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ORIGINAL RESEARCH

Gait Impairments in Patients Without Lower Limb Hypertonia Early Poststroke Are Related to Weakness of Paretic Knee Flexors

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Abstract

Objective: To describe gait characteristics of patients without clinical evidence of lower limb hypertonia within 2 months of stroke and explore the relationship between gait and residual motor function.

Design: Cohort study.

Setting: Motion analysis laboratory in a tertiary-care free-standing rehabilitation hospital.

Participants: Consecutive sample of 73 eligible inpatients (first-known stroke <2 months postonset, walking independently, modified Ashworth score of 0 in the paretic lower limb) and 27 healthy controls (N = 100).

Interventions: Not applicable.

Main Outcome Measures: Gait speed, stride and step lengths and cadences, stance time, single-support and double-support times, and associated symmetry measures in patients at self-selected normal speed and controls at very slow speed (51.1±32.6 cm/s and 61.9±21.8 cm/s, respectively, $P = .115$); Fugl-Meyer lower extremity motor score (FM-LE) and isometric knee flexion and extension strength in patients.

Results: Except the stride/step cadence, all temporospatial parameters significantly differed between the stroke and control participants. Furthermore, significantly greater asymmetries were found in the patients for the overall stance time, initial double-support and single-support times, and step cadence, reflecting smaller values in the paretic than nonparetic limb. Most temporospatial parameters moderately to strongly correlated with the gait speed ($|r|$: .72-.94, $P < .0001$), FM-LE ($|r|$: .42-.62, $P \leq .0005$), and paretic knee flexor strength ($|r|$: .47-.57, $P \leq .0004$).

Conclusions: Gait of patients without clinical evidence of lower limb hypertonia within 2 months of stroke is characterized by many temporospatial deviations and asymmetries. The self-selected normal gait speed, FM-LE, and paretic knee flexor strength can discriminate gait impairments in these patients shortly before inpatient discharge. It remains to be determined whether the observed relationships between paretic knee flexor strength and gait measures warrant the development of interventions for strengthening of the paretic knee flexors in order to improve gait early poststroke.

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Most individuals at the peak of recovery poststroke walk at slower than normal speeds, due to decreased stride length and cadence, and with an asymmetric gait pattern.[1-3] The paretic limb, compared with the nonparetic limb, shows longer step length and step time and shorter stance and single-support times.[3-5] The self-selected gait speed has shown an inconsistent relationship with temporospatial gait parameters in previous chronic stroke studies. Olney et al,[6] unlike other investigators,[7,8] have reported a significant negative correlation between gait speed and paretic stance time. It has been found also that gait speed is related to the asymmetry in stance and swing ratio by 2 groups[2,9] but not by Roth et al.[8]

Several features of the impaired motor control poststroke may account for the long-lasting changes in temporospatial gait parameters. A recent review concluded that strength in the paretic ankle dorsiflexors, but not knee extensors, is closely related to gait speed.[10] However, a recent study showed that isokinetic torques of the paretic knee extensors and flexors were better predictors of the average 6-minute walk speed than the ankle and hip torques.[11] Since muscle hypertonia commonly accompanies weakness,

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particularly later poststroke, many studies have documented the associated temporospatial gait deviations.[12,13] Spastic motor behaviors (synergies, co-contractions), which typically coexist with the lower limb resting hypertonia, are believed to amplify the negative impact of weakness on gait.[14-16] However, the absence of resting muscle hypertonia in the paretic lower limb provides an opportunity to establish a clearer relationship between the changes in gait and residual motor function, particularly with the muscle strength.

Temporospatial gait characteristics have not been examined extensively early poststroke. Among the existing reports, the recruited samples vary greatly with respect to the timing of evaluation. To illustrate the point, the previous instrumented gait analysis studies labeled as the *subacute* recruited participants within the first 3 weeks,[17] 6 weeks,[18] 9 weeks,[19] 3 months,[20] 5 months,[21] and 6 months[22] poststroke. Furthermore, most studies included patients with hypertonia in the paretic limb (eg, [22,23]) or made no reference to it (eg, [17,19,21]).

In light of the above observations, the purpose of this study was to address the insufficient characterization of gait among patients without lower limb hypertonia, the inconsistent findings on the relationship between gait characteristics and knee muscle strength, and the wide scatter between the time of evaluation and stroke onset. The objectives were (1) to characterize the temporospatial characteristics of gait in ambulatory patients with stroke without clinical evidence of resting hypertonia in the paretic muscles at the end of inpatient rehabilitation; (2) to compare the temporospatial gait patterns between the subacute stroke sample and healthy controls; and (3) to determine if temporospatial and symmetry measures were related to gait speed, Fugl-Meyer lower extremity motor score (FM-LE), and paretic knee strength in subacute stroke. We first predicted significant differences in temporospatial and symmetry measures between the subacute stroke and control groups (hypothesis 1). Since gait speed is a reliable marker of ambulatory function poststroke,[24] we anticipated that the gait speed would be closely related to temporospatial and symmetry measures in the subacute stroke sample (hypothesis 2). Finally, we hypothesized that the temporospatial and symmetry measures would be significantly correlated with the residual motor function (FM-LE, strength of the paretic knee extensors and flexors) (hypothesis 3). The participants were recruited within 2 months of stroke that coincides with the fastest rate of motor recovery[25,26] and the typical time of inpatient discharge in the United States, with the intent to provide knowledge that could be used to better define goals of gait training in the outpatient settings.

