Title: NEUROMUSCULAR STRATEGIES IN THE PARETIC LEG DURING CURVED WALKING IN INDIVIDUALS POST-STROKE

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Running Head: Curved walking in people with stroke
Reduced flexibility over the neuromotor control of paretic leg muscles may impact the extent to which individuals post-stroke modulate their muscle activity patterns to walk along curved paths. The purpose of this study was to compare lower limb movements and neuromuscular strategies in the paretic leg of individuals with stroke to age-matched controls during curved walking. Participants walked at their preferred walking velocity along four different paths of increasing curvature while lower limb kinematics and muscle activity were recorded. A second group of able-bodied individuals walked along the four paths matching the walking speed of the stroke group. The stroke group showed reduced lower limb joint excursion and disordered modulation of foot pressure during curved walking, accompanied by reduced modulation of muscle activity patterns. In the controls, the postero-medial (medial gastrocnemius and medial hamstring) muscles of the inner leg of the curved showed decreasing EMG amplitude as path curvature increased. Conversely, activity of the postero-lateral musculature of the outer leg was decreased with increasing path curvature. Activity in the tibialis anterior and gluteus medius were also modulated with path curvature. However, in the stroke group, we found reduced modulation of muscle activity in the paretic leg during curved walking. The extent of modulation was also associated with the level of physical impairment due to stroke. The results of this study provide further knowledge about neuromuscular control of locomotor adaptations post-stroke.

**Keywords:** Gait, Turning, Hemiparesis
The ability to turn and change direction safely while walking is an important aspect of functional ambulation. Indeed, up to 45% of all steps taken in everyday walking involve changes in direction (Glaister et al. 2007). Previous research has shown that walking along curved paths is accompanied by a modulation of locomotor patterns normally observed during straight walking, including adaptations in postural coordination and neuromuscular activation patterns (Courtine and Schieppati 2003a; b; Imai et al. 2001; Orendurff et al. 2006; Patla et al. 1999). Temporal and spatial features of bilateral leg movements become asymmetric (Courtine et al. 2006; Orendurff et al. 2006; Patla et al. 1991) and trunk movements must be adapted to maintain equilibrium against the additional medial-lateral instability imposed by turning (Courtine and Schieppati 2003a; Imai et al. 2001; Patla et al. 1999). Characteristic adaptations of muscle activation patterns to walking along a curved path have also been identified (Courtine et al. 2006). In general, as path curvature increases, the activity of the muscles located in the medial compartments of the outer leg of the turn (e.g. medial gastrocnemius) tends to increase while activity of muscles located in the lateral compartments (e.g. lateral gastrocnemius) tends to decrease (Courtine et al. 2006). Conversely, in the inner leg of the turn, muscles in the medial compartments show decreased EMG amplitude while those in the lateral compartments show increased EMG amplitude with increasing path curvature (Courtine et al. 2006). Despite these underlying biomechanical and neuromotor modifications, turning appears effortless, as exemplified by our ability to make abrupt turns without altering the ongoing rhythm of walking (Hase and Stein 1999).
One important factor in maintaining upright balance during turning is medial-lateral stability (Courtine and Schieppati 2003a; Hoogvliet et al. 1997; Imai et al. 2001; Orendurff et al. 2006; Patla et al. 1999). For example, there is a tendency to lean towards the inside leg of the turn in order to produce the centripetal forces and angular rotation required for turning (Courtine and Schieppati 2003a; b; Imai et al. 2001; Orendurff et al. 2006; Patla et al. 1999). The distribution of pressure under the foot needs to shift towards the lateral edge (Hase and Stein 1999; Orendurff et al. 2006) and concomitant with this is modulation in medial and lateral gastrocnemius muscle activity in the inside leg of the turn (Courtine et al. 2006). This is one example of the fine-tuning in neuromuscular control that corresponds with biomechanical needs of the walking task. After stroke, there are several impairments in the neural and biomechanical control of walking that could affect the ability to turn safely. People with stroke have impairments in the regulation of frontal plane (medial-lateral) stability during straight walking (Dettmann et al. 1987; Pai et al. 1994; Turnbull et al. 1996), which may limit turning capacity. Disorders in the sequencing of rotation of axial body segments may also contribute to difficulties in making discrete changes in direction across corners (Hollands et al. 2010; Lamontagne and Fung 2009; Lamontagne et al. 2007), although there is some indication that there may be differences in postural coordination in individuals <1 year post-stroke (Lamontagne and Fung 2009) vs. those who are >2 years post-stroke (Hollands et al. 2010).

