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Post-stroke deficits in the step-by-step control of paretic step width

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ABSTRACT

Background: Humans partially maintain gait stability by actively controlling step width based on the dynamic state of the pelvis – hereby defined as the “dynamics-dependent control of step width”. Following a stroke, deficits in the accurate control of paretic leg motion may prevent use of this stabilization strategy.

Research Question: Do chronic stroke survivors exhibit paretic-side deficits in the dynamics-dependent control of step width?

Methods: Twenty chronic stroke survivors participated in this cross-sectional study, walking on a treadmill at their self-selected (0.57 ± 0.25 m/s; mean \pm s.d.) and fastest-comfortable (0.81 ± 0.30 m/s) speeds. To quantify the dynamics-dependent control of step width, we calculated the proportion of the step-by-step variance in step width that could be predicted from mediolateral pelvis dynamics, and used partial correlations to differentiate the relative effects of pelvis displacement and velocity. Secondly, we calculated the mean and standard deviation of more traditional gait metrics: step width; lateral foot placement; and mediolateral margin of stability (MoS). We used repeated measures ANOVA to test for significant effects of leg (paretic vs. non-paretic) and speed (self-selected vs. fastest-comfortable) on these measures.

Results: Relative to non-paretic steps, paretic steps exhibited a weaker ($p \leq 0.005$) link between step width and pelvis dynamics, attributable to a decreased partial correlation between step width and pelvis displacement ($p \leq 0.001$). Paretic steps were also placed more laterally ($p < 0.0001$), with a larger ($p < 0.0001$) and more variable ($p = 0.003$) MoS. The only effect of faster walking speeds was a narrower step width ($p < 0.0001$).

Significance: Pelvis displacement was less tightly linked to step width for paretic steps than for non-paretic steps, indicating a decrease in the step-by-step reactive control normally used to ensure mediolateral stability. Instead, stroke survivors placed their paretic leg farther laterally to ensure a larger MoS, behavior consistent with a greater reliance on a generalized feed-forward gait stabilization strategy.

1. Introduction

Many chronic stroke survivors are at high risk for losses of balance while walking [1]. As in all populations, such losses of balance can be caused by external mechanical perturbations (e.g. slips, trips, pushes). However, stroke survivors exhibit an additional increased risk of “intrinsic falls” that are not caused by environmental factors, but are instead attributed to individual-specific impairments in controlling balance [2]. While perhaps surprising, many falls in the community are due to self-generated movement errors (e.g. incorrect weight shifting) that accompany poor balance, rather than to external perturbations [3]. In order to develop new rehabilitation methods to reduce the risk of these intrinsic falls, we must first understand how the strategies used to ensure stability during unperturbed walking change following a stroke.

While bipedal gait stability requires the coordination of many degrees of freedom, one relatively simple stabilization strategy consistently used by neurologically-intact controls is the step-by-step adjustment of mediolateral foot placement, as recently reviewed by Bruijn and van Dieën [4]. Essentially, humans tend to adjust their swing leg trajectory to ensure a “dynamically-appropriate” step width – one that is not so narrow as to risk a lateral loss of balance [5], but not so wide as to cause excessive energy losses [6]. This behavior can be quantified mathematically by relating pelvis displacement and velocity (hereby termed ‘pelvis dynamics’) to step width on a step-by-step basis [7,8]. However, it is presently unclear if this gait stabilization strategy is still present following a stroke.

The accuracy of paretic leg movements is reduced in many chronic stroke survivors [9], raising the question of whether an inability to

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accurately adjust paretic step width contributes to the gait stability deficits common in this population. While mediolateral motion of the body is often altered during post-stroke gait [10], it is not clear whether the typical dynamics-dependent control of step width is diminished. Such deficits may contribute to the post-stroke preference for slower walking speeds, as the need to rapidly (and accurately) reposition the swing leg appears reduced at slower speeds [8].

The purpose of this study was to investigate the dynamics-dependent control of step width among chronic stroke survivors using a recently developed metric [8]. Unlike more traditional measures of mediolateral motion during gait (e.g. step width; mediolateral margin of stability (MoS)), this metric can directly quantify the relationship between pelvis dynamics and step width throughout a step, and may thus provide unique insight into post-stroke deficits related to altered gait balance. We quantified the relationship between mediolateral pelvis dynamics and step width for paretic and non-paretic steps, while participants walked at self-selected and fastest-comfortable speeds. We hypothesized that the link between pelvis dynamics and step width would be weaker for paretic than non-paretic steps, and would become stronger at faster walking speeds. Secondly, we investigated whether these potential differences in the step-by-step adjustments of step width were accompanied by changes in more traditional gait measures.

