Reduced gait stability in high-functioning poststroke individuals

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Krasovsky T, Lamontagne A, Feldman AG, Levin MF. Reduced gait stability in high-functioning poststroke individuals. J Neurophysiol 109: 77–88, 2013. First published October 10, 2012; doi:10.1152/jn.00552.2012.—Falls during walking are a major cause of poststroke injury, and walking faster may decrease the ability to recover following a gait perturbation. We compared gait stability between high-functioning poststroke individuals and controls and evaluated the effect of gait speed on gait stability. Ten stroke subjects and ten age-matched controls walked on a self-paced treadmill at two speeds (matched/faster). Movement of the nonparetic/dominant leg was arrested unexpectedly at early swing. Poststroke individuals lowered the perturbed leg following perturbation (58% of cases) while controls maintained the leg elevated (49% of cases; P < 0.01). In poststroke individuals, double-support duration was restored later than in controls (4.6 ± 0.8 vs. 3.2 ± 0.3 strides; P < 0.007), and long-term phase shifts of arm and leg movements were larger and less coordinated on the paretic side. A moderate speed increase (~20%) enhanced the incidence of leg lowering in controls but not in stroke subjects. Faster walkers in both groups had a more coordinated response, limited to the nonparetic side in the stroke group. However, faster walkers were not more stable following perturbation. Our results suggest that gait perturbations can target basic control processes and identify neurological locomotor deficits in individuals with fall risk. Central regulation of body translation in space is involved in recovery of steady-state walking. Impaired descending control (stroke) decreases the ability of the motor system to recover from perturbations and regulate interlimb phase relationships, especially when changing gait speed. However, interlimb coordination may not be a major factor in the recovery of gait stability.

CVA; gait; interlimb coordination; locomotion; perturbation

ACCORDING TO DYNAMIC PATTERN theory (Kugler et al. 1982), stability of the locomotor system can be characterized by the ability to return to steady-state walking following perturbation. This ability is provided by position- and velocity-dependent intrinsic muscle properties and local or global whole body reactions. Responses to perturbations are controlled by descending and other neural systems that guide the translation of body equilibrium in the environment (Feldman et al. 2011). In healthy adults, a leg perturbation during gait generates a coordinated, global resetting of gait rhythm in all four limbs (Krasovsky et al. 2012), i.e., a shift in timing of gait events following perturbation, which presumably involves both spinal and supraspinal levels (Feldman et al. 2011).

If steady-state walking is not regained following perturbation, a fall may occur. Seventy percent of community-dwelling poststroke individuals fall during the first year (Weerdesteyn et al. 2008), and most falls occur due to loss of balance when walking (Hyndman et al. 2002) especially outdoors (Jørgensen et al. 2002). Even apparently well-recovered individuals continue to have deficits in lower limb mobility (Jørgensen et al. 1995; Keenan et al. 1984), which affect the ability to perform step corrections (Nonnemakers et al. 2010) and may also extend to walking. These deficits may be undetected by clinical scales, which do not evaluate the response to unexpected perturbations during walking. Evaluating deficits in gait stability may be especially important for high-functioning poststroke individuals, who are more likely to ambulate in complex environments. However, to date, stability in poststroke individuals has mainly been investigated during standing and not during walking (for a review, see Geurts et al. 2005). In this study, we compared the stability of steady-state walking in high-functioning poststroke and healthy control subjects by evaluating short-term and longer term responses to an unexpected perturbation during gait.

One of the common characteristics of poststroke gait is decreased gait speed (Olney and Richards 1996). Gait speed is an excellent predictor of survival in older adults (Studenski et al. 2011) and an important marker of function poststroke (Perry et al. 1995). As such, a major goal of poststroke gait rehabilitation is to increase gait speed. Poststroke individuals are able to voluntarily increase gait speed even in the acute stage (Lamontagne and Fung 2004). Increases in gait speed are known to be associated with improved arm-leg coupling (Kwakkel and Wagenaar 2002) and better temporal coordination between movements of the trunk segments (Wagenaar and Beek 1992). These improvements may occur through better descending control of neuronal coupling of upper and lower limbs (Ferris et al. 2006). However, the functional effect of increasing gait speed on walking stability is unknown. When walking faster, the available response time to an unexpected event is shortened (Pavol et al. 1999). Thus walking at slower speeds may be a protective mechanism against falls among older adults (Maki 1997) and possibly also after stroke. A perturbation during faster walking may challenge stability more in both healthy and post-stroke individuals. A better understanding of the relationship between gait speed and stability may provide guidelines for incorporating gait stability training into gait rehabilitation strategies that emphasize speed (Pohl et al. 2002; Sullivan et al. 2002). This may be especially important for well-recovered poststroke individuals, who are community walkers and thus may be more frequently exposed to gait perturbations.