In addition to disorders in medial-lateral control postural coordination, disorders in the modulation of lower limb muscle activity may also contribute to the neuromotor control of curved walking in people post-stroke. Locomotor patterns post-stroke are
characterized by reduced or absent muscle activity (Burridge et al. 2001; Hirschberg and Nathanson 1952; Marks and Hirschberg 1958) and disorders in the timing of muscle activity (Burridge et al. 2001; Knutsson and Richards 1979; Lamontagne et al. 2001; Peat et al. 1976; Shiavi et al. 1987). Recent findings have suggested that individuals post-stroke have reduced complexity in the neuromotor control of lower limb muscle activity during gait (Clark et al. 2010). Individuals with more severe impairments due to stroke have less independent control between different muscles groups on the paretic side while the neuromotor control of muscles in the non-paretic leg are similar to that observed in able-bodied controls (Clark et al. 2010). Such reduced flexibility over the neuromotor control of paretic leg muscles may impact the extent to which individuals post-stroke are able to modulate their locomotor patterns to achieve curved walking.

Thus, the purpose of this study was to characterize how locomotor activity of the paretic leg is modulated to walking paths of different curvatures in individuals with stroke compared to able-bodied controls. We hypothesized that individuals with stroke will show limited modulation of muscle activation patterns in the paretic leg with changes in path curvature both for turning towards the paretic and non-paretic side, concomitant with impaired control over the distribution of foot pressure in the medial-lateral plane and lower limb joint kinematics, compared to age-matched controls. Further, we hypothesized that impairments in the modulation of lower limb muscle activity patterns on the paretic side would be associated with the level of physical impairment due to stroke.
METHODS:

Participants

Fourteen individuals (10 males; 4 females) with hemiparesis (3 with left-sided
hemiparesis) due to a stroke were recruited. Their ages ranged from 28 to 70 years
(mean age: 53 years; SD: 11.9 years). The weight of the participants was between 52
and 98 kg (mean: 77.5 kg; SD: 11.5 kg), and the height between 1.5 and 1.9 m (mean:
1.7 m; SD: 0.1 m). Time post-stroke ranged from 2 to 22 years (mean: 8.0 years; SD:
5.9 years). All participants were able to walk a minimum of 10 meters unassisted and
none had any other musculoskeletal or other neurological disease affecting balance or
mobility, or cardiovascular condition for which exercise would be contra-indicated. The
extent of physical impairment due to stroke was assessed by the Chedoke-McMaster
Stroke Assessment (CMSA) impairment inventory for the foot and leg, which scores the
degree of physical impairment on a scale ranging from 1 (flaccid paralysis) to 7 (normal
movement patterns) (Gowland et al. 1993). The CMSA leg score of the stroke group
ranged from 2 to 6 (median: 5) and that for the foot ranged from 1 to 6 (median: 3).

Able-bodied individuals were recruited into 2 control groups: the first control
group (CG1) walked at their preferred comfortable walking speed while the second
control group (CG2) matched their walking speed to that of the stroke group. CG1
included 9 able-bodied individuals (2 males; 7 females) with no history of stroke. Their
ages ranged from 32 to 72 years (mean age: 58 years; SD: 14.7 years). Their weight
was between 52 and 70 kg (mean: 62.7 kg; SD: 6.2 kg), and their height between 1.6
and 1.7 m (mean: 1.7 m; SD: 0.1 m). CG2 included 8 able-bodied participants (1 male,
7 females). Their ages ranged from 34 to 62 years (mean age: 45.1 years; SD: 11.6 years). Their weight was between 48 and 87 kg (mean: 65.1 kg; SD: 12.7 kg) and the height ranged between 1.5 and 1.8 m (mean: 1.6 m; SD: 0.1 m). There were no significant differences in the age of the individuals between the 3 participant groups. All participants gave their written and informed consent before participation in this study. This study was approved by the Behavioral Research Ethics Board at the University of British Columbia.

**Recording Procedures**

All signals were recorded unilaterally from either the paretic leg of the stroke participants or the dominant leg of the controls. Leg dominance was established by having the participants indicate which leg they would use to kick a ball. In the stroke group (SG) and CG1, a twin-axis goniometer (Biometrics, Gwent, UK) was taped over the hip joint to measure flexion/extension and abduction/adduction. Single-axis goniometers were also taped over the knee and ankle joints of the paretic leg to measure sagittal-plane kinematics. Three force sensitive resistors (FSRs, Interlink Electronics Inc., Camarillo, CA) were affixed under the heel, the first metatarsal head (medial aspect of the ball of the foot) and the fifth metatarsal head (lateral aspect of the ball of the foot) of the foot. Surface electromyography (EMG) (Delsys Inc., Boston, MA) was recorded from all participants in the SG, CG1 and CG2. EMG from the following muscles were recorded: tibialis anterior (TA), medial (MG) and lateral (LG) gastrocnemius, rectus femoris (RF), vastus lateralis (VL) and medialis (VM), medial hamstrings (MH) and biceps femoris (BF) and gluteus medius (GM). All signals were
converted on-line to digital form (NI PCI-6031E, National Instruments Corporation, Austin, TX) at 1000 Hz and saved to hard disk with commercially available data acquisition software (EMGWorks 3.1, Delsys Inc., Boston, MA).