Methods

Participants

A consecutive sample of 73 eligible patients who received inpatient rehabilitation within 2 months of stroke were recruited shortly before discharge. The inclusion criteria were (1) first documented stroke; (2) able to follow simple instructions; (3) able to walk independently for 7 meters with or without assistive devices; (4) no artificial lower extremity joint replacement; and (5) no resting muscle hypertonia in the major hip, knee, and ankle muscles (modified Ashworth score[27] of 0, but not flaccid). They were compared to 27 healthy controls available in our gait database (table 1). Because gait of subjects without stroke is known to be consistent, as also documented here by small SD values of analyzed parameters, the control sample was deemed sufficient for comparisons to the patients with stroke. All participants signed the informed consent approved by the institutional review board.

Prior to gait evaluation, the paretic limb was assessed using the FM-LE motor section[28] (not performed in 6 patients because the research therapist was not available). Knee strength data were collected in patients after the gait assessment.

List of abbreviations:

FM-LE **Fugl-Meyer lower extremity motor score**
SI **symmetry index**

Gait evaluation

Patients with stroke completed 4 passes on either a GAITRite[a] (4.3-m length) or Zeno[b] (6.1-m length) electronic walkway. Despite the shorter length, the GAITRite provided enough data for analysis since patients walked with short steps. They were asked to walk in their own shoes at a self-selected comfortable speed and with a customary assistive device, if any (see table 1). Nine patients wore a short, nonrigid polypropylene ankle-foot orthosis on the paretic side to prevent foot drop and 5 of them used an assistive device (4, a walker; 1, a cane).

The data for those without stroke were extracted from our gait database and the methodological details have been described elsewhere.[29] The gait of those without stroke was captured using a camera-based motion capture system.[c] A high agreement between the electronic walkway and motion capture gait analysis has been demonstrated in those without stroke and patients with stroke.[30] To account for the effect of gait speed on temporospatial parameters,[12] the data for the very slow speed were selected for comparison. The 2 sides of controls were averaged for each step parameter and used in the statistical analysis.

Knee strength evaluation

An isokinetic dynamometer[d] was used for assessing isometric strength at 90° knee flexion angle in the stroke sample. The torque signals were collected by a Cortex data acquisition system[e] and also exported to an analog-to-digital converter[e] to provide a real-time visual feedback on a liquid crystal display monitor.[31] Patients were asked to produce in turn maximum knee extension and flexion contractions (3 each). Each trial lasted for 3 to 4 seconds with a 1-minute rest in between. Knee strength data were not available in 20 patients due to time constraints (n=53).

Data reduction

GAITRite[a] or PKMAS[b] software was used to export the toe and heel locations, and timing of each initial foot contact and toe-off instants for each trial. A computer program written in MATLAB[f] was used to calculate different temporospatial parameters (see computational definitions in appendix 1). Only full gait cycles were analyzed based on the presence of all 5 critical instants (first ipsilateral initial foot contact, contralateral toe-off, contralateral initial foot contact, ipsilateral toe-off, and second ipsilateral initial foot contact). The temporospatial symmetry was assessed using a symmetry index (SI): {SI=(paretic − nonparetic)/

Table 1 Demographics of the recruited samples and characteristics of lower extremity motor function in the stroke sample shown as frequency and mean ± SD (range)

Variable	Stroke (n=73)	Control (n=27)
Sex (men/women)	45/28	14/13
Age (y)	62±13 (32-86)	56±13 (36-80)
Body mass index (kg/m^2)*	30±6 (16-46)	23±4 (14-28)
Gait speed (cm/s)	51±33 (6-170)	62±22 (23-105)
Time postonset (d)	20±10 (6-52)	NA
Stroke type (ischemic/hemorrhagic)	62/11	NA
Paretic side (right/left)	34/39	NA
Ankle-foot orthosis (right/left)	5/4	NA
Assistive device (cane/rolling walker)	7/13	NA
Fugl-Meyer lower extremity motor† (0-34)	24±7 (7-34)	NA
Knee strength‡ (row 1: Nm, row 2: Nm/kg)		
Flexion, paretic	32±24 (0.2-104)	NA
	0.36±0.26 (0.01-1.16)	
Flexion, nonparetic	56±23 (19-130)	NA
	0.63±0.24 (0.21-1.28)	
Extension, paretic	100±56 (13-275)	NA
	1.08±0.53 (0.20-2.33)	
Extension, nonparetic	144±63 (44-329)	NA
	1.61±0.62 (0.58-3.23)	

Abbreviation: NA, not applicable or available.
* Significant group difference (unpaired t test, $P \leq .001$).
† n=67.
‡ n=53.

[.5×(paretic + nonparetic)]×100%}.[32] This computation of SI is preferred because it indicates both the direction and magnitude of the asymmetry.

For each knee muscle group, the largest torque of the 3 trials was used for analysis. To account for the variation in body weight, knee strength was normalized to subject's body mass (Nm/kg).

