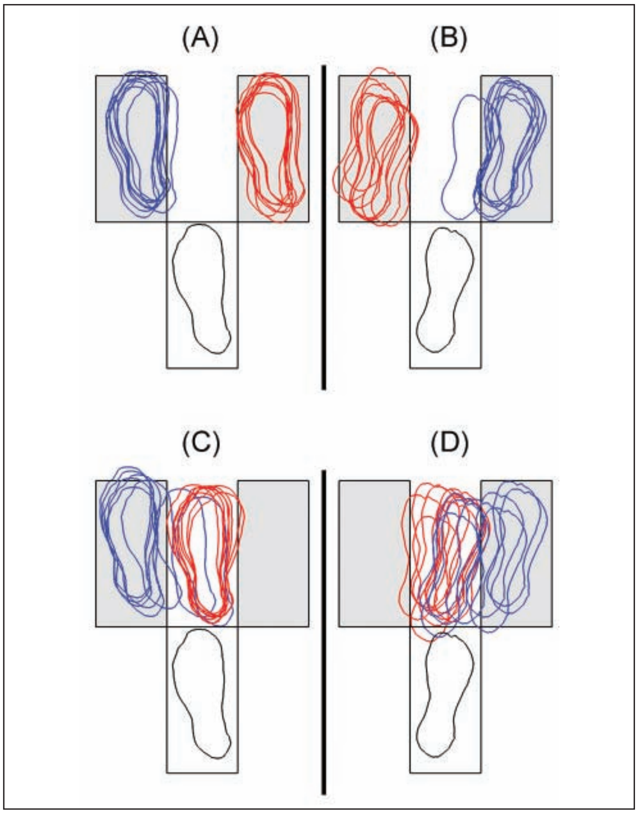

Figure 2. Footprints from a representative stroke subject. The subject was either supported by an external frame (A, B) or unsupported (C, D) during left-foot steps (A, C) and right-foot steps (B, D). Footprints show starting position (black) and final positions following either medial (red) or lateral (blue) target jumps. Gray rectangles denote targets. Steps when target did not jump are not shown.

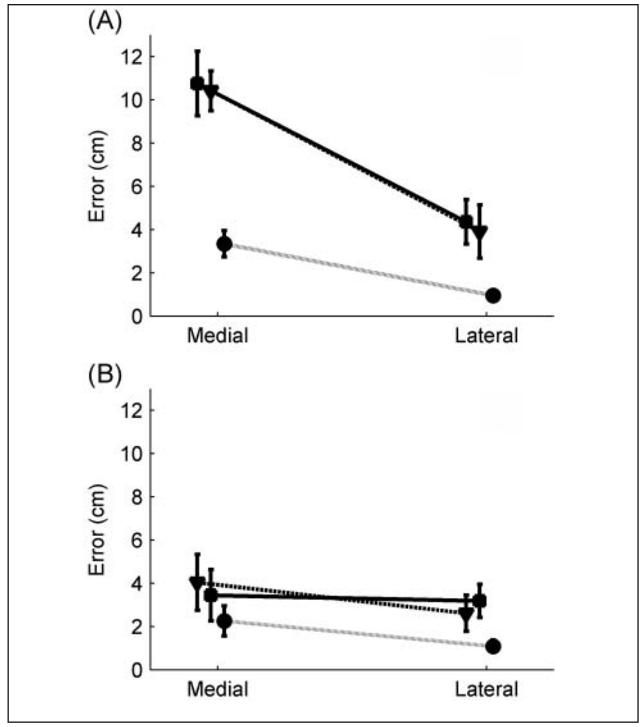

Figure 3. Mean (±standard error of mean) mediolateral foot placement errors. Data from stroke patients (affected side: dashed line, ▼; unaffected side: solid line, ■) and control patients (gray line, ●) during the unsupported condition (A) and supported condition (B). Positive values denote undershooting.

Foot Placement Accuracy

Figure 2 shows an example of foot placements from stroke subject. This subject was affected on the right side, but her performance was similar for both legs. When unsupported she successfully deviated her foot in the lateral direction to acquire the target but was not able to substantially adjust her step medially. During the supported condition, she adjusted her step reasonably accurately in both directions.

This pattern of foot placement was representative of the stroke group overall (Figure 3). In general, stroke patients undershot the target more than control patients (*group*, $F_{1,18}$ = 17.621, P = .001). Both stroke and control patients tended to undershoot the target more for medially than for laterally jumping targets (*jump direction*, $F_{1,18}$ = 75.409, P = .001), but stroke patients had particular difficulty with medial adjustments (*group* × *jump direction*, $F_{1,18}$ = 8.412, P = .010).