2. Methods

Twenty chronic stroke survivors participated in this experiment, with general characteristics listed in Table 1. This sample size was based on prior work [8], in which a sample of 12 controls was sufficient to detect a significant effect of speed on the dynamics-dependent control of step width. All participants exhibited lower extremity hemiparesis (Fugl-Meyer motor score < 34), but could walk independently on a treadmill for at least 3-minutes. Written informed consent was obtained from each participant using a form approved by the Medical University of South Carolina Institutional Review Board and consistent with the Declaration of Helsinki.

Participants walked on a treadmill (Bertec; Columbus, Ohio), and verbally identified their self-selected speed (speed they use to walk around the house) and fastest-comfortable speed (fastest speed that still feels safe). Participants then performed a 3-minute trial at each speed, in randomized order. LED markers (PhaseSpace; San Leandro, California) were placed on the pelvis and legs, with the present analyses focused on markers on the sacrum, heels, and lateral aspect of the midfoot. Marker locations were sampled at 120 Hz, and low-pass filtered at 10 Hz.

We defined the step start as the time when the ipsilateral heel marker velocity changed from posterior to anterior, and the step end as the time when the contralateral heel marker velocity changed from posterior to anterior [11]. For each step, we calculated step width, defined as the mediolateral distance from the ipsilateral heel marker at the end of the step to the contralateral heel marker at the start of the step. We also calculated lateral foot placement location, defined as the

mediolateral distance from the ipsilateral heel marker to the sacrum at the end of the step. Throughout each step, we quantified pelvis displacement - defined as the mediolateral distance from the sacrum to the heel of the stance foot, and pelvis velocity - defined as the mediolateral velocity of the sacrum marker.

The primary focus of this study was on the dynamics-dependent control of step width, as in a prior study focused on young, neurologically-intact control participants [8]. Here, we compared the dynamics-dependent control of step width for steps taken with the paretic and non-paretic legs. To quantify the extent to which step-by-step variation in step width was related to ongoing pelvis dynamics, for each participant and trial we performed a linear regression in which step width was predicted from a linear combination of pelvis displacement and pelvis velocity. The strength of this relationship was quantified as the R^2 value of the regression. We also calculated two partial correlation measures to provide insight into whether the step-by-step variation in step width was dominated by fluctuations in pelvis displacement or velocity. Specifically, we calculated the partial correlation between step width and pelvis displacement (ρ_{disp}), accounting for pelvis velocity. We also calculated the partial correlation between step width and pelvis velocity (ρ_{vel}), accounting for pelvis displacement. We calculated each of these measures (R^2 , ρ_{disp} , and ρ_{vel}) throughout the course of a step as the dynamic state of the pelvis changed. However, our statistical analyses will focus on values at the start and end of the step [8].

Secondarily, we calculated several more commonly presented metrics of mediolateral motion during gait, quantified for steps taken with the paretic and non-paretic legs. For each participant and trial, we calculated mean step width, mean lateral foot placement location, and mean mediolateral MoS. Mediolateral MoS was calculated using established methods [8,12], as we first estimated the mediolateral position of the extrapolated center of mass (x_{CoM}) from sacrum position (x_{sacrum}) and velocity (v_{sacrum}) [13], normalizing sacrum velocity by the natural frequency (ω_0) of a pendulum 1.34 times the length of the participant's leg [12]:

$$x_{CoM} = x_{sacrum} + v_{sacrum}/\omega_0 \quad (1)$$

We then estimated the mediolateral MoS for each stance phase by calculating the minimum difference between the extrapolated center of mass location and the mediolateral position of the lateral midfoot. For each of these metrics (step width, lateral foot placement location, and mediolateral MoS), we quantified variability by calculating the metric's standard deviation within each trial. We chose to use intrasubject standard deviations as our measure of variability [14] due to our primary interest in the magnitude of the step-by-step changes in the quantified variables. While coefficients of variation (CV) have also been used to quantify gait variability, CV values can become artificially inflated when a metric's mean value approaches zero [15,16] – as was the case in some participants for several metrics quantified in this study.