Statistical analysis

Mean and SD were calculated for each temporospatial parameter corresponding to the paretic limb, nonparetic limb, and control limb. For the first hypothesis, stride parameters and SIs were compared between patients and controls using 2-tailed unpaired t tests. For each step parameter, a paired t test was used to compare the paretic with nonparetic limb in patients and 2 unpaired t tests to compare each limb of patients with controls (all 2-tailed). The second hypothesis was tested in the stroke sample by deriving coefficients of correlation between gait speed and each temporospatial and symmetry measure. For the third hypothesis, the same coefficients of correlation were computed between measures of motor function (FM-LE, paretic knee muscle strength) and different temporospatial parameters and SIs. Prior to each correlation analysis, the skewness of each variable was determined. The Pearson correlation was used when the skewness for both variables was within the range of ±1. Otherwise, the Spearman rank correlation was used. Considering the number of statistical tests performed, more stringent alpha levels of .01 and .001 were adopted for the t tests and correlations, respectively. All statistical tests were completed using the Statistics Toolbox of MATLAB.

Results

The subacute stroke and control groups were not different in age (2-tailed unpaired t test, $P=.062$) and gait speed ($P=.115$), but the patients had greater body mass index ($P<.001$, see table 1) due to a greater body mass (89±18 kg vs 68±16 kg, $P<.001$). In terms of the first hypothesis, patients walked slower and with lower cadence at their self-selected speed than controls at the very slow speed, but the differences were not significant ($P \geq .115$, table 2). However, the stride length was significantly shorter and the step width was significantly wider in the subacute stroke than control group ($P \leq .004$, see table 2). As to the step parameters (see table 2), the paretic limb, in comparison to the nonparetic limb, showed significantly longer step length and prolonged late double-support time but slower step cadence and shorter stance time, due to shorter initial double-support and single-support times ($P \leq .005$). Compared to controls, the paretic limb had significantly shorter single-support and prolonged late double-support times (both $P<.001$), whereas the nonparetic limb had significantly shorter step length and prolonged stance and initial double-support times ($P \leq .002$). Although most SIs were greater in the subacute stroke than controls, the between-group differences were only significant for the SIs of stance time, initial double-support time, and step cadence ($P \leq .01$, see table 2) due to smaller values in the paretic than nonparetic limb (negative SIs).

Regarding the second and third hypotheses, the gait speed, FM-LE score, and paretic knee flexor strength, but not extensor strength, significantly correlated with the stride length and cadence ($P \leq .0001$, fig 1). The gait speed also significantly correlated with all step parameters (all $P<.0001$) on both the

Table 2 Group mean ± SD values for temporospatial gait parameters with the corresponding comparisons

Variable	ST/PR	NP	CT	ST vs CT	PR vs NP	PR vs CT	NP vs CT
Stride Parameter							
Gait velocity (cm/s)	51.1±32.6	NA	61.9±21.8	NS	NA	NA	NA
Stride length (cm)	81.6±25.3	NA	97.7±20.2	*	NA	NA	NA
Stride cadence (steps/min)	69.3±24.7	NA	74.5±16.5	NS	NA	NA	NA
Step width (cm)	12.8±3.3	NA	10.0±3.8	†	NA	NA	NA
Step Parameter							
Stance time (%)	70.0±6.6	75.3±8.1	67.7±4.3	NA	†	NS	†
Initial double-support time (%)	20.0±5.0	25.5±11.4	17.7±4.3	NA	†	NS	†
Single-support time (%)	24.7±8.1	29.9±6.5	32.3±4.3	NA	†	†	NS
Late double-support time (%)	25.4±11.3	19.9±4.9	17.7±4.3	NA	†	†	NS
Step cadence (steps/min)	66.1±26.5	77.7±21.3	74.5±16.5	NA	†	NS	NS
Step length (cm)	42.7±12.0	38.8±15.5	48.9±10.1	NA	*	NS	*
SI (%)							
Stance time	-7.1±8.3	NA	-0.2±1.9	†	NA	NA	NA
Initial double-support time	-18.3±35.5	NA	-0.1±9.6	*	NA	NA	NA
Single-support time	-22.5±27.8	NA	-0.5±3.9	†	NA	NA	NA
Late double-support time	18.0±35.7	NA	0.4±10.1	NS	NA	NA	NA
Step cadence	-20.6±27.6	NA	-0.4±4.0	†	NA	NA	NA
Step length	13.7±38.5	NA	0.8±6.2	NS	NA	NA	NA

NOTE. Stride and symmetry parameters are inclusive of both lower limbs for the ST and CT groups. Step parameters in the ST group are reported separately for the PR and NP lower limbs and averaged across the 2 limbs in the control group.
Abbreviations: CT, control; NA, not applicable; NP, nonparetic; NS, not significant; PR, paretic; ST, stroke.
* $P \leq .01$.
† $P \leq .001$.

paretic and nonparetic sides (fig 2 and 3). Both the FM-LE score and the paretic knee flexor strength were significantly related to the paretic step cadence and single-support time ($P \leq .0002$, see fig 2) as well as the nonparetic step length, step cadence, and stance time ($P \leq .0005$, see fig 3). Only FM-LE correlated with the paretic late double-support ($r = -.44$, $P = .0002$, not shown; same as the nonparetic initial double-support), whereas the paretic knee flexor strength correlated with the paretic step length ($P = .0002$, see fig 2).