Patients tended to make smaller errors during the supported condition than during the unsupported condition (*support*, $F_{1,18}$ = 20.592, P = .001). Stroke patients benefited the most from support (*group* × *support*, $F_{1,18}$ = 12.759, P = .002), especially during the medial jumps (*group* × *jump direction* × *support*, $F_{1,18}$ = 6.188, P = .023).

In stroke patients, there was no significant difference in foot placement error between the affected leg and the nonaffected leg (*leg*, $F_{1,9}$ = 0.560, P = .474).

Onset Latencies

Figure 4 shows an example of the onset latencies derived from the mean acceleration curves of the affected foot of a stroke subject and a control subject. Group data are given in Table 2.

Onset latencies were modestly but significantly delayed in stroke patients by 15 ms on average (*group*, $F_{1,18}$ = 5.339; P = .034). Latencies were on average 9 ms faster for lateral than medial jumps (*jump direction*, $F_{1,18}$ = 17.490; P = .001), but there was no effect of support condition (*support*, $F_{1,18}$ = 1.239, P = .281). In stroke patients, the affected leg was on average 8 ms more delayed than the nonaffected leg (*leg*, $F_{1,9}$ = 5.194, P = .049).

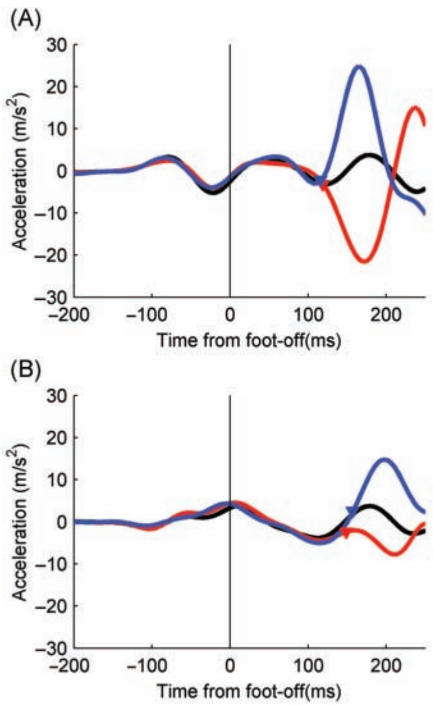

Figure 4. Mean acceleration of the foot in the mediolateral direction. Mean traces (lateral direction positive) from a control subject (A) and the affected leg of a stroke subject (B). Shown are control steps (black) and medial (red) and lateral (blue) jumps. ▼ denotes onset latency.

Swing Phase Duration

In control trials, the swing phase duration was the same for the stroke (369 ± 21 ms) and the control (361 ± 21 ms) patients (*group*, $F_{1,18} = 0.063$, $P = .805$), and there was no effect of support condition (*support*, $F_{1,18} = 2.770$, $P = .113$). In jump trials (Table 3), the swing phase duration during the supported condition was significantly longer (by 80 ms on average) than during the unsupported condition (*support*, $F_{1,18} = 23.32$, $P < .001$). The longer swing duration in the supported condition was particularly evident in stroke patients for the medial jumps (*group* × *jump direction* × *support*, $F_{1,18} = 4.734$, $P = .043$).

Correction Speed

Figure 5 illustrates for each condition the mean speed of the foot during the mediolateral correction phase in the jump trials. Overall, stroke patients' foot corrections were executed at a slower speed than control patients (*group*, $F_{1,18} = 10.995$, $P = .004$). Medial jumps were performed slower than lateral jumps (*jump direction*, $F_{1,18} = 24.769$, $P < .001$), but this medial slowness was more pronounced in stroke patients (*group* × *jump direction*, $F_{1,18} = 14.270$, $P = .001$).

The effect of body support on the correction speed depended on the direction of target jump (*jump direction* × *support*, $F_{1,18} = 12.692$, $P = .002$). In response to lateral jumps, for both groups, the foot tended to correct at a faster speed when the body was unsupported. However, this increase in speed was not observed for medial jumps. Crucially, for the stroke patients there was a dramatic decrease in correction speed in the medial direction when the body was unsupported (*group* × *jump direction* × *support*, $F_{1,18} = 5.394$, $P = .032$).

Discussion

In this study we have attempted to identify which components of the step adjustment process are impaired in patients who had made good recovery of lower limb function following a stroke. We asked specifically whether there were problems due to slowness of movement initiation, problems of movement execution, or an inability to integrate balance responses into the movement. We found 3 distinct aspects of mid-step adjustments that were deficient in our stroke patients. Two were related to problems with the initiation and execution of mediolateral movement of the stepping leg, and a third, we shall argue, was related to balance difficulties. These deficiencies led to a pattern of foot placement error that varied with target-jump direction and body support.