**Experimental Protocol**

Participants walked along the following paths marked on the laboratory floor with tape: small circle (length = 3.1 m, radius = 0.5 m, curvature = 2 m), medium circle (length = 6.3 m, radius = 1 m, curvature = 1 m), large circle (length = 12.6 m, radius = 2 m, curvature = 0.5 m) and a straight path (length = 6 m, curvature = 0 m). Participants were monitored during the experiment to ensure that they were following the path (the line outlining the path curvature was maintained between the two feet). Walking speed was recorded by dividing the length of the path traveled (in meters) by the time elapsed (in seconds). Participants walked the circular paths in both the clockwise and the counterclockwise direction. Data were divided according to whether the recorded leg was the *inner leg* (turning towards the paretic side in participants with stroke or turning toward the dominant side in controls) or the *outer leg* (turning toward the non-paretic side in participants with stroke or turning toward the non-dominant side in controls). The paths were randomly presented to minimize any effects due to practice or fatigue. Sufficient rest was allowed between walking trials to minimize fatigue.

Participants in the SG and CG1 walked at their preferred comfortable walking speed. Participants in CG2 walked at the same average speed as the SG for each walking path, receiving feedback after each lap from the experimenter to monitor their
walking speed. Data were only recorded once the appropriate walking speed could be maintained for each path curvature.

**Data Analysis**

Data were analyzed off-line with custom-written routines in Matlab (Mathworks, Natick, MA). Signals from the electrogoniometers and FSRs were low pass filtered at 6 Hz with a 4\textsuperscript{th} order Butterworth filter. The EMG data were high pass filtered at 6 Hz with a 3\textsuperscript{rd} order Butterworth filter, full wave rectified and low pass filtered at 25 Hz. Signals from the FSRs under each foot were summed and used to define the times of foot contact and toe-off. The step cycle was defined as the period between successive foot contact times.

FSR, kinematic, and EMG data of all steps within a walking path were averaged and normalized to 100\% of the step cycle to yield average gait patterns for each participant. For the analysis of joint angular excursions, we divided the step cycle into seven functional phases beginning at heel strike: loading (0-10\% of step cycle), mid-stance (10-30\% of step cycle), terminal stance (30-50\% of step cycle), pre-swing (50-60\% of step cycle), initial swing (60-73\% of step cycle), mid-swing (73-90\% of step cycle) and terminal swing (90-100\% of step cycle). Peak joint angles during each of these phases of the step cycle were computed.

Signals from each of the FSRs during each step were normalized to the average peak FSR amplitude from the straight walking trial for each participant. The peak and
the time to peak of the signal from each of the sensors under the foot were calculated from each walking condition.

EMG amplitude during each step was normalized to the average peak EMG amplitude of each respective muscle from the straight walking trial for each participant. EMG activity from each muscle while walking along the circular paths was ensemble averaged across all participants in CG1 and compared to that of the straight walking trials (i.e. straight vs. large circle, straight vs. medium circle and straight vs. small circle). From these comparisons, we identified 'periods of interests'. These periods of interest were defined by time intervals, lasting at least 10% of the step cycle, where muscle activity during one of the curved paths was significantly different than that of straight walking. This was determined by using two-tailed paired t-tests to compare the EMG amplitude of straight walking vs. one of the curved paths at each of the 100 time points and evaluating the statistical difference between them ($p < 0.05$) (Fig. 1). Because three separate comparisons were used to establish a period of interest (i.e. straight vs. large circle, straight vs. medium circle and straight vs. small circle), the onset of the period was established as the time of the step cycle when at least two of these comparisons were significantly different. These periods of interest for each muscle and for turns to either direction were subsequently used to define the analysis of EMG amplitude in the CG2 and SG. The average rectified EMG amplitude over these periods of interest was calculated from each participant for each muscle and walking path.
Statistical Analysis

All statistical analyses were conducted with a commercially available software package (SPSS Inc, Chicago, IL). A critical alpha value of 0.05 was used to test the significance of all the statistical tests. Trends were defined by $P < 0.10$. When warranted after ANOVA testing, all post hoc pair-wise comparisons using the Bonferroni correction were performed.

A two-way analysis of variance (ANOVA) was used to compare differences in walking speed between CG1, CG2, SG for turns to the paretic side (SG-P), and SG for turns to the non-paretic side (SG-NP), and between the four paths (straight, large circle, medium circle, and small circle). Note that this was the only time in the analysis when data from the control groups were collapsed between turns towards both sides of the body.

To determine changes in lower-limb kinematics with path curvature, Spearman’s rho ($\rho$) correlation coefficients between peak joint angles during specific phases of the step cycle and path curvature were computed for CG1 for both the inner and outer legs. If a statistically significant correlation existed between peak joint angles and path curvature, the data from each participant in the CG1 and SG were fit with a linear equation. The slope of this linear equation was used to define the modulation of joint kinematics across path curvature. Differences in slopes for each joint were compared between SG and CG1 with independent $t$-tests.