In terms of symmetry, both the gait speed and FM-LE score significantly correlated with all step SIs ($P \leq .0009$, fig 4). There was no significant correlation between the strength of the paretic knee flexors or extensors and any of the SIs (see fig 4).

Discussion

This study revealed temporospatial gait deviations and asymmetries in patients without clinical evidence of muscle hypertonia in the paretic lower limb within 2 months of stroke. The only parameter that did not significantly differ between the subacute stroke and control groups was the stride cadence. Most temporospatial gait parameters were moderately to strongly associated with the overall gait speed and the residual motor function (FM-LE, paretic knee flexor strength). To emphasize clinically relevant results, the discussion is focused on the correlations of gait speed and motor function with temporospatial and symmetry measures.

Gait in patients with and without subacute stroke

The hypothesis of significant differences in temporospatial and symmetry measures between the patients with subacute stroke and those without stroke (hypothesis 1) was largely confirmed except for the stride and step cadences, and SIs of step length and late double-support time (see table 2). No significant difference in the step length asymmetry despite a big difference in the mean SIs is likely due to the large variance in our stroke sample (range -27% to 156%). In fact, about 50% of our patients had a small negative SI due to a slightly longer step on the nonparetic side. This agrees with the previous report of a greater step length on the nonparetic side in 9 of 20 participants with chronic stroke.[33] Thus, the temporal asymmetry may discriminate patients with subacute stroke from those without stroke better than the spatial asymmetry.

Among different step parameters, 82% of our patients had a negative SI for single-support time, which differed from controls the most (see table 2). The significantly shorter single-support time on the paretic compared to nonparetic side may be associated with the paretic limb weakness and impaired coordination and balance.[4,34] The instability during the paretic single-support may limit the advancement of the nonparetic limb during swing and shorten these 2 corresponding phases of gait. The overall results reinforce the view that gait analysis poststroke should focus on the symmetry in the single-support time.[9,35] Moreover, increased gait speed but not symmetry in the chronic stage may reflect compensation rather than improvement in the paretic limb function.[36]

Relationship between gait speed and temporospatial parameters

The hypothesis of close relationships between the gait speed and different temporospatial and symmetry measures (hypothesis 2) was essentially confirmed since the gait speed significantly correlated with all temporospatial parameters and SIs (see fig 4). In line with previous findings in subacute stroke,[23] both the stride length and cadence highly correlated with gait speed (see