To evaluate gait stability in well-recovered poststroke individuals, we used perturbations that have been shown to directly target the basic control processes underlying locomotion in humans, the
ability to regulate the gait phase, rhythm, choose between different patterns of responses, and, most important, influence translation of the body equilibrium in the environment to maintain stability despite the perturbation (Feldman et al. 2011; Krasovsky et al. 2012). In this way, we investigated how poststroke individuals recovered from a leg perturbation compared with age-matched healthy subjects walking at matched speeds. To match gait speed to comfortable speed of stroke subjects, healthy subjects were required to walk slower than their comfortable speed. We also investigated the role of gait speed in the ability to recover from a gait perturbation within each group by comparing responses at matched and faster speeds. We hypothesized that poststroke individuals would have less stable steady-state walking than controls. Specifically, at matched speeds, recovery of steady-state walking after a perturbation would take longer in poststroke individuals. We further hypothesized that walking faster would negatively influence gait stability in both groups. Finally, we hypothesized that the ability to recover steady-state walking after perturbation would be related to the clinical severity of the poststroke hemiparesis. Preliminary results have appeared in abstract form (Krasovsky et al. 2010).

**METHODS**

**Participants**

The research protocol and the consent form signed by all participants were approved by the Ethics Committee of the Centre for Interdisciplinary Research in Rehabilitation. Experiments were conducted using the protocol previously described (Krasovsky et al. 2012). A convenience sample of 10 male stroke subjects (aged ≤76 yr; Table 1) who had sustained a single stroke >9 mo previously in either hemisphere were recruited, after signing informed consent forms. Inclusion criteria were the ability to walk for at least 10 consecutive steps without assistive devices and to understand verbal instructions. Subjects were excluded if they had visuospatial neglect or visual field deficits according to their medical record, marked arm motor deficits: 

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age</th>
<th>Height, cm</th>
<th>Weight, kg</th>
<th>Time Since Stroke, mo</th>
<th>Side, Type, Site of Lesion</th>
<th>FGA (/30)</th>
<th>ABC (/100)</th>
<th>Overground gait speed, m/s (10-m walk)</th>
</tr>
</thead>
<tbody>
<tr>
<td>S1</td>
<td>54</td>
<td>185.4</td>
<td>95.0</td>
<td>48</td>
<td>L, ischemic, MCA-internal capsule</td>
<td>17</td>
<td>91.25</td>
<td>1.29</td>
</tr>
<tr>
<td>S2</td>
<td>72</td>
<td>182.0</td>
<td>99.7</td>
<td>83</td>
<td>R, ischemic, MCA</td>
<td>19</td>
<td>87.50</td>
<td>1.00</td>
</tr>
<tr>
<td>S3</td>
<td>69</td>
<td>175.2</td>
<td>93.0</td>
<td>9</td>
<td>R, ischemic, bulbar</td>
<td>21</td>
<td>81.80</td>
<td>1.27</td>
</tr>
<tr>
<td>S4</td>
<td>57</td>
<td>166.0</td>
<td>98.4</td>
<td>9</td>
<td>R, ischemic, caudate nucleus</td>
<td>22</td>
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<tr>
<td>S5</td>
<td>58</td>
<td>183.0</td>
<td>99.0</td>
<td>15</td>
<td>L, ischemic, thalamus, internal capsule</td>
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<td>39</td>
<td>162.0</td>
<td>65.0</td>
<td>34</td>
<td>R, hemorrhagic, subcortical</td>
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<td>85.31</td>
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<td>52</td>
<td>172.0</td>
<td>87.0</td>
<td>27</td>
<td>R, ischemic, anterior choroidal artery</td>
<td>24</td>
<td>94.38</td>
<td>1.35</td>
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<tr>
<td>S8</td>
<td>70</td>
<td>182.0</td>
<td>80.0</td>
<td>13</td>
<td>L, hemorrhagic, putamen, internal capsule</td>
<td>27</td>
<td>95.90</td>
<td>1.11</td>
</tr>
<tr>
<td>S9</td>
<td>56</td>
<td>185.0</td>
<td>90.7</td>
<td>39</td>
<td>R, ischemic, MCA</td>
<td>27</td>
<td>86.80</td>
<td>1.35</td>
</tr>
<tr>
<td>S10</td>
<td>76</td>
<td>170.0</td>
<td>63.5</td>
<td>12</td>
<td>L, ischemic, subcortical</td>
<td>28</td>
<td>84.38</td>
<td>1.17</td>
</tr>
<tr>
<td>Means</td>
<td>60.3</td>
<td>176.3</td>
<td>87.1</td>
<td>28.9</td>
<td></td>
<td>23.2</td>
<td>86.30</td>
<td>1.22</td>
</tr>
<tr>
<td>SD</td>
<td>11.3</td>
<td>8.4</td>
<td>13.5</td>
<td>23.4</td>
<td></td>
<td>3.6</td>
<td>11.00</td>
<td>0.22</td>
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</table>