Changes in force (normalized to straight walking) under each FSR according to path curvature were determined by computing Spearman’s $\rho$ correlation coefficients
between path curvature and peak force for CG1 for both the inner and outer legs. The
same analysis was used to evaluate the relationship between path curvature and time
to peak force (expressed as a % of step cycle). If a statistically significant correlation
existed between peak force and path curvature and between time to peak force and
path curvature, the data from each participant in CG1 and SG were fit with a linear
equation. The slope of this linear equation was used to represent the modulation of
peak force and time to peak force with path curvature. Differences in slopes of each
FSR were compared between the SG and CG1 with independent t-tests.

Changes in EMG amplitude with path curvature were evaluated by computing
Spearman’s ρ correlation coefficients between the average rectified EMG activity over
the periods of interest (described above) and path curvature from the data from CG1 for
both the inner and outer legs. If there was a statistically significant correlation between
EMG activity and path curvature, the data from each participant in CG1, CG2, and SG
were fit with a linear equation and the slope used to represent the magnitude of EMG
modulation. The magnitude of EMG modulation with path curvature was compared
across CG1, CG2, and the SG using a one-way ANOVA.

Spearman’s rho (ρ) correlation coefficient was also used to determine whether
there was a significant relationship between physical impairment due to stroke (defined
by sum of the foot and leg scores of the CMSA) and the amount of EMG amplitude
modulation due to path curvature. The latter was defined in the SG as the slope of a
linear equation between EMG amplitude and path curvature.
RESULTS

Temporal Gait Parameters

Changes in over-ground walking speed across different path curvatures are illustrated in Figure 2. There was a significant main effect for group ($P < 0.001$). CG1 walked faster than CG2 ($P = 0.000$), SG-P ($P < 0.001$) and SG-NP ($P < 0.001$). There were no significant differences in walking speed between CG2, SG-P and SG-NP ($P = 1.000$ for all 3 comparisons).

There was also a statistically significant main effect for path ($P < 0.001$). Participants walked slower with increasing path curvature: straight vs. large circle ($P < 0.001$), large vs. medium circle ($P < 0.001$), and medium vs. small circle ($P < 0.001$).

There was a significant interaction effect between participant group and walking path on walking speed ($P < 0.001$) suggesting that walking speed was reduced to a greater degree with increasing path curvature for CG1 relative to the other three groups.

Lower Limb Kinematics

Figure 3 illustrates the joint angular excursions of the hip, knee and ankle in the SG and CG1 across the different walking paths. Angular excursions in all joints tended to decrease with increasing path curvature, for both the inner (Fig. 3A) and outer leg (Fig. 3B). In the inner leg, there were significant correlations between path curvature and peak plantarflexion ankle ($\rho = -0.34; P = 0.04$), peak knee flexion during mid-stance ($\rho = -0.46; P = 0.005$), and peak hip extension ($\rho = -0.59; P < 0.001$) (Fig. 3A). In the outer leg, there were significant correlations between path curvature and peak
plantarflexion ankle ($\rho = -0.50; \ P = 0.002$), peak knee flexion during swing ($\rho = -0.41; \ P = 0.01$), and peak hip extension ($\rho = -0.51; \ P = 0.002$) (Fig. 3B). No significant relationships were found between hip abduction/adduction and path curvature.

Although the SG exhibited the same general pattern of joint angular excursion during the walking as seen in CG1, their pattern was executed over a smaller range of motion than the control group (Fig. 3). There were significant differences in the magnitude of modulation between CG1 and SG for peak knee flexion during mid-stance ($\rho = 0.01$) and peak hip extension during pre-swing ($\rho = 0.01$), but no significant differences between the two groups for peak ankle plantarflexion of the inner leg ($\rho = 0.69$). For the outer leg, there were significant differences between the two groups in peak ankle plantarflexion ($\rho = 0.03$), peak knee flexion during mid-swing ($\rho < 0.001$) and peak hip extension ($\rho = 0.007$).

**Foot Pressure Distribution**

Modulation of foot pressure under each foot was apparent with changes in path curvature in the controls (Fig. 4A & B). In the inner leg, peak force under the heel significantly decreased ($\rho = -0.24; \ P = 0.02$), peak force under the medial metatarsal significantly decreased ($\rho = -0.60; \ P < 0.001$), and peak force under the lateral metatarsal significantly increased ($\rho = 0.22; \ P = 0.04$) with increasing path curvature. Also, both time to peak force under the heel ($\rho = 0.30; \ P = 0.003$) and under the lateral metatarsal significantly increased ($\rho = 0.21; \ P = 0.05$) with increasing path curvature (Fig 4A). In the outer leg, peak force under the heel significantly decreased ($\rho = -0.23; \ P = 0.03$), peak force under the medial metatarsal significantly decreased ($\rho = -0.42; \ P <$
0.001), and peak force under the lateral metatarsal significantly decreased ($\rho = -0.61; P < 0.001$) with increasing path curvature. There was also a significant increase in time to peak force under the heel with increasing path curvature ($\rho = 0.27; P < 0.01$) (Fig. 4B).