Delayed Response Latencies

Day and Lyon[18] demonstrated that 2 classes of response are involved in mid-flight arm corrections when reaching for a target that jumps. They suggested that the 2 responses are generated by separate visuomotor processes, one being fast (120-160 ms) and highly automatic and the other being slower (>160 ms) but more flexible. There is evidence that the fast component is driven by a subcortical visuomotor process both for the arm[19] and for the leg.[20,21] The latencies measured in the present experiments relate to this fast component. Although the stroke patients had delayed latencies, the mean delay was only a modest 15 ms, suggesting that the putative subcortical pathway was still available to them.

Delayed onset latencies have been observed previously in stroke patients when an obstacle-avoidance task was used to evoke stepping adjustments on a treadmill.[13] Obstacle-avoidance latencies in healthy patients[9,22] were remarkably similar to those observed in the present task (~120 ms), suggesting that the 2 tasks elicit similar responses. However, the delay in the stroke patients' obstacle-avoidance latency was considerably more pronounced than in the present study, being ~100 ms slower than normal. This is unlikely to be due to differences in balance demands because supporting the body with an external framework in the present study did not affect latency. One important difference between the

Table 2. Mean Onset Latencies (ms)

	Lateral Jump		Medial Jump	
	Unsupported	Supported	Unsupported	Supported
Stroke affected leg	152 ± 8	145 ± 7	148 ± 12	147 ± 5
Stroke nonaffected leg	130 ± 5	136 ± 4	143 ± 5	153 ± 12
Control patients	121 ± 3	126 ± 4	134 ± 4	136 ± 4

Table 3. Mean Swing Duration (ms)

	Lateral Jump		Medial Jump	
	Unsupported	Supported	Unsupported	Supported
Stroke patients	400 ± 30	497 ± 29	383 ± 23	507 ± 28
Control patients	418 ± 18	474 ± 17	408 ± 16	452 ± 17

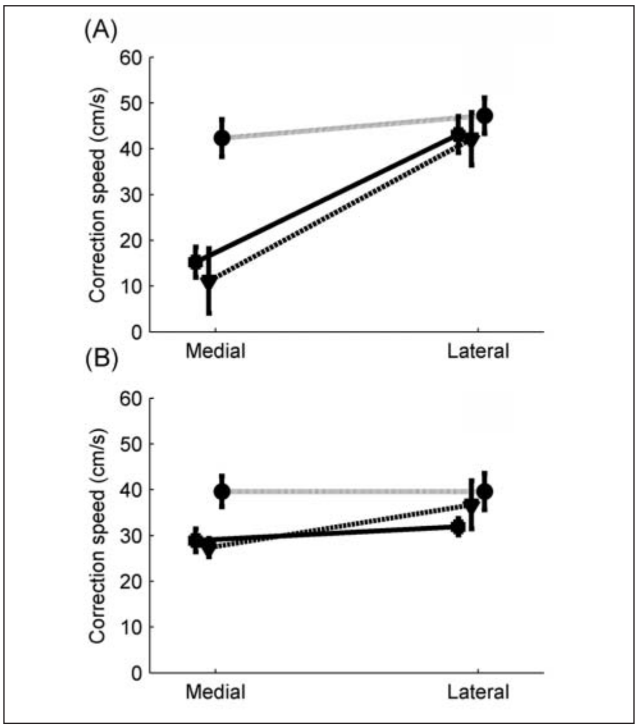

Figure 5. Mean (±standard error of mean) mediolateral correction speeds. Data from stroke patients (affected side: dashed line, ▼; unaffected side: solid line, ■) and control patients (gray line, ●) during the unsupported (A) and supported conditions (B).

2 tasks is that obstacle avoidance involves selecting a new foot placement from a number of possible locations, whereas for target acquisition there is no choice. The excessive delay with obstacle avoidance could arise, therefore, if there were a deficit in response selection. Another possibility is that onset delay may depend on the severity of the poststroke deficits. In the present study, lower extremity Fugl-Meyer scores were 86% to 100%, whereas they were 50% to 89% in the obstacle-avoidance study.[13]

Slow Correction Speeds

Although the 2 groups stepped forward at the same speed in unperturbed trials, during target jumps the stroke group executed mediolateral adjustments at a slower than normal speed. This deficit was more evident when the leg was moved in the medial direction and was similar for ipsilesional and contralesional legs. The most striking observation, however, was the further reduction in the stroke patients' medial correction speed when the body support was removed, a phenomenon that was not present in healthy patients or in either group when the leg was adjusted laterally. This extra slowing most likely stems from a balance control deficit (see below).