Control (n = 10)

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age</th>
<th>Height, cm</th>
<th>Weight, kg</th>
<th>Time Since Stroke, mo</th>
<th>Side, Type, Site of Lesion</th>
<th>FGA (/30)</th>
<th>ABC (/100)</th>
<th>Overground gait speed, m/s (10-m walk)</th>
</tr>
</thead>
<tbody>
<tr>
<td>S1</td>
<td>66.4</td>
<td>174.1</td>
<td>83.9</td>
<td>28.7†</td>
<td>R, left; L, middle cerebral artery, FGA, Functional Gait Assessment; ABC, Activities-Specific Balance Confidence Questionnaire. *P &lt; 0.05; †P &lt; 0.01.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SD</td>
<td>4.2</td>
<td>8.7</td>
<td>9.2</td>
<td>1.3</td>
<td></td>
<td>3.7</td>
<td>0.2</td>
<td></td>
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positioned behind the treadmill. Movement of the swing leg was arrested by actuating one of the solenoids to clamp the rod for 250 ms at ~20% of swing length (Krasovsky et al. 2012). The perturbation was applied to the dominant leg in control subjects, determined as the preferred leg used for kicking, and to the nonparetic leg in stroke subjects. Subjects were instructed to continue walking despite the perturbation. Since the response to the first perturbation may differ from those of subsequent perturbations (Marigold and Patla 2002; but see also, Visser et al. 2010), we allowed subjects to habituate to 3 initial perturbations before collecting data from the 12 experimental trials. Movement kinematics were recorded with 12 Vicon-512 cameras (120 Hz; Vicon, Oxford, UK) and filtered using a dual-pass 6th-order Butterworth filter (cutoff 10 Hz).

Data Analysis

Gait speed on the self-paced treadmill before perturbation was quantified by dividing step length by step time and averaging the result over three strides. Swing length and time and sagittal ranges of motion of the arms during nonperturbed walking were measured using movement of heel and finger markers. Several variables were calculated to evaluate recovery from perturbation in both the short (1–2 steps) and longer term (3 steps and more).

Short-term responses. The immediate response of the perturbed leg to perturbation was classified into three leg strategies (Eng et al. 1994; Schillings et al. 2000; Krasovsky et al. 2012). In the lowering strategy, the perturbed leg was lowered to the ground with a reduced step length and time. In the elevating strategy, the perturbed leg was maintained in the air for a prolonged period and the step length was the same or longer than in unperturbed steps. In the combined strategy, an elevation strategy was attempted but the leg was eventually lowered at a shorter step length. For each subject, the percentage of occurrence of each leg strategy was calculated.