The SG also showed a reduction in peak force under the medial metatarsal of both the inner and outer leg with increasing path curvature (Fig. 4C & D). There were no significant differences in the magnitude of modulation between the controls and SG for peak force under the medial metatarsal of the inner leg ($P = 0.22$) or outer leg ($P = 0.81$). However, individuals with stroke did not show a corresponding increase in force under the lateral metatarsal with increasing path curvature, as observed in the controls (Fig. 4C vs. 4A and 4D vs. 4B). Instead, there was a reduction in peak force under the lateral metatarsal with increasing path curvature. Accordingly, there was a significant difference between the control and SG in the modulation of peak force under the lateral metatarsal of the inner leg ($P = 0.04$) and the outer leg ($P = 0.01$). There was also a significant difference between the two groups for time to peak force under the lateral metatarsal ($P = 0.002$) suggesting that the peak force occurs later in the stance phase for the control group with increasing path curvature.

Peak force under the heel of the outer leg was also significantly different between the two groups ($P = 0.01$). The control group showed a reduction in the peak force under the heel of the outer leg with increasing path curvature whilst the SG showed an increase.
Adaptation of Muscle Activity to Path Curvature

Ensemble averaged EMG profiles during walking along each path curvature in CG1, CG2, and SG are illustrated in Figures 5, 6, and 7, respectively. The grey shaded bars indicate the periods of interest from which EMG amplitude was quantified in each muscle across all groups. In the inner leg of CG1 (Fig. 5A), there was a significant decrease in EMG amplitude with increasing path curvature for the TA at heel strike ($\rho = -0.38$, $P = 0.02$), MG during stance ($\rho = -0.74$, $P < 0.001$), RF during stance ($\rho = -0.55$, $P < 0.001$), VL during stance ($\rho = -0.54$, $P = 0.001$), VM during stance ($\rho = -0.59$, $P < 0.001$), and MH at end-swing ($\rho = -0.72$, $P < 0.001$). There was a significant increase in GM EMG amplitude during stance with increasing path curvature ($\rho = 0.63$, $P < 0.001$).

In the outer leg of CG1 (Fig. 5B), there was a significant decrease in EMG amplitude with increasing path curvature for the TA at heel strike ($\rho = -0.69$, $P < 0.001$), LG during stance ($\rho = -0.65$, $P < 0.001$), RF during stance ($\rho = -0.49$, $P = 0.002$), VL during stance ($\rho = -0.55$, $P < 0.001$), VM during stance ($\rho = -0.45$, $P = 0.005$), and BF at end-swing ($\rho = -0.72$, $P < 0.001$). Also, GM EMG amplitude during swing significantly increased with increasing path curvature ($\rho = 0.70$, $P < 0.001$).

The ensemble averaged patterns of EMG activity of CG2 and the SG are illustrated in Figs. 6 and 7. It is evident from the comparison between Figs. 5, 6, and 7, that even when controlling for walking speed (CG1 vs. CG2), the SG showed differences in the modulation of EMG activity compared to CG1 or CG2. The following text summarizes the results of inter-group analysis of differences in EMG activity across
the different path conditions (Fig. 8). Note that only muscles whose activity showed a significant correlation to path curvature in CG1 are highlighted (see Methods).

**EMG activity of the inner leg**

Figure 8A illustrates the change in TA EMG amplitude of the inner leg between groups and across path curvatures. TA EMG modulation was significantly different across groups \((P = 0.013)\) and post hoc pair-wise comparisons showed significant differences between the SG vs. CG2 \((P = 0.022)\). The comparison between the SG vs. CG1 approached significance \((P = 0.077)\). In the MG (Fig. 8B), EMG modulation was significantly different across groups \((P = 0.005)\) and post hoc pair-wise comparisons showed significant differences between the SG vs. CG1 \((P = 0.014)\) and the SG vs. CG2 \((P = 0.023)\). For the RF (Fig. 8C) and VL (Fig. 8D), there were no significant differences in EMG modulation between the three groups \((P = 0.060\) for RF and \(P = 0.118\) for VL). There was a significant difference in VM EMG modulation across groups \((P = 0.043, \text{Fig. 8E})\). Post hoc pair-wise comparisons of VM EMG modulation showed significant differences between CG1 vs. CG2 \((P = 0.046)\). No significant differences in MH EMG modulation were found between the three groups \((P = 0.521, \text{Fig. 8F})\). For the GM (Fig. 8G), EMG modulation was significantly different across groups \((P = 0.004)\). Post hoc pair-wise comparisons showed significant differences between SG vs. CG1 \((P = 0.008)\) and SG vs. CG2 \((P = 0.037)\).

