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Asymmetry in walking performance and postural sway in patients with chronic unilateral cerebral infarction

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Abstract—The asymmetrical nature of hemiparetic gait is well known; however, the role of walking asymmetry for speed performance is unclear. The purpose of the present study was to determine whether the range of walking speeds in chronic hemiparetic patients is associated with their gait asymmetry and postural sway. Twenty ambulatory patients with chronic unilateral supratentorial infarction were studied. Foot-ground contact patterns during swing and stance phases at various self-selected walking speeds were analyzed. The magnitude and direction of asymmetry in durations of stride phases were evaluated and compared with healthy subjects. Posturographic studies were performed to estimate the postural sway during quiet standing. Hemiparetic patients walked slower, more asymmetrically, and swayed more laterally favoring their nonaffected leg than did healthy persons. Although there was variability in durations of stride phases when comparing the two sides, a prolonged swing on the affected side and a prolonged stance on the nonaffected side were observed in all patients. The magnitude of asymmetry in stride phases varied among the patients; however, it was significantly higher than in controls ($p<0.03$). Increased mean lateral sway during quiet standing was indicative of restricted velocity performance during walking. Patients with higher swing asymmetry achieved their maximum speed performance at lower velocity levels. However, the ability of patients to ambulate with a number of self-selected speeds was not associated with the magnitude of their overall gait asymmetry. Patients with right hemisphere lesions appeared to have less ambulatory ability than patients with left hemisphere lesions.

Key words: *cadence, hemiplegia, posture, stroke, walking.*

INTRODUCTION

Impairments in posture, walking, and voluntary movements are common motor deficits in hemiparetic stroke patients. The degree of disequilibrium and the abnormalities of hemiparetic locomotion have been clinically evaluated and carefully described (1–6). A number of investigations using different approaches have shown the asymmetrical nature of hemiparetic standing and walking (7–12). Studies on standing balance in stroke patients have revealed a greater proportion of body weight distributed on the non-paretic than on the paretic limb (10,12). Stance duration during walking has been found to be relatively shorter for the affected than for the nonaffected leg along with a prolongation of the swing phase on the affected side (13–15). Furthermore, spatio-temporal parameters of gait have been shown to relate closely to the changes in walking velocity in both healthy subjects and stroke patients, suggesting that gait deficit can be classified on the basis of walking speed (16,17), although only some gait studies have used the walking speed as a parameter for evaluation of

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hemiparetic ambulation (14,16,17). Walking asymmetry has been found to decrease when reaching the maximum walking speed (18). High correlation between postural stability, locomotor functions, and functional assessments using Fugl-Meyer Scale and Barthel Index has been established (19). However, the underlying mechanisms of restoration of ambulation after stroke and the relationship between the asymmetry in hemiparetic locomotion and the speed performance are unclear.

The purpose of the present study was to describe the ability of patients with chronic unilateral stroke to ambulate at different speeds and to examine the relationship between their postural sway and temporal parameters of gait. This was investigated evaluating the foot position patterns of various self-selected walking speeds, and the location of the center of gravity and the body sway during quiet standing. To understand the relationship between the variation of walking speed and the walking asymmetry, the direction and the magnitude of asymmetry in the durations of swing and stance phases were analyzed and correlated with walking velocity. The effects of age, degree of peroneal muscle paresis, localization of the brain lesion, and the time since the onset of stroke on hemiparetic ambulation were also evaluated.