Swing length was measured as the antero-posterior distance between heel positions at toe-off and foot-contact of the same leg on the
Longer term recovery from perturbation was quantified by the number of strides required to regain double-support duration and by whole body phase shifts (Krasovsky et al. 2012). Double-support is defined as the time period between foot-contact and the consecutive toe-off of the contralateral leg. Double-support duration is reportedly longer in unperturbed walking in older compared with young adults (Winter et al. 1990) and has also been reported to be increased in poststroke individuals (Olney et al. 1994). In our study, double-support duration was compared before and after perturbation. Most of the variability of the double-support duration measure stems from the asymmetry between the durations of the double-support phases 1 and 2 within the gait cycle, rather than from baseline variability of double-support durations of each side. Thus double-support durations were calculated separately for each double-support phase, where the leading leg was the paretic/nondominant or the nonparetic/dominant leg. Three preperturbed cycles were averaged for each side, and the number of strides required to regain the baseline double-support time was determined. Double-support recovery time was defined as the first stride at which both double-support durations returned to baseline ± 10%. A value of 10% was chosen since baseline variability did not exceed 10% in either group.

The longer lasting change in gait stability following perturbation was measured by the phase shift between pre- and postperturbed limb positions (Feldman et al. 2011). Antero-posterior positions of the heel and finger markers from three preperturbation cycles were projected forward from the last foot-contact before perturbation (legs) or from the forward to backward movement reversal point (arms; Fig. 1B). The phase difference between the actual and the projected limb positions in each of three postperturbed cycles was computed as:

\[ \varphi = 360^\circ \left( T_\text{pre} - T_\text{post} \right) \]

where \( T_\text{pre} \) was the projected cycle period and \( T_\text{post} \) was the minimal time difference between projected and actual foot-contact or reversal point, respectively. The sign of \( \varphi \) was considered negative for phase advances and positive for phase delays compared with the unperturbed limb position.

Interlimb coupling. In young healthy individuals, the degree of phase shift following a leg perturbation was similar between all four limbs (Feldman et al. 2011). In this study, we compared absolute differences in phase shifts between arm-leg pairs on the paretic (nondominant) and nonparetic (dominant) sides (arm-leg coupling), as well as between the two arms and between the two legs. Phase shift differences allow for estimation of temporal coupling between segments regardless of possible differences in range of motion.

Statistical Analysis

Student’s \( t \)-tests were used to compare clinical scores of poststroke individuals and controls, as well as to test for group differences in preperubtturbed walking patterns and immediate leg response strategies following perturbation. Paired \( t \)-tests evaluated the effect of walking speed on leg response strategies. Since the immediate response strategy may affect other response parameters, it was used as an independent variable in further analyses (Krasovsky et al. 2012). Swing length, swing time, and double-support recovery at matched speeds were compared between groups and across strategies using generalized estimating equations (GEE) with an exchangeable working correlation matrix. GEE is a statistical method that takes into account the intersubject correlation of responses and is designed to handle missing data compared with repeated-measures ANOVA, which requires complete, balanced datasets (Ballinger 2004). Walking speed in individual trials was a covariate in the model. For phase shifts of all four limbs (circular variables), Harrison-Kanji tests were used, i.e., circular two-way ANOVAs with strategy and group as independent variables (Harrison and Kanji 1988). GEE was used to investigate the effect of response strategy and speed condition on recovery from perturbation in both groups.

Pearson correlations were used to relate performance on functional tests (FGA, ABC for both groups, and CM for the stroke group) and parameters of recovery from perturbation. Spearman rank order correlations were used for non-normally distributed data (Shapiro-Wilk test). Analyses were carried out in SPSS (version 17.0), except for circular data analyses that were done in Matlab 6.5 (Mathworks, Natick, MA) using the circular statistics toolbox (Berens 2009). A significance level of \( P < 0.05 \) was used for all tests.