**EMG activity of the outer leg**

Figure 8H illustrates the change in TA EMG amplitude of the outer leg between groups and across path curvatures. TA EMG modulation was significantly different
across groups ($P < 0.001$). Post hoc pair-wise comparisons showed significant differences between the SG vs. CG1 ($P = 0.002$) and the SG vs. CG2 ($P = 0.001$). LG EMG modulation was also significant different across groups ($P = 0.008$, Fig. 8I). Post hoc pair-wise comparisons showed significant differences between the SG vs. CG1 ($P = 0.008$). For the RF (Fig. 8J), VL (Fig. 8K), and VM (Fig. 8L), there were no significant differences in EMG modulation between the three groups ($P = 0.486$ for RF; $P = 0.354$ for VL; $P = 0.280$ for VM). In the BF (Fig. 8M), EMG modulation was significantly different across groups ($P = 0.001$). Post hoc pair-wise comparisons of BF EMG modulation showed significant differences between the SG vs. CG1 ($P = 0.001$). The difference between CG1 vs. CG2 approached significance ($P = 0.066$). For the GM (Fig. 8N), EMG modulation was significantly different across groups ($P = 0.001$). Post hoc pair-wise comparisons showed significant differences between the SG vs. CG1 ($P < 0.001$). CG1 and CG2 were also significantly different ($P = 0.002$). The comparison between the SG and CG2 approached significance ($P = 0.070$).

**Relationship of EMG Modulation to Impairment**

In addition to group data analysis, we assessed if there was any relationship between stroke severity (as assessed by the CMSA) and the amount of EMG modulation in each muscle in the SG (quantified by the slope of a linear equation between EMG amplitude and path curvature). We found that the CMSA score was significantly correlated to the modulation of BF EMG activity at end-swing when the paretic leg was the outer leg ($\rho = -0.71, P = 0.010$) and of the MG during stance when the paretic leg was the inner leg ($\rho = -0.62, P = 0.020$).
DISCUSSION

This study compared the neuromuscular strategies used by individuals with stroke and able-bodied controls to adapt locomotor patterns to curved paths. Our data from able-bodied participants were consistent with previous findings (Courtine et al. 2006). Specifically we showed that as path curvature increases, foot pressure shifts to the lateral aspect of the foot of the inner leg. This shift of pressure towards the inside of the turn was accompanied by a modulation of the amplitude of certain leg muscle activity. Our data suggests that individuals with stroke have altered modulation of EMG amplitude, accompanied by disordered medial-lateral shifts under the foot and reduced range of joint motion when walking along paths of increasing curvature.

Modulation of EMG activity to path curvature

To adapt locomotor patterns to turning, able-bodied participants exhibited opposing modulation patterns between the activity of the postero-medial (MG and MH) and postero-lateral muscles (LG and BF) of the inner and outer leg, respectively. In the inner leg, EMG activity of the postero-medial muscles decreased as path curvature increased. In the outer leg, EMG activity of the postero-lateral muscles decreased with increasing path curvature. These adaptations in MG, MH, LG, and BF EMG amplitude with turning are consistent with recent findings (Courtine et al. 2006). The opposite modulation patterns seen in the BF and MH between the inner and outer legs was a novel finding which extends the previous observations made in the gastrocnemii muscles. These differences between the medial and lateral compartments of the
posterior aspect of the leg could be necessary to adapt to the heightened medio-lateral balance demands of turning (Courtine and Schieppati 2003a; Orendurff et al. 2006). While walking along a curved path the body tilts (Patla et al. 1999) and the pelvis shifts toward the inside of the curve (Courtine et al. 2006). Data from the FSRs located underneath the foot of the inner leg are consistent with the idea that the center of mass shifts to the inside of the curve, toward the lateral aspect of the inner foot. This modulation seen in the lower limb may be used to change direction (Courtine and Schieppati 2003b) by allowing the segments to rotate in the direction of travel and as a strategy to maintain balance while the body center of mass shifts towards the inside of the circle (Hase and Stein 1999; Orendurff et al. 2006).

Our data also showed differential modulation of GM amplitude between the inner and outer legs. The main role of the GM during straight walking is to prevent the contralateral pelvis from dropping and to keep the swinging leg slightly raised to provide ground clearance for the foot (Ghori and Luckwill 1985). The increased amplitude of the GM of the inner leg during single stance as path curvature increased could serve to stabilize the upper body as the contra-lateral pelvis elevates and rotates the swinging leg in the direction of travel (Hase and Stein 1999). The increased amplitude of the GM during swing phase of the outer leg could serve to abduct the swinging leg bringing the foot closer to the midline of the body for heel strike and may be related to walking speed as it was not present when the velocity of the stroke group was matched.

In CG1, we noted that EMG amplitude in the quadriceps group (particularly VL and VM) decreased with increasing path curvature. This group also showed a decrease
in walking speed with increasing path curvature and we surmise that the accompanying change in quadriceps EMG activity was likely related to the decrease in speed (den Otter et al. 2004). Indeed, when we controlled for walking velocity in CG2, the activity of these muscles was not as strongly modulated in response to path curvature. Thus, the modulation of activity in the quadriceps group (VM, VL and RF) was likely mainly due to changes in walking speed rather than path curvature.