We performed a series of repeated measures ANOVA with interactions to determine whether our primary outcome variables (step start R^2 ; step end R^2 ; step start ρ_{disp} ; step end ρ_{disp} ; step start ρ_{vel} ; step end ρ_{vel}) or secondary outcome variables (mean step width; step width variability; mean lateral foot placement; lateral foot placement variability; mean MoS; MoS variability) were significantly influenced by leg side (paretic vs. non-paretic) or walking speed (self-selected vs. fastest-comfortable). We interpreted p-values less than 0.05 as significant.

3. Results

The overall relationship between the dynamic state of the pelvis and step width was generally weaker for paretic steps than non-paretic steps. This relationship is illustrated over the course of a step in terms of R^2 magnitude (Fig. 1A), ρ_{disp} magnitude (Fig. 1B), and ρ_{vel} magnitude (Fig. 1C). While substantial variability was observed across individual post-stroke participants, both R^2 magnitude and ρ_{disp} magnitude were lower for paretic steps, based on comparisons made at either the start

Table 1

Participant characteristics. All non-categorical values are presented in terms of means \pm intersubject standard deviations.

Characteristic	Value(s)
Gender	6 F / 14 M
Paretic leg	3 L / 17 R
Age (yrs)	59 \pm 10
Time since stroke (mos)	51 \pm 46
Height (cm)	174 \pm 12
Mass (kg)	85 \pm 14
Leg length (cm)	90 \pm 7
Self-selected speed (m/s)	0.57 \pm 0.25
Fastest-comfortable speed (m/s)	0.81 \pm 0.30
Fugl-Meyer motor score	25 \pm 4

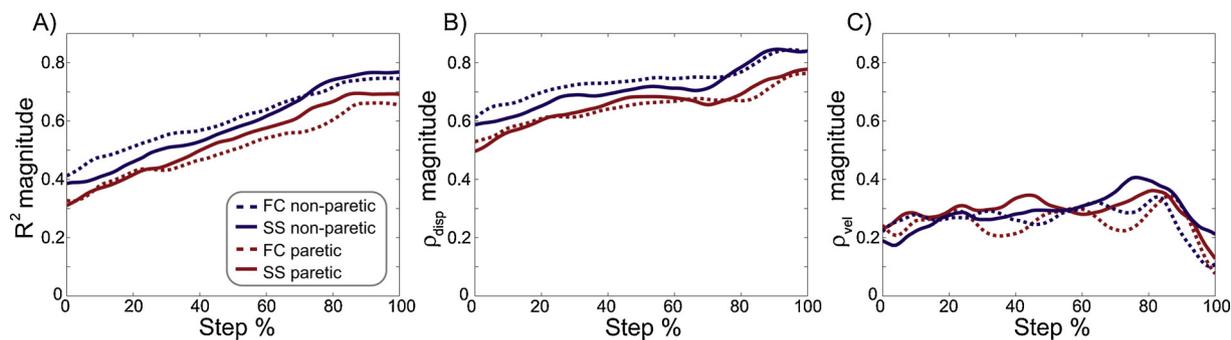


Fig. 1. The relationship between step width and pelvis displacement/velocity varied over the course of a step. This relationship was quantified in terms of R^2 magnitude (A), ρ_{disp} magnitude (B), and ρ_{vel} magnitude (C). Mean values for each of these metrics are plotted over the course of a step for the parietic and non-parietic legs during self-selected (SS) and fastest-comfortable (FC) walking conditions. Indications of variability are not illustrated due to excessive overlap between conditions. Measures of variability at the start and end of the step are instead provided in Table 2.

Table 2

Effects of leg side and walking speed on metrics of mediolateral balance control. Values are presented as means \pm intersubject standard deviations. Significant effects are indicated with asterisks (*) and italics.