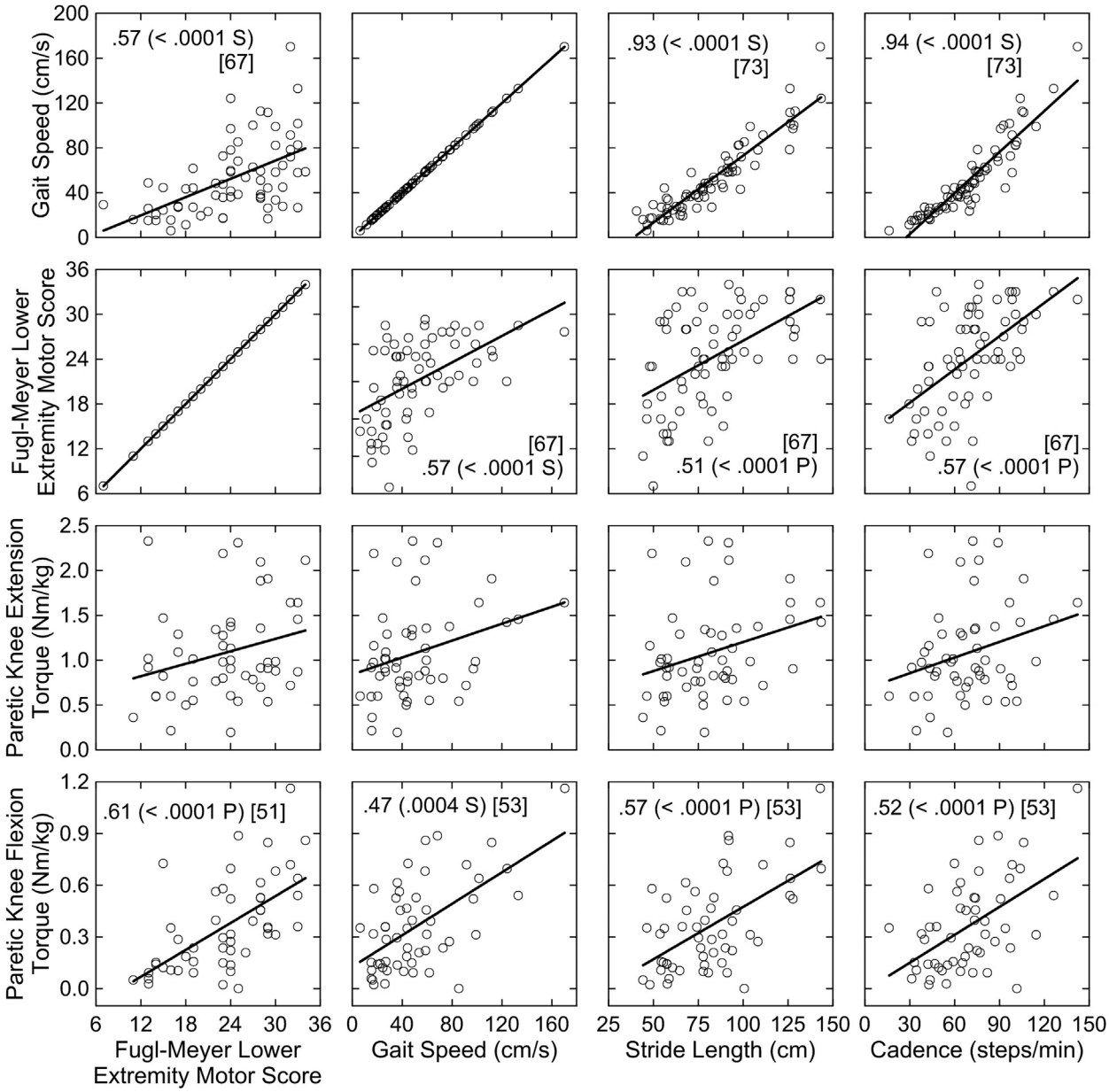

Fig 1 Correlation matrix between gait velocity, Fugl-Meyer lower extremity motor score, normalized knee strength, and temporospatial stride parameters. Significant correlations are indicated by correlation coefficients. The associated *P* values and the test type, Pearson (P) or Spearman (S) correlation, are in parentheses. The sample sizes are in square brackets.

fig 1), indicating that gait speed is modulated by proportional changes in both the stride length and cadence. Strong associations between the gait speed and step parameters of both limbs suggest that motor impairment and adaptive behavior may coexist even early poststroke. The relations between gait speed and temporal step parameters seem to be nonlinear and deserve further investigation.

Relationship between motor function and temporospatial parameters

Our third hypothesis was only partially supported because while selected temporospatial and symmetry parameters significantly correlated with FM-LE and the paretic knee flexor strength, none correlated with the paretic knee extensor strength. Given the items that comprise FM-LE, the results suggest that the motor deficits in the paretic limb drive the bilateral changes in gait poststroke.

The novel finding of this study is that strength in the paretic knee flexors but not extensors emerged as the main determinant of temporospatial measures. This is in contrast with the reports of significant correlations between the knee extensor strength and selected temporospatial parameters,[37,38] which is likely due to sampling and methodological differences. Since our patients had stronger knee extensors than the subjects in 2 previous subacute stroke studies[39,40] but walked somewhat slower, the lack of significant correlations between temporospatial parameters and knee extensor strength should not come as a surprise.