METHODS

Subjects

Twenty patients (13 men and 7 women, mean age ± SD, 57.9 ± 12.9 years, range 33–77 years) with residual hemiparesis due to stroke were studied. The inclusion criteria were: an ambulatory patient; a unilateral stroke more than 6 months ago, medically stable; and no severe hearing, visual, cognitive, or communication problems. In our population, the time since the onset of stroke varied from 8 months to 12 years (mean ± SD, 42.9 ± 37.5 months). These patients had survived unilateral cerebral infarctions in the middle cerebral artery territory confirmed by computer tomography (CT) during the acute stage of the disease. There were 13 right-sided infarctions and 7 left-sided infarctions. Clinical assessment performed for each patient at the time of the investigation revealed a hemiparesis contralateral to the ischemic lesion, upper extremity being weaker than the lower extremity. Twelve of the patients had primary motor and eight had combined motor and sensory deficits. The strength of peroneal muscle groups, known to be severely affected after stroke, was assessed by a modified Manual Muscle Test (MMT) using a 0–5 score system (20). At the time of the study, 12 of the patients were capable of independent standing although 4 of them used additional orthotic support during walking. The remaining 8 of the patients stood and walked with orthotic devices: an ankle foot orthosis and/or a cane.

Two control groups were used. Twenty right-handed healthy subjects (10 men and 10 women, mean age ± SD, 40 ± 9.4 years, range 29–67 years) were controls for posturography, and 12 right-handed healthy subjects (4 men and 8 women, mean age ± SD, 43.9 ± 10.5 years, range 30–70 years) were controls for the gait study. Seven healthy subjects were common to both investigations. The controls were free of medication and without any of the primary risk factors for cerebrovascular disease. All subjects and patients gave an informed consent prior to the studies. The Institutional Review Board for Human Research had approved the studies.

Recording methods

The posturographic study was performed on 12 stroke patients capable of independent standing (orthotic devices were removed prior to the study) and the control group. A Kistler piezoelectric force plate connected to a computer system (HP 2100) was used. All subjects were instructed to stand on the platform in a quiet, relaxed posture, with heels together and an angle of 30° between the medial aspects of the feet. The heels were 120 mm behind the center of the platform. To minimize variations in visual and vestibular input, subjects were instructed to look straight ahead at a target. The posturographic study consisted of six trials, three with eyes open (EO) and three with eyes closed (EC), each trial lasting 51.2 sec. The parameters of static posture and body sway were measured and calculated from the vertical forces exerted by the feet onto the platform. The forces were sampled at a rate of 40 Hz. The mean displacement of the center of force in sagittal and in lateral direction was determined as positive if the lateralization of the center of force was anterior (for controls and patients), or to the right side of the coordinate system (for controls). In stroke patients, a positive

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lateral displacement of the center of force indicated lateralization to the nonaffected side (NS), and a negative value indicated lateralization to the affected side (AS). The body sway was estimated by measuring the mean sagittal (Msd) and the mean lateral (Mld) displacements. Both parameters were computed in mm from the center of the coordinate system. Individual and grand mean values from all trials with eyes open and eyes closed were determined.

The gait was analyzed in 20 stroke patients and in the control group. Foot switches were used to measure the timing of foot contacts with the ground during the stride. Conductive tape was fastened bilaterally under comfortable shoes on the heel, the ball, and the toes leaving a space of 1 cm from the tip of the toe and the heel (21). The encoded patterns of foot-ground contacts were connected to a recording system through a cable following the subject via a rail in the ceiling. The subjects were asked to walk along a 10 m special metal net walkway at five self-selected different speeds, which they considered as ordinary, faster, fastest, slower, and slowest. Speed was individually selected for each subject and was repeated twice. Most patients were able to repeat their walking speeds well. Three or four strides of each walking speed were analyzed and the individual and grand mean values of the following gait variables were determined: average velocity (m/s), range of velocity (m/s) between the slowest and the fastest walking, duration of stride phase and its components; swing (s), stance (s), and the total double support (the total time when both feet are in contact with the ground during one full stride cycle in s). The velocity was calculated from the time needed for traversing the whole walkway. The time was measured from the video records. The best speed performances for each subject were analyzed. Comparisons were made between the ordinary, the slowest, and the fastest speeds.

To obtain information about the symmetric nature of the walking pattern, the temporal variables of gait were evaluated separately for the right and the left leg, and for different speeds. The asymmetry was calculated as an asymmetry index (AI) according to the formula:

$$AI = \frac{(AS - NS)}{(AS + NS)/2} \times 100$$

where AS was stance, swing, or stride duration on the affected side, NS was the same parameters on the nonaffected side. The same parameters of the left and the right legs were used for controls. The sign of the AI indicated the direction of the asymmetry, while the magnitude of this ratio indicated the degree of asymmetry.