Stroke alters EMG modulation during curved walking

In the SG, we observed reduced modulation of EMG activity with path curvature compared to either CG1 or CG2 in many of the muscles. Even in comparison to CG2, where walking speed was controlled, the SG showed reduced modulation in the TA, MG, and GM of the inner leg, and the TA of the outer leg. Modulation in the LG, BF, and GM of the outer leg also tended to be less in the SG compared to controls. A recent study suggests that fewer modules (or muscle synergies) could account for the muscle activity pattern recorded during walking from individuals with stroke compared to controls, suggesting reduced flexibility in the neural commands for walking (Clark et al. 2010). Furthermore, there was an association between locomotor performance (e.g. self-selected walking speed) and the number of modules required to account for the EMG activity (Clark et al. 2010). We also found an association between the extent of modulation in the MG and BF to curved walking and the CMSA, which has been shown to be associated with locomotor impairments post-stroke (Patterson et al. 2008). Locomotor activity post-stroke is associated with impairments in corticospinal drive (Nielsen et al. 2008; Piron et al. 2005) and one possibility is that this could have
contributed to the reduced modulation of muscle activity patterns we observed during curved walking.

Another possible contributing factor to this lack of modulation is disordered coordination of gaze and head orientation (Lamontagne et al. 2007). In able-bodied individuals, reorientation of gaze and head position towards the direction of travel precedes changes in walking trajectory (Grasso et al. 1996; Hicheur et al. 2005; Hollands and Marple-Horvat 2001). It has been suggested that neck and vestibular afferent inputs resulting from this anticipatory head and gaze re-orientation (Courtine and Schieppati 2003a; Grasso et al. 1996; Hollands and Marple-Horvat 2001) are a prerequisite to successfully travel along a curved path (Bove et al. 2001; Fitzpatrick et al. 1999). Indeed, difficulties in turning are apparent if the head is immobilized with respect to the shoulders (Hollands and Marple-Horvat 2001). Stroke participants show impaired sequencing of gaze and body reorientation when turning a sharp 90 degree corner (Lamontagne et al. 2007). Severely impaired participants rotated gaze and body simultaneously only upon reaching the corner instead of anticipating the turn with gaze. Both neck and vestibular inputs project onto the vestibular nuclei of the brainstem, which in turn are believed to contribute to the tuning of EMG in select leg muscles (Gdowski et al. 2000; Gdowski and McCrea 1999). Hence in individuals with stroke, it is possible that the reduced vestibular and neck sensory inputs associated with anticipatory head rotation could contribute to the reduced modulation of lower limb muscle activity during turning. Further studies are required to investigate the association between activation of neck and vestibular afferent inputs and the modulation of lower limb EMG activity during turning.
Peripheral afferent input has also been shown to contribute to the modulation of lower limb muscle activity during gait. Stimulation of the sural nerve during the stance phase of walking can yield either suppressive or facilitatory responses in the gastrocnemius muscles, depending on the intensity of stimulation (Zehr et al. 1998). Our FSR data from the inner leg suggests that with increasing path curvature, there is a shift in pressure towards the lateral aspect of the foot, which would correspond to the receptive field of the sural nerve. Concomitant with this was a reduction in MG EMG activity of the inner leg during stance, which corresponds to the reflex response to higher intensity stimulation of the sural nerve (Zehr et al. 1998). Further research is planned to elucidate the contribution of cutaneous afferent input on the modulation of EMG activity during turning. Compromised peripheral afferent feedback in people with stroke (Kim and Choi-Kwon 1996) could have contributed to the reduced modulation of EMG activity with turning, but this needs to be confirmed by future studies.

One limitation of the present study was that we did not record activity from the non-paretic leg during turning. It is likely that there would be compensations on the non-paretic side for the impairment and muscle weakness of the paretic side. However, such compensations should be investigated with a combination of whole-body kinematic, kinetic (e.g. ground reaction forces) and electromyographic measures, which were beyond the scope of the present study. Future studies in our lab are planned to provide such detailed analysis in order to gain an appropriate understanding of the compensations required of the non-paretic limb.
Conclusions

Turning in able-bodied individuals requires modulation of the kinematic and EMG patterns required for straight ahead walking. EMG amplitude of the muscles on the postero-lateral aspect of the outer leg and on the postero-medial aspect of the inner leg decreases with increasing path curvature. Modulation of EMG amplitude is also evident in the GM during mid-stance of the inner leg with changes in path curvature. These modulation patterns are not observed in the paretic leg of the stroke participants regardless of whether the leg was on the inside or outside of the turn. Further research is warranted to evaluate the importance of gaze, head orientation, and peripheral sensory input on the modulation of muscle activity during curved walking.
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REFERENCES


FIGURE LEGENDS

Figure 1: Identification of periods of interest in EMG activity across the four walking paths.

Windows of interest for quantifying EMG activity were determined by comparing EMG patterns between A) straight vs. large, B) straight vs. medium, and C) straight vs. small circular paths in CG1. The ensemble average traces of the rectified EMG of the MG muscle is used here as an example. Dotted lines in each graph represent the probability (P value) of the difference between the averaged traces as assessed by paired t-tests (see Methods). D) The time of occurrence of a significant difference between the straight condition and at least two of the other path curvatures for a minimum of 10 points (indicated by grey shaded area). The average rectified EMG for each muscle was calculated during this time window.

Figure 2: Change in over ground walking speed with increasing path curvature.