RESULTS

Stroke subjects and controls were similar in age, height, and weight (Table 1). Overground walking speed was slower (10MWT, \( t_{18} = 3.49, \ P < 0.004 \)), FGA (\( t_{18} = 4.55, \ P < 0.002 \)), and ABC scores (\( t_{18} = 2.78, \ P < 0.019 \)) were lower in stroke compared with control subjects. Examples of sagittal trajectories of arms and legs during walking at matched speed are shown for one control and one stroke subject in Fig. 1, B and C, respectively. Stable unperturbed walking patterns were observed in both cases. All subjects walked slower on the treadmill compared with overground, but the comfortable treadmill speed of stroke subjects (0.72 ± 0.20 m/s) was similar to the slower (matched) treadmill speed of healthy controls (0.83 ± 0.20 m/s). Compared with controls, poststroke individuals walked with shorter swing length and duration bilaterally and less arm swing on the paretic side (Table 2). Stroke subjects increased their gait speed by ~18% (to 0.85 ± 0.20 m/s) when walking faster, which was still significantly lower than the comfortable speed of controls (1.08 ± 0.14 m/s, \( P < 0.02 \)). The comfortable (faster) gait speed in controls was 27% faster than their matched speed. Since the speed difference between conditions was different between groups, the effect of gait speed on gait stability was analyzed separately for each group. The force of perturbation was similar for both conditions and both groups: 38.6 ± 7.6 N for stroke subjects (matched), 38.7 ± 6.8 N (faster), 35.0 ± 5.7 N for the control group (matched), and 39.4 ± 7.78N (faster; \( P > 0.05 \) for all). The perturbation resulted in a small decrease in gait speed (control: matched = 3.6 ± 2.6%, faster = 4.6 ± 2.6%; stroke: matched = 6.1 ± 6.7%, faster = 5.8 ± 7.3% ) that did not differ between groups.

Recovery from Perturbation at Matched Speeds

In the short-term after perturbation at matched speed, stroke and control subjects used different strategies. Poststroke individuals used a leg elevation strategy in response to perturbation in 5% of cases, compared with 49% in controls (\( t_{18} = 4.51, \ P < 0.001 \)). In contrast, a lowering strategy was used in over half the cases in poststroke individuals (58%) compared with only 5% in controls (\( t_{18} = -3.75, \ P < 0.002 \); Fig. 2). Subjects in both groups were equally likely to use the combined strategy (stroke: 37%, healthy: 46%).

Swing length (Fig. 3, A and B) and time (Fig. 3, C and D) were reduced for both groups compared with baseline in the perturbed and first postperturbed steps. This reduction depended on leg strategy, such that the largest decrease occurred for the lowering strategy in both the perturbed (nonparetic/dominant leg) and nonperturbed swing (perturbed swing:.
Table 2. Characteristics of nonperturbed treadmill walking for subjects with stroke and control subjects

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Comfortable Speed (Machined)</th>
<th>Arm</th>
<th>Arm</th>
<th>Arm</th>
<th>Arm</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Speed, m/s</td>
<td>Swing time, s</td>
<td>Swing amplitude, mm</td>
<td>Treadmill length, mm</td>
<td>Time, s</td>
</tr>
<tr>
<td>Stroke (n = 10)</td>
<td>0.80</td>
<td>0.83</td>
<td>0.81</td>
<td>0.80</td>
<td>0.80</td>
</tr>
<tr>
<td>Controls (n = 10)</td>
<td>0.63</td>
<td>0.72</td>
<td>0.70</td>
<td>0.63</td>
<td>0.63</td>
</tr>
</tbody>
</table>

Effect of Gait Speed on Recovery from Perturbation

Compared to comfortable walking speed, gait speed increased in four stroke subjects by >20% (21–26%), while six subjects increased gait speed by <20% (9–16%). For the control group, matched gait speed was 76% of their comfortable speed (60–88%; Fig. 5A; absolute gait speed values are noted in Table 2).

Among poststroke individuals, walking faster did not influence the leg strategies used (Fig. 2). In addition, speed did not influence double-support recovery time (Fig. 5B), swing length and time of the first two steps (Fig. 5, C and D, time not shown; P > 0.05), phase shifts, or interlimb coupling (phase shift differences). In contrast, in the control group the matched (slower) speed condition was associated with more leg elevation responses (49% compared with 5%) and less leg lowering responses (5% compared with 35%; Fig. 2) compared with comfortable speed. In controls, swing length and time decreased with the increase in gait speed in both the perturbed and postperturbed steps (Fig. 5, C and D, time not shown; \( \chi^2 > 9.68, P < 0.003 \) for all). However, the longer term recovery of steady-state walking was similar across the two speed conditions (Fig. 5B).