Patients and controls were compared with analysis of covariance and repeated measures analysis of covariance using age as a covariate in order to correct for the differences in ages. Paired *t*-tests were used to compare posturographic parameters in EO and EC conditions, and to compare gait variables between both sides. Spearman correlation was performed to examine the relationship between posturographic and gait parameters.

RESULTS

First, the ambulation of healthy persons and patients as one group was evaluated. With changing walking speed temporal variables of gait changed in both groups (**Table 1**). Hemiparetic patients walked slower than controls in all gait trials due to longer stride duration ($p<0.05$). Patients' range of velocity of gait was significantly lower than controls ($p<0.001$). In both groups, total double support time decreased with increasing velocity. Hemiparetic patients had different durations of swing and stance phases between AS and NS ($p<0.001$). Their walking asymmetry is discussed in detail later.

In comparison to healthy persons, hemiparetic patients showed a tendency to increased mean lateral displacement toward the nonaffected side during quiet standing. Removal of visual input did not significantly influence posturographic parameters in either patient or in control group.

Second, the stroke patients were divided into subgroups according to (*a*) the side of the infarction and (*b*) the range of velocity between slowest and fastest walking (**Table 2**). Three patient subgroups were formed based on the velocity range: 1) ≥0.73 m/s; 2) 0.72–0.26 m/s; 3) ≤0.25 m/s. The first subgroup included patients who were able to produce velocity ranges similar to the control group. The remainder of the patients were subdivided based on their velocity range and the clinical evaluation of their walking performance. The subgroups were similar regarding their age, sex, side of infarction,

Table 1.
Mean (x) and standard deviation (SD) of temporal variables of gait and posturographic parameters in control group and in patient group.

Parameters	Trial	Control Group			Patients		
		n	Side	x ± SD	n	Side	x ± SD
Velocity (m/s)	Slowest	12		0.64 ± 0.20	20		0.40 ± 0.23
	Ordinary	12		1.18 ± 0.23	20		0.57 ± 0.34***
	Fastest	12		1.86 ± 0.36	20		0.89 ± 0.60***
	Range	12		1.22 ± 0.39	20		0.49 ± 0.41***
Swing phase (s)	Slowest	12	R	0.71 ± 0.16	20	AS	0.71 ± 0.26
		12	L	0.71 ± 0.13	20	NS	0.52 ± 0.15
		12	AI%	0.9 ± 11.1	20	AI%	29.9 ± 19.4***
	Ordinary	12	R	0.51 ± 0.05	20	AS	0.64 ± 0.22
		12	L	0.52 ± 0.05	20	NS	0.47 ± 0.12
		12	AI%	1.5 ± 3.8	20	AI%	29.2 ± 19.5***
	Fastest	12	R	0.43 ± 0.06	20	AS	0.54 ± 0.13
		12	L	0.44 ± 0.05	20	NS	0.42 ± 0.09
		12	AI%	1.1 ± 4.1	20	AI%	25.6 ± 15.9***
Stance phase (s)	Slowest	12	R	1.08 ± 0.27	20	AS	1.40 ± 0.71
		12	L	1.07 ± 0.24	20	NS	1.60 ± 0.73
		12	AI%	−0.9 ± 4.8	20	AI%	−13.9 ± 11.4**
	Ordinary	12	R	0.67 ± 0.13	20	AS	1.17 ± 0.71
		12	L	0.66 ± 0.11	20	NS	1.36 ± 0.73
		12	AI%	−0.6 ± 2.2	20	AI%	−16.2 ± 13.1***
	Fastest	12	R	0.48 ± 0.09	20	AS	0.97 ± 0.75
		12	L	0.48 ± 0.08	20	NS	1.09 ± 0.78
		12	AI%	−0.7 ± 4.6	20	AI%	−14.5 ± 8.8***
Total double support (s)	Slowest	12		0.37 ± 0.23	20		0.89 ± 0.75
	Ordinary	12		0.15 ± 0.12	20		0.72 ± 0.75
	Fastest	12		0.05 ± 0.07	20		0.55 ± 0.75
Mld (mm)	EO	20		−4.4 ± 4.3	12		10.3 ± 12.9
	EC	20		−3.9 ± 5.8	12		9.8 ± 14.3
Msd (mm)	EO	20		−17.2 ± 17.9	12		−14.1 ± 11.2
	EC	20		−16.4 ± 17.8	12		−14.7 ± 13.9