Metric	Self-selected		Fastest-comfortable		p-values		
	Paretic step	Non-paretic step	Paretic step	Non-paretic step	Leg side	Walking speed	Inter-action
R^2	0.31 \pm 0.15	0.39 \pm 0.12	0.32 \pm 0.13	0.41 \pm 0.13	0.005*	0.43	0.86
step start							
R^2	0.69 \pm 0.15	0.77 \pm 0.06	0.65 \pm 0.16	0.74 \pm 0.11	0.0004*	0.18	0.76
step end							
ρ_{disp}	0.50 \pm 0.15	0.59 \pm 0.10	0.53 \pm 0.13	0.61 \pm 0.12	0.001*	0.28	0.85
step start							
ρ_{disp}	0.78 \pm 0.13	0.84 \pm 0.05	0.76 \pm 0.11	0.84 \pm 0.08	0.0005*	0.60	0.78
step end							
ρ_{vel}	0.23 \pm 0.19	0.19 \pm 0.24	0.24 \pm 0.19	0.22 \pm 0.16	0.46	0.62	0.82
step start							
ρ_{vel}	0.13 \pm 0.19	0.21 \pm 0.20	0.08 \pm 0.17	0.11 \pm 0.24	0.14	0.054	0.52
step end							
Step width (cm)	16.8 \pm 2.9	16.8 \pm 2.9	15.9 \pm 2.8	15.9 \pm 2.8	0.99	< 0.0001*	0.99
Step width variability (cm)	2.1 \pm 0.7	2.1 \pm 0.6	1.9 \pm 0.5	2.1 \pm 0.6	0.58	0.22	0.34
Lateral foot placement (cm)	9.0 \pm 1.8	5.6 \pm 3.3	8.9 \pm 1.9	5.8 \pm 2.7	< 0.0001*	0.88	0.82
Lateral foot placement variability (cm)	1.3 \pm 0.3	1.2 \pm 0.4	1.2 \pm 0.3	1.2 \pm 0.3	0.09	0.84	0.76
Average MoS (cm)	8.8 \pm 3.0	5.4 \pm 2.0	8.8 \pm 3.4	5.4 \pm 2.0	< 0.0001*	0.96	0.95
MoS variability (cm)	1.7 \pm 0.7	1.4 \pm 0.3	1.6 \pm 0.4	1.4 \pm 0.3	0.003*	0.83	0.42

($p \leq 0.005$) or end ($p \leq 0.0005$) of a step (Table 2). In contrast, ρ_{vel} did not differ between the legs at either tested time point, with values substantially lower than those observed for ρ_{disp} (Table 2). None of the metrics quantifying the relationship between the dynamic state of the pelvis and step width were affected by walking speed, or by an interaction between walking speed and leg side (Table 2).

The secondary, more traditional metrics of mediolateral gait motion were variably affected by leg side and walking speed. All values and statistical comparisons are provided in Table 2, with specific comparisons highlighted here. While mean step width was identical for parietic and non-parietic steps – as required for forward straight-line walking – the foot was placed farther laterally from the pelvis for steps taken with the parietic leg ($p < 0.0001$). Parietic steps were followed by a larger ($p < 0.0001$) and more variable ($p = 0.003$) MoS during the subsequent stance phase, relative to non-parietic steps. At the faster walking speed, mean step width decreased ($p < 0.0001$) but none of the other metrics changed significantly. None of the tested metrics were affected by an interaction between walking speed and leg side.

4. Discussion

The dynamics-dependent control of step width differed between steps taken with the parietic and non-parietic legs. Supporting our hypothesis, the link between pelvis dynamics and step width was weaker for parietic steps. Asymmetries in our secondary measures of

mediolateral motion during gait may reflect the use of an alternative gait stabilization strategy that does not depend on accurate step-by-step control. Contradicting our hypothesis, the dynamics-dependent control of step width did not change at faster walking speeds, despite the use of narrower mean step widths.

The observed gait asymmetries are consistent with reduced dynamics-dependent control of step width with the parietic leg to maintain walking balance. Throughout a step, pelvis displacement was less tightly linked to the upcoming step width for parietic steps than non-parietic steps. As a result, the relative locations of the pelvis and parietic foot were more variable at the end of a parietic step, contributing to increased variability in the mediolateral MoS during the subsequent parietic stance phase. This greater MoS variability could presumably increase the risk of a lateral loss of balance [5]. However, none of the participants actually fell, suggesting their use of a different strategy to ensure mediolateral balance. We propose that the observed behavior reflects a post-stroke shift from reactive balance control to more feed-forward control, as previously suggested by Wu and colleagues to occur in patients with neurological injuries [17]. Here, “reactive balance control” is defined as the step-specific adjustments in step width that occur in response to natural step-by-step variation in body dynamics (which can itself be considered a series of internal mechanical perturbations [18]). In contrast, we define “feed-forward control” as a more general strategy that is present in every step to help ensure mediolateral balance. The more lateral foot placement and larger MoS observed for

paretic steps can be considered an example of just such a feed-forward strategy, as this mean behavior occurs for every step and would be expected to reduce the risk of an immediate loss of balance toward the paretic leg.