Faster walkers in the stroke group had tighter arm-leg coupling of the nonparetic arm-leg pair (\( \chi^2 = 13.6, P < 0.001 \)) but not of the paretic arm-leg pair. Leg-leg coupling was also better for faster walkers in this group (\( \chi^2 = 5.62, P < 0.02 \)). In controls, arm-leg movements were more coupled on both sides for faster-walking subjects (\( \chi^2 > 12.5, P < 0.001 \) for both sides). In addition, arm-arm coupling was greater in faster walkers (\( \chi^2 = 15.52, P < 0.01 \)). However, there were no differences in interlimb coordination between speed conditions in either group.
Gait stability in healthy older adults has been assessed using a variety of kinematic measures (Hamacher et al. 2011), some of which have also been applied in subjects with stroke (Balasubramanian et al. 2009). To the best of our knowledge, ours is the first study to investigate differences in gait stability between stroke and healthy subjects following a trip perturbation. The evaluation of the return to steady-state walking following a leg perturbation undertaken in this study is a direct way to assess the stability properties of gait in poststroke and healthy older adults. Although none of the subjects in this study fell, short- and longer term differences in recovery from perturbation between groups showed that stability of steady-state walking was reduced in high-functioning poststroke individuals. Thus an equally powerful perturbation in both groups generated a stronger, global response in poststroke individuals in terms of strategy and timing of gait rhythm, which was also more demanding in terms of the return to steady-state walking.

The sample of poststroke individuals in our study was high-functioning and relatively homogeneous in terms of functioning and relatively homogeneous in terms of function, but subjects differed in terms of lesion type and location (Table 2). As we were unable to control for the effect of lesion type and location due to the small size of the sample, we used baseline walking characteristics of each subject as their own control when estimating recovery of steady-state walking following perturbation.

**Stroke Affects Gait Stability in Response to an Unexpected Perturbation**

Our results demonstrated that immediately following a perturbation, poststroke individuals were less likely to use a leg elevation and more likely to use a leg lowering strategy, while healthy adults used elevation most often. Although an elevation response may require simpler control and less energy
(Forner Cordero et al. 2005; Krasovsky et al. 2012; de Boer et al. 2010), it may be more difficult to perform by poststroke subjects, who adopt a safer alternative (Eng et al. 1994). Specifically, the perturbation occurred during swing of the nonparetic leg such that maintaining the perturbed leg in the air would require subjects to support their body weight for a longer period of time on their weaker, paretic leg. Stroke subjects most often lowered the perturbed leg and minimized the time spent on the hemiparetic leg at the cost of a prolonged disruption in subsequent steps (Fig. 3). The importance of the nonperturbed leg in response to a trip perturbation was previously shown in young and older adults when performing a leg elevation following a trip (Pijnappels et al. 2005). Among older adults, those who generated adequate joint moments in the support limb did not fall as a result of a trip perturbation. In poststroke individuals, the ability to perform an elevation strategy may also depend on factors related to the paretic leg, such as muscle strength and reaction time (Pijnappels et al. 2008; van den Bogert et al. 2002). The relative contribution of these factors needs to be determined in future studies.