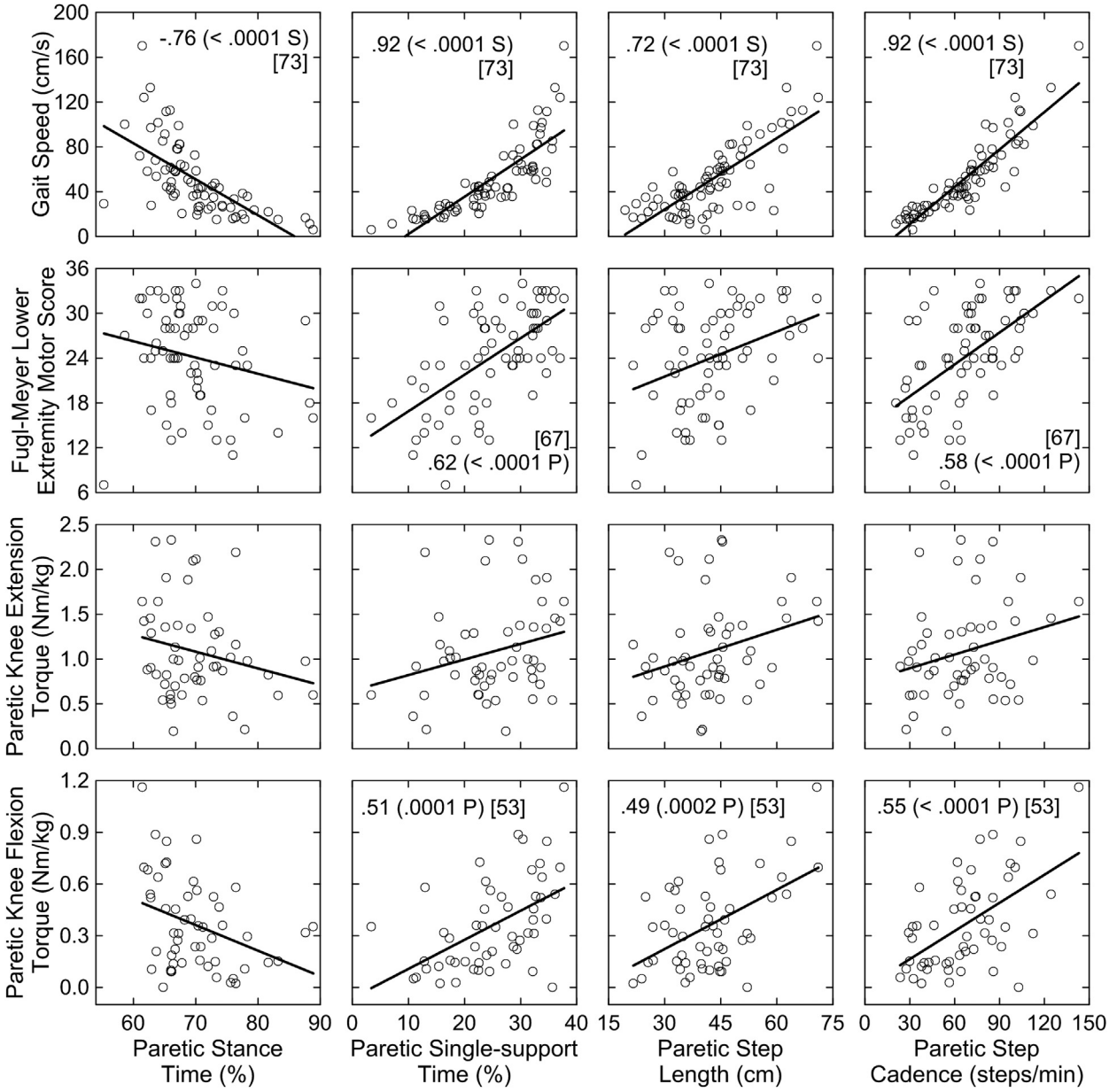

Fig 2 Correlation matrix between gait velocity, Fugl-Meyer lower extremity motor score, normalized knee strength, and paretic temporospatial step parameters. Significant correlations are indicated by correlation coefficients. The associated *P* values and the test type, Pearson (P) or Spearman (S) correlation, are in parentheses. The sample sizes are in square brackets.

Relationship between gait speed and motor function

Although not examined as a formal hypothesis, our results concur with the studies in chronic stroke showing that gait speed is closely associated with FM-LE[15,41] and the paretic knee flexor strength[42,43] (see fig 1). Compared to 3 studies performed on average at 2 months poststroke, a moderate correlation between gait speed and paretic knee extensor strength reported here ($r=.30$, $P=.027$, see fig 1) agrees with Bohannon[37] ($r=.36$, average speed 51 cm/s). However, the same author[44,45] reported stronger correlations in 2 other cohorts ($r=.68$ and .73) that walked on average much slower than our patients (29 and 37 cm/s). The validity of our results is reinforced by additional analyses showing that the gait speed also correlated with the absolute (Nm) paretic knee flexor torque ($r=.56$, $P<.001$) but not the nonparetic knee flexor torque or the knee extensor torque on either side ($r\leq.25$, $P\geq.070$).

Clinical implications

The first implication of our results is that gait can be markedly impaired even in the absence of resting muscle hypertonia in the lower limb affected by stroke. Although it may be tempting to ascribe the reported gait impairments to muscle weakness or incoordination, the absence of resting hypertonia does not imply