While the metrics of primary interest (R^2 , ρ_{disp} , ρ_{vel}) have not previously been quantified post-stroke, our more traditional measures of mediolateral motion during gait are only partially consistent with prior work. Specifically, the findings of the present study are supported by previous reports of more lateral foot placement of the paretic leg [19,20], as well as an increase in MoS variability for paretic steps [21]. However, our observed larger MoS for paretic steps conflicts with prior reports of no difference between legs [21], or larger non-paretic than paretic MoS values (albeit not at the time of minimal MoS) [22]. This discrepancy may be due to a higher level of function among prior studies' participants (self-selected speeds of 0.9–1.0 m/s, compared to ~0.6 m/s here).

Contrary to our expectations, walking speed did not affect the step-by-step adjustments of step width. Despite a clear increase in speed (of ~0.2 m/s) and an accompanying decrease in mean step width from the self-selected to fastest-comfortable condition, we observed no increase in step start R^2 or ρ_{disp} , as is present in neurologically-intact controls [8]. Perhaps this inability to increase the dynamics-dependent control of step width under more challenging conditions is one of the reasons for the slower gait speeds often preferred by chronic stroke survivors. The lack of an effect of walking speed on average MoS and MoS variability is consistent with a previous comparison of self-selected and faster speeds [21].

While the present results revealed a paretic-side deficit in the control of step width, they do not provide insight into other potential causes of post-stroke losses of balance. Most notably, we did not investigate the response to external perturbations, which could clearly contribute to falls. Additionally, our focus on gait kinematics does not allow us to distinguish between active (muscle-driven) and passive contributions to the stabilizing adjustments in step width. While speculative, we suspect that the reduced dynamics-dependent control of step width for paretic steps can be attributed to altered active control. This suggestion is based on prior modeling work that found mediolateral gait stabilization to require active control, while anteroposterior stability can be achieved passively [23]. Further supporting this idea, our prior work found that stroke survivors with balance deficits exhibit a reduced ability to appropriately activate their paretic leg gluteus medius to control swing leg motion during gait [20]. Finally, we neglected gait motion in the anteroposterior and vertical directions, instead focusing on motion in the frontal plane that has previously been cited as particularly important for functional mobility in clinical populations [24]. While multi-planar interactions are clearly present during human walking [25], recent work found that step-by-step predictions of step width are dominated by mediolateral motion of the pelvis, with only a negligible effect of anteroposterior and vertical pelvis dynamics [7].

The mechanism underlying the altered control of paretic step width is not entirely clear. One promising explanation is a reduced ability to accurately control mediolateral paretic leg motion during the swing phase, potentially causing stepping inaccuracy [9,26]. In turn, this reduced control accuracy could be due to multiple factors, including: weakness of the muscles used to control hip abduction [20]; proprioceptive deficits preventing accurate detection of where the swing leg is in space [27]; or an inability to follow the normal swing leg path due to altered joint ranges of motion from spasticity, contractures, or foot drop. None of these potential deficits were measured in the present study. Alternatively, stroke survivors with a fear of falling may habitually prefer to place their paretic foot far laterally in every step, reducing the risk of a loss of balance toward the paretic leg, but increasing the resultant mechanical and metabolic demands of walking [6,28]. Future work should seek to identify individual patients who exhibit a deficit in the dynamics-dependent control of paretic step

width, and classify these patients in terms of the mechanistic cause of this deficit (e.g. weakness, limited range of motion, etc.). Targeted interventions focused on strengthening appropriate musculature or increasing range of motion could subsequently serve to improve the dynamics-dependent control of step width normally used to ensure a stable gait pattern.

Despite the potential complexity underlying the altered control of paretic step width, a recent study suggests that appropriate paretic step width modulation can be provoked in a subpopulation of stroke survivors [29]. Specifically, this study found that both the paretic and non-paretic legs exhibit similarly scaled step width adjustments following mediolateral mechanical perturbations. Therefore, at least some stroke survivors are able to execute dynamics-dependent control of step width when placed in an appropriate mechanical context. This subpopulation of stroke survivors could potentially benefit from a future intervention involving repeated practice of this behavior, targeting the restoration of a gait pattern in which pelvis dynamics influences step width in every step.

Conflict of interest statement

The authors declare they have no conflicts of interest.

Declarations of interest

None.

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