Aside from the difference in leg strategy selection, poststroke individuals had larger residual longer term phase shifts in all four limbs (Fig. 4B). Phase resetting of gait patterns during fictive locomotion has been demonstrated in turtles and cats following electrical stimulation of muscle afferents or mechanical perturbations (Conway et al. 1987; Lennard 1985; Saltiel and Rossignol 2004), suggesting that perturbations are capable of causing a resetting of central timing mechanisms controlling multiple limbs (Hultborn et al. 1998). We have shown that resetting is possible in humans following a mechanical perturbation. Indeed, the differences in phase resetting between groups in this study are assumed not to arise from processing of afferent input from the nonparetic leg, since in high-functioning poststroke individuals, the ability to process afferent input from the nonparetic leg remains intact, at least from the tibial nerve (Kloter et al. 2011). Instead, interlimb phase shift differences between poststroke individuals and controls in our study may stem from deficits in control from descending motor pathways. In the cat, overcoming an obstacle in the path involves increased activation of pyramidal tract
Fig. 4. Recovery of steady-state walking and interlimb coupling following perturbation. For A–F, error bars mark ± 1SD; *p < 0.05, **p < 0.01. A: recovery of double-support duration (measured in gait cycles) according to group (control, stroke) and strategy (lowering, combined, elevation). B: phase shift in the perturbed leg following perturbation according to group and strategy. C–F: difference between phase shifts (measured in degrees) in arm-leg pairs on the nonparetic/dominant side (C) and the paretic/non-dominant side (D), leg-leg pair (E), and arm-arm pair (F). Limb pair analyzed in A–F shown by lighter limbs on stick figures.
neurons during elevation of the leg (Drew 1988). Supraspinal motor centers may play a role in gait modifications in humans as well, possibly by changing the supraspinal drive to lower (e.g., spinal) levels (Varraine et al. 2000). In our study, the ability to generate the appropriate amount of phase shift in response to a perturbation was disrupted in poststroke individuals. The disruption persisted across the three response strategies for the legs and all limbs. This may have functional consequences for gait stability in poststroke individuals, since there may be an “optimal” amount of phase resetting which is required to prevent falling (Yamasaki et al. 2003).

Gait stability deficits were previously demonstrated in poststroke individuals during unperturbed gait. Specifically, spatiotemporal gait parameters (step length, stride time) were shown to be more variable in poststroke individuals compared with controls (Balasubramanian et al. 2009). This suggests that in these subjects the motor system exhibits increased fluctuations with respect to a stable state attractor (Schöner and Kelso 1988). An additional expectation is that in response to perturbation an unstable system would be unable to return to the attractor state (in this case, a fall would occur) or would take longer to recover steady-state patterns (critical slowing down, Scholz et al. 1987). In our study, the latter frequently occurred in poststroke individuals who took longer to recover baseline walking patterns. These results contrast with those obtained using obstacle circumvention paradigms. Poststroke individuals were less likely to use a lowering strategy when an expected obstacle appeared in the walking path compared with controls (Den Otter et al. 2005). To avoid leg contact with an expected obstacle, stroke subjects lifted their leg over the obstacle with higher toe clearance (Said et al. 2001). This difference highlights the task-specific nature of strategy selection in recovery from perturbation in poststroke subjects. A less-destabilizing strategy can be chosen if the obstacle is expected. However, if the obstacle is unexpected, the motor system is challenged to select a suboptimal strategy, favoring balance over efficiency of recovery from perturbation (Nonnekes et al. 2010).

**Gait Speed, Arm-Leg Coupling, and Gait Stability Following Unexpected Perturbation**

The decreased ability of poststroke individuals to maintain steady-state walking following perturbation remained unchanged following a speed increase of <20%. Indeed, this speed increase was neither associated with better stability nor with adverse effects. The modest speed increase implemented in this study was motivated by safety considerations and met the requirements of common training protocols (Pohl et al. 2002). Whether larger differences in speed could have resulted in a larger effect on gait stability remains to be determined. However, when walking faster, functional capacity (CM, FGA) in poststroke individuals was related to the ability to recover steady-state walking, suggesting that the faster walking condition brought out impairments of balance and walking that may impact on recovery from perturbation and might otherwise remain undetected. The leg response strategies of poststroke individuals at both walking speeds were similar to those of controls walking at their comfortable speed but not to responses that occurred when control subjects walked at slower speeds. This implies that the deficit in gait stability is not related to the absolute value of walking speed. Unlike subjects in the stroke group, when controls walked at slow speed they were able to use the leg elevation strategy more frequently.
This is in accordance with the suggestion that slower gait in older adults may be a protective strategy by which stability can be increased to prevent falls (Maki 1997). In contrast, poststroke individuals did not modify their responses when changing gait speed. This suggests that slower walking in stroke subjects may be related to actual and perceived endurance and balance limitations (Lamontagne and Fung 2004). These limitations refer to both comfortable and faster gait speeds. Indeed, poststroke individuals were not asked to walk slower than their comfortable gait speed; under such conditions, better gait stability may have been observed. Alternatively, the need to constrain gait to a noncomfortable (slower or faster) speed may contribute to the deficit in stability. Interestingly, in both groups only when walking at the noncomfortable speed (slower for controls, faster for stroke) an association was observed between overground walking performance and recovery of steady-state walking.