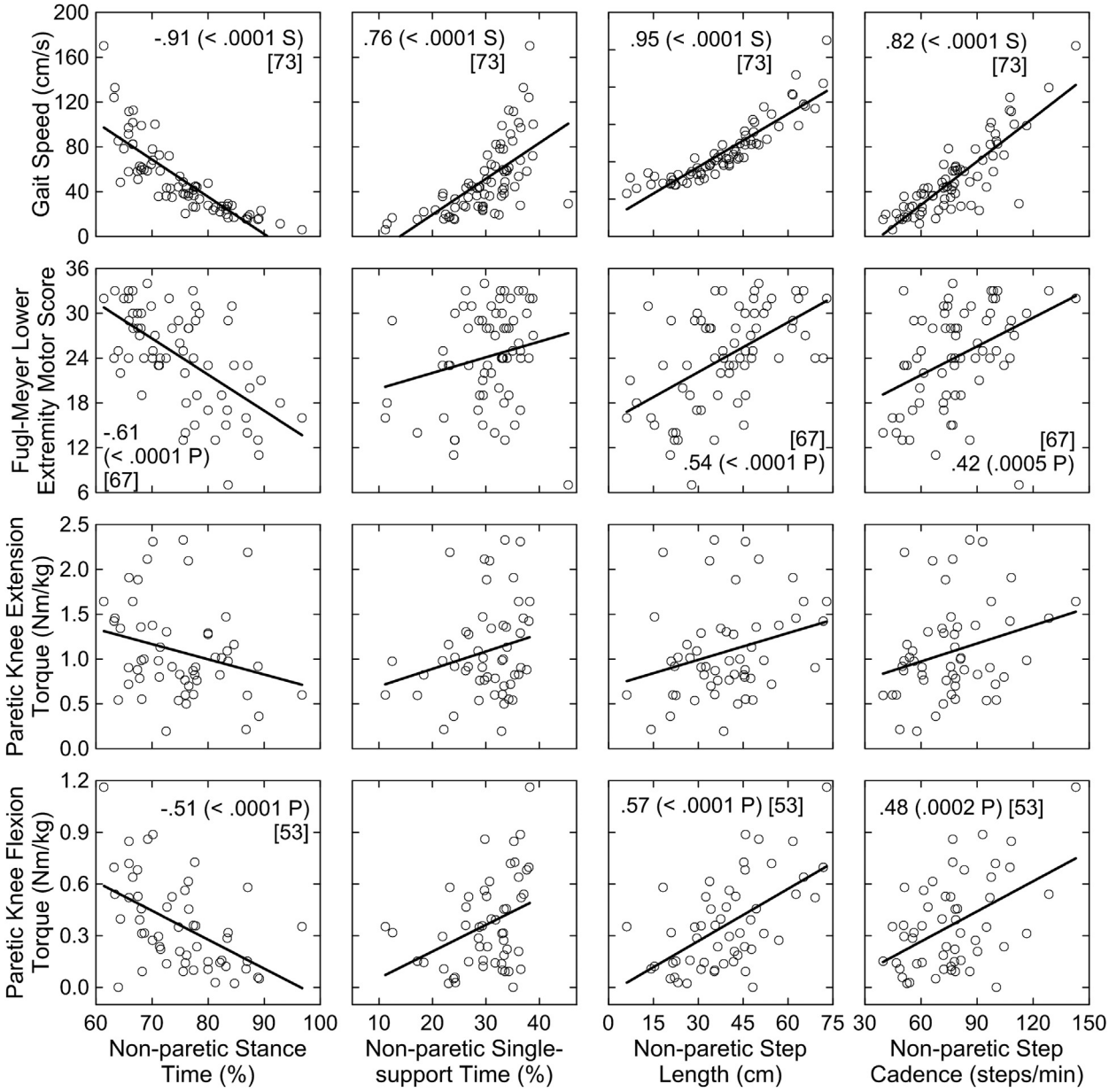

Fig 3 Correlation matrix between gait velocity, Fugl-Meyer lower extremity motor score, normalized knee strength, and non-paretic temporospatial step parameters. Significant correlations are indicated by correlation coefficients. The associated *P* values and the test type, Pearson (P) or Spearman (S) correlation, are in parentheses. The sample sizes are in square brackets.

that other positive features of the upper motor neuron syndrome (eg, muscle synergies, co-contractions) were not present during gait, which warrants further studies.

The second implication is that it may be warranted to include strengthening exercises for the paretic knee flexors in attempt to improve gait early poststroke. Although ankle muscle strength is related to ambulatory function,[10] the knee flexor weakness may also impact gait. In individuals without stroke, the knee flexor muscles are mainly active during the late-swing and early-stance[46,47] and act in synergy with the hip extensors and ankle plantar flexors to reinforce the stance.[48] However, a prolonged activation of the paretic knee flexors lasting from early swing to midstance has been reported in people with chronic stroke[49,50] who walked within the range of speeds reported here. The role of knee flexors is further supported by improvements in gait after providing functional electrical stimulation to the paretic knee flexors in patients with stiff knee gait.[51] Thus, it seems justified to target the knee flexors for improving ambulation early poststroke.

Study limitations

Only linear correlations were examined even though some scatter plots suggest nonlinear relationships. The lower limb muscle hypertonia was assessed only clinically while resting supine, which does not exclude the possibility that some features of the spastic motor syndrome were present during gait, which requires further studies. The mean difference in gait speed between the stroke and control groups may be viewed as a limitation, however, it was not statistically significant. Moreover, because gait asymmetry post-stroke is more pronounced at faster speeds, the reported