Foot Placement Error

Overall, stroke patients tended to undershoot the displaced target more than controls. Delayed onset latencies and slowness of movement would have contributed to this, although when the body was supported they were able to compensate to some extent by prolonging the duration of the step. Their reduced accuracy is in line with obstacle-avoidance tasks. When attempting to step over a stationary obstacle on a walkway, Said et al[10] found that the number of obstacle contacts was significantly higher for stroke patients than for controls. Similar results were reported for a time-critical obstacle-avoidance task on a treadmill but with more pronounced differences in failure rates.[12]

Stroke patients' foot placement error was similar for ipsilesional and contralesional legs. This is consistent with the obstacle-avoidance tasks described above[10,12] in that the number of obstacle contacts was not significantly different for the 2 legs. A possible explanation for the bilaterally impaired accuracy could be related to the fact that a mid-step adjustment is not a purely unilateral task. The appropriate execution of stepping adjustments (either to acquire a shifted target or to avoid an obstacle) depends on adjustments in the stance leg as well as the stepping leg.[8] The resulting bilateral muscle activation pattern probably engages both hemispheres. Any disruption of the coupled activity of the 2 hemispheres might therefore result in reduced accuracy for either leg.

Balance Deficits

Although stroke patients were generally less able than control patients to adjust their foot trajectory mid-step, there was 1 condition that stood out. Adjustments were excessively slow with large undershoots when they were unsupported and were required to move in the medial direction. This was not because they were unable to move their foot with greater speed or extent, as they showed when supported by the external framework. Rather, it suggests that balance constraints were at the root of this particular difficulty.

However, before we explore a possible balance deficit we must first consider an alternative explanation that arises from our protocol. The supported and unsupported conditions were performed as blocked trials with the more difficult unsupported task being performed first. We did this to avoid stroke-related fatigue effects corrupting the results (see Methods). It is possible though that this fixed-order blocked-trial method introduced a learning effect. Thus, the stroke patients may have performed better in the supported condition than the unsupported condition because they had by then learned to perform the adjustment. To investigate this we compared the foot placement error for the final trial of the unsupported condition with that of the first trial of the supported condition. We reasoned that any difference in these 2 temporally adjacent trials would be better explained by the presence of the support rather than through learning. The result of this analysis showed that for the stroke patients the foot placement errors were still very large in the last unsupported trial, whereas in the first supported trial the errors were substantially lower. The reduction in foot placement errors as a result of body support was significantly larger in the stroke patients compared to the controls (*group × support interaction*, $F_{1,18} = 5.094$, $P < .05$). Hence, the main findings described are likely to result from the effects of the support rather than learning.

Why should balance difficulties be particularly pronounced for a medial step adjustment? During a forward step the body's center of mass (CoM) lies medial to the supporting foot and so the body falls laterally (toward the side of the stepping foot) under gravity.[23] The fall trajectory is controlled by the mediolateral momentum imparted to the body prior to the step and is highly dependent on where the subject intends to land the stepping foot.[23,24] If the intended foot placement is changed mid-step, then ideally the trajectory of the fall also needs to change. However, previous work has shown that the CoM trajectory can be adjusted mid-flight to travel further in the lateral direction, but it seems virtually impossible to increase its travel in the medial direction.[25] Even without medial CoM trajectory adjustments it is still possible to adjust the foot placement medially to a limited extent, as long as the body's CoM lies inside the base of support when the stepping foot lands. However, this may present a secondary control problem because the support base (defined by the distance between the feet) would be made narrower when the foot lands. This would require finer mediolateral control to maintain balance after the step, which may be problematic for stroke patients because it is in the frontal plane that they have most prominent balance difficulties.[26]

Therefore, stroke patients may be compelled to suppress stepping adjustments in the medial direction to safely complete the step, thereby anticipating their potential instability. Such a feed-forward suppression mechanism could be functionally important for the prevention of falls. Indeed, the stroke patients in the present study never fell into the harness under any of the stepping conditions. In the real world, however, suppression of step adjustments could mean that the foot lands in a perilous location, which paradoxically may itself lead to a fall.

Implications

Although our stroke patients were very mildly affected, deficits could be demonstrated in onset latencies, movement speed, stepping accuracy, and their interactions with a compromised balance system. This was true even for the patients who had a maximum score in the Motricity Index and the Fugl-Meyer assessment, which are frequently used clinical tests to determine the severity of residual poststroke impairments. These "hidden" deficits may reflect the difficulties that even mildly affected stroke patients experience in walking over unpredictable terrain. The identified balance problem also highlights the importance of challenging dynamic stability in exercise programs aimed at improving gait adaptability and preventing falls. In elderly persons, agility training programs, including for instance obstacle courses, have been proven effective in improving gait adaptability and reducing the risk of falls.[27-29] Such programs are expected to be very beneficial for stroke patients as well, but their effects in this population still need to be demonstrated in randomized trials.

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Declaration of Conflicting Interests

The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

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