L, left; R, right; AS, affected side; NS, nonaffected side; Mld, mean lateral displacement; Msd, mean sagittal displacement; EO, eyes open; EC, eyes closed; ** $p<0.01$, *** $p<0.001$, significance between controls and patients (ANCOVA after adjustment for age).

and the time since the onset of stroke. The subgroup with lowest velocity range consisted of slightly older patients and significantly lower MMT score in comparison to the patients with highest velocity range. The pure motor deficit was predominant in patients with left-sided infarction (77.8 percent). The use of orthotic devices was more common among the patients with a small velocity range. Individual patients utilized their range of velocity differently. For example, some good walkers, patients with velocity ranges similar to normals (>0.73 ms/s), utilized only 18–38 percent of their range of velocity in achieving an ordinary walking speed, preserving the rest of the velocity range for fast walking. This was also seen in the control group. The worst walkers from the group with smallest velocity range utilized almost all of their range of velocity to achieve their ordinary walking speed.

Comparing the two sides, a prolonged swing on the AS and a prolonged stance on the NS were observed in all subgroups although there was variability in the duration of the stride phases (**Figure 1**). During quiet standing, all patient subgroups were found to favor their nonaffected leg; however, some quantitative differences were observed among them (**Figure 2**). The changes in postural sway and gait

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Table 2.
Characteristics of patient subgroups.

Subgroups	Total n	Men n	Women n	Age x ± SD	Months after stroke x ± SD	Manual Muscle Test x ± SD	Orthotic Devices n	% [a]
Side of infarction								
Left	7	5	2	58.4 ± 10.5	49.7 ± 41.2	2.9 ± 1.8	4	57%
Right	13	8	5	57.6 ± 13.6	39.3 ± 34.8	2.5 ± 1.5	8	62%
Range of velocity								
≥0.73 m/s	4	4	0	53.5 ± 10.1	56.7 ± 49.5	4.0 ± 1.0	1	25%
0.72 – 0.26 m/s	10	6	4	55.6 ± 10.5	46.1 ± 37.9	3.1 ± 1.4	6	60%
≤ 0.25 m/s	6	3	3	64.6 ± 14.6	28.5 ± 17.4	1.8 ± 1.1 [b]	5	83%
Total	20	13	7	57.9 ± 12.6	42.9 ± 37.5	2.8 ± 1.5	12	60%

[a] The percentage of orthotic devices was calculated from the total number of patients of each group.
[b] $p<0.05$, significance between the subgroups with highest and lowest range of velocity (ANOVA followed by multiple comparisons).

variables were more pronounced in the subgroup with right-sided infarction. The subgroup with lowest range of velocity showed a significantly longer stance duration during all speed trials in comparison to other subgroups.

The magnitude of asymmetry in duration of stride phases was analyzed comparing left and right sides in controls, and the AS and the NS in patients. Healthy persons had nearly symmetrical swing and stance durations, which contrasted with marked asymmetries in the patient group ($p<0.03$). This difference between controls and patients was not associated with age. When the swing AI increased, the stance AI also increased but toward the other side (**Figure 3**). The sign in **Figure 3** indicates the direction of asymmetry; for instance, if swing and stance AI both were negative, the patient would lean continuously to one side during walking.

The asymmetries were remarkably present in all patient subgroups regardless of the speed trial. The patients with right-sided infarction had somewhat higher AI in stride phases than the subgroup with left-sided infarction (**Figure 4**). The asymmetries persisted also in patients who were able to produce a range of velocity similar to