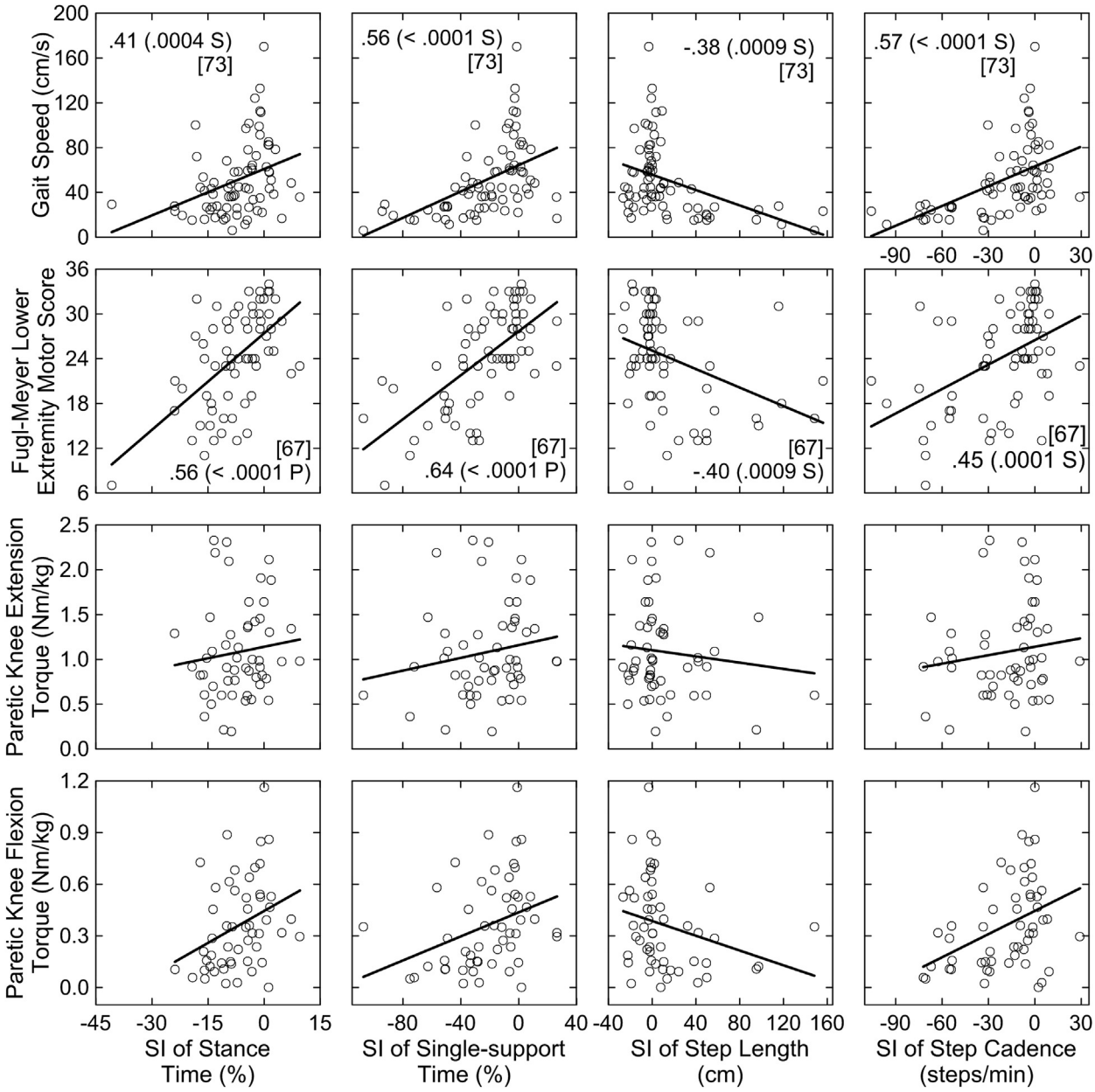

Fig 4 Correlation matrix between gait velocity, Fugl-Meyer lower extremity motor score, normalized knee strength, and temporospatial SIs. Significant correlations are indicated by correlation coefficients. The associated *P* values and the test type, Pearson (P) or Spearman (S) correlation, are in parentheses. The sample sizes are in square brackets.

differences in SIs may be underestimated since our patients walked somewhat slower than patients without stroke. Since we did not examine ankle muscle strength, the associations with the observed gait impairments remain unknown.

Conclusions

Temporospatial characteristics of gait are altered in patients without clinical evidence of resting hypertonia in the affected limb muscles within 2 months of stroke. The moderate-to-strong relationships between gait speed, bilateral temporospatial step measures, and the residual motor function suggest that the observed gait deviations are the product of motor impairment and compensatory adaptation. Since weakness of the paretic knee flexors may be one of the key determinants of gait in this population, future studies should determine if the strengthening of knee flexors early after a stroke might have a lasting beneficial effect on gait.

Suppliers

a. GAITRite Walkway; CIR Systems, Inc.
b. Zeno Walkway; ProtoKinetics.
c. Digital cameras and OrthoTrak Gait Analysis software; Motion Analysis Corp.
d. Biodex System 3; Biodex Medical Systems, Inc.
e. Model USB-6009, National Instruments Corp.
f. MATLAB; MathWorks Inc.

Keywords

Hamstring muscles; Muscle strength; Neurologic gait disorders; Rehabilitation; Stroke rehabilitation; Walking speed

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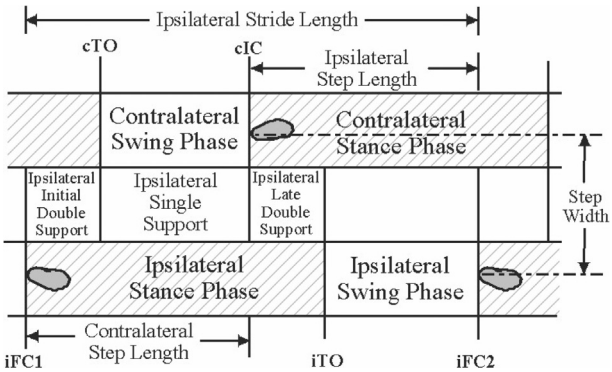
Appendix 1 Computational Definitions of Temporospatial Gait Variables



Variable	Definition	Unit
Stride time	Duration from iIC1to iIC2.	s
Stance time	Duration from iIC1 to iTO.	%ST
Initial double-support time	Duration from iIC1 to cTO.	%ST
Late double-support time	Duration from cIC to iTO.	%ST
Single-support time	Duration from cTO to cIC.	%ST
Step time	Duration from cIC to iIC2.	%ST
Step cadence	60 divided by step time.	steps/min
Step length	Location of ipsilateral heel at iIC2 relative to the contralateral heel at cIC in the fore-after direction (line of progression).	cm
Stride length	Displacement of the ipsilateral heel location from iIC1 to iIC2 in the fore-after direction.	cm
Stride velocity	Stride length divided by stride time.	cm/s
Cadence	Average over left and right cadences.	steps/min
Step width	Distance between heel locations at cIC and iIC2 in the mediolateral direction.	cm
Gait speed	Average over left and right stride velocities.	cm/s

Abbreviations: cIC, contralateral initial foot contact; cTO, contralateral toe-off; iIC1, first ipsilateral initial foot contact; iIC2, second ipsilateral initial foot contact; iTO, ipsilateral toe-off; ST, stride time.

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