Average walking speed of each participant group for each path curvature. Participants in CG1 (black circles) and the SG (grey and white box symbols) walked at their self-selected speed in each path curvature. Participants in CG2 (grey circles) were instructed to match the over ground walking speed of the SG. SG-P, stroke group, turns to paretic side; SG-NP, stroke group, turns to non-paretic side. Error bars represent standard deviation.
**Figure 3:** Average joint excursion of the ankle, knee and hip during walking along different path curvatures.

In each panel, joint range of motion during walking along each of the different path curvatures is plotted superimposed on each other. All data were normalized in time to 100% of the step cycle, separated according to whether the leg was A) the inner or B) the outer leg of the curve, and ensemble averaged within each group.

**Figure 4:** Changes in foot pressure during walking along different path curvatures.

In each panel, signals from the FSRs during walking along each of the different path curvatures are plotted superimposed on each other. All data were normalized in time to 100% of the step cycle, separated according to whether the leg was the inner (top panel) or outer (bottom panel) leg of the curve, and ensemble averaged for the control (A and B) and stroke group (C and D).

**Figure 5:** Lower limb EMG activity patterns during walking along different path curvatures in controls at self-selected walking speed.

In each panel, EMG gait patterns in the control group 1 (CG1) during each of the different path curvatures are plotted superimposed on each other. All data were normalized in time to 100% of the step cycle, separated according to whether the leg was A) the inner or B) the outer leg of the curve, and ensemble averaged across all control participants. Grey shaded areas indicate windows of interest from which EMG activity was quantified for subsequent analysis.
Figure 6: Lower limb EMG activity patterns during walking along different path curvatures in controls at matched walking speed to the stroke group.

In each panel, EMG gait patterns in the control group 2 (CG2) during each of the different path curvatures are plotted superimposed on each other. All data were normalized in time to 100% of the step cycle, separated according to whether the leg was A) the inner or B) the outer leg of the curve, and ensemble averaged across all control participants. Grey shaded areas indicate windows of interest (identified by the data from CG1, see Fig. 5) from which EMG activity was quantified for subsequent analysis.

Figure 7: Lower limb EMG activity patterns during walking along different path curvatures in individuals with stroke.

In each panel, EMG gait patterns in the stroke group (SG) during each of the different path curvatures are plotted superimposed on each other. All data were normalized in time to 100% of the step cycle, separated according to whether the leg was A) the inner or B) the outer leg of the curve, and ensemble averaged across all stroke participants. Grey shaded areas indicate windows of interest (identified by the data from CG1, see Fig. 5) from which EMG activity was quantified for subsequent analysis.

Figure 8: Changes and modulation of EMG amplitude with path curvature.

Average rectified EMG amplitude over periods of interest in each muscle that showed significant relationship to path curvature (as identified by the data from CG1,
see Fig. 5). Left panel: Modulation of EMG amplitude of the TA, MG, RF, VL, VM, MH, and GM in the inner leg (A-G). Right panel: Modulation of EMG amplitude of the TA, LG, RF, VL, VM, BF, and GM in the outer leg (H-N). Numbers to the right side of each plot indicates the average slope (and standard deviation) of the relationship between EMG amplitude and path curvature for control group 1 (CG1, black circles), control group 2 (CG2, white circles), and the stroke group (SG, grey squares). Asterisks represent differences in EMG modulation between groups at $P < 0.05$ and double-cross symbols represent differences at $P < 0.10$. Error bars represent standard deviation.
Figure 1

A. 

B. 

C. 

D. 

- straight
- large
- medium
- small
- onset
Figure 2

The figure shows the relationship between walking speed (m/s) and path curvature (m) for different conditions.

- SG-P
- SG-NP
- CG1
- CG2

The graph indicates a decrease in walking speed as the path curvature increases for all conditions.
A. inner leg control stroke

B. outer leg control stroke

ankle flex/ext

knee flex/ext

hip flex/ext

hip abd/add

% of step cycle

% of step cycle

joint angle (degrees)

Straight

Large

Medium

Small
Figure 4

A. control-inner

B. control-outer

C. stroke-inner

D. stroke-outer

- heel FSR
- medial FSR
- lateral FSR

Force (normalized units)

% of step cycle

Straight  Large  Medium  Small
Figure 5

A. CG1: inner leg

B. CG1: outer leg

EMG amplitude (normalized units)

% of step cycle

Straight  Large  Medium  Small
Figure 6

A. CG2: inner leg

B. CG2: outer leg

EMG amplitude (normalized units)

% of step cycle

Straight | Large | Medium | Small

Figure 6
Figure 7

A. SG: inner leg

B. SG: outer leg

EMG amplitude (normalized units)

% of step cycle

Straight
Large
Medium
Small
Figure 8

EMG amplitude (normalized units)

inner leg

A. TA

B. MG

C. RF

D. VL

E. VM

F. MH

G. GM

outer leg

H. TA

I. LG

J. RF

K. VL

L. VM

M. BF

N. GM

path curvature (m)