Stroke subjects had larger arm-leg phase shift differences compared with controls on the paretic but not on the nonparetic side (Fig. 4). In controls, a coordinated response to perturbation consists of similar phase shifts in all limbs, resulting in the recovery of preperturbed coordination patterns (Feldman et al. 2011). In the case of poststroke individuals, arm-leg phase shift differences on the paretic side were greater regardless of the initial response strategy. These results contrast with those of Stephenson et al. (2009) who found no differences in interlimb coordination between high-functioning stroke subjects and controls when walking on a treadmill without perturbations. In that study, coordinated walking may have been achieved using various compensations (Kim and Eng 2004) such that the deficit in interlimb coordination may be revealed only when the system was challenged by an unexpected perturbation.

Faster walkers had smaller phase shift differences on both sides in controls and on the nonparetic side in stroke subjects. In addition, poststroke subjects improved nonparetic arm-leg and bilateral leg-leg coupling with gait speed. This is consistent with previous studies showing that intersegmental coupling increased with gait speed during nonperturbed walking in healthy adults (van Emmerik and Wagenaar 1996) and in poststroke individuals (Wagenaar and Beek 1992). Similarly, interjoint (Daly et al. 2011) and arm-leg (Kwakkel and Wagenaar 2002) coordination improved with postrehabilitation gait speed increases (Hollands et al. 2011). Increased interlimb coupling may result from optimized central drive to locomotor pattern generators (Ferris et al. 2006). In our sample, this improvement was limited to the nonparetic side while arm-leg coordination on the paretic side remained lower across walking speeds. Thus stroke-related brain damage may lead to impairments in the modulation of arm-leg coordination with gait speed that are specific to the paretic side. During nonperturbed walking, modulation of arm-leg coordination with gait speed was recently shown to be impaired in individuals with spinal cord injury (Tester et al. 2012), supporting the notion of centralized control of interlimb coordination. Our results further suggest that different mechanisms of control may be associated with gait stability and interlimb coordination. Unlike arm-leg coordination, gait stability did not vary with gait speed in either controls or poststroke individuals. Thus, the increased coordination associated with faster gait speed was not functionally relevant to recovery from perturbation.

Clinical Significance

Our study illustrates the benefits of using basic knowledge of control processes underlying unimpaired gait, regulation of postperturbation response patterns, translation of body equilibrium in the environment (gait stability), and interlimb coordination, to the analysis of deficits of these processes in stroke subjects. Achieving community ambulation is an important goal in poststroke rehabilitation (Robinson et al. 2011) that includes the ability to overcome unexpected perturbations. However, even well-recovered community ambulators have difficulties in performing this complex task. Stability deficits were identified in a challenging paradigm that forced the paretic leg to perform a compensatory step. Thus assessment of reactive responses in stroke may provide additional information about walking performance in everyday life (Marigold and Misiaszek 2009), specifically in tasks requiring compensatory stepping of the paretic leg (Pelton et al. 2010). The presence of deficits in gait stability among well-recovered subjects supports the inclusion of unexpected perturbations in gait rehabilitation programs for this subgroup of stroke patients (Marigold and Misiaszek 2009; Nonnikes et al. 2010). Future studies are needed to investigate the relationship between reactive mechanisms of gait stability and fall risk.

Both interlimb coordination and gait stability represent important constructs to be measured during gait rehabilitation. However, our results suggest that although faster walking may be associated with better interlimb coordination, better coordination is not necessarily associated with better gait stability and should be carefully interpreted.

Limitations

We used a custom-built self-paced treadmill to study functional walking patterns and used metronome pacing. Although the metronome pacing may have had an effect on movement patterns, previous studies have shown that this effect would have been similar in both groups (Roerdink et al. 2007). In addition, short- and long-lasting phase shifts in our study occurred despite the absence of changes in the phase of the metronome pacing, showing that synchronizing gait with the metronome was not a priority in reactions to perturbations. The modest speed differences may be considered as a limitation, and larger speeds may be used in the future. Finally, this study involved a relatively small sample (n = 20) that consisted only of male subjects. Future studies should determine whether gender affects the response to perturbation.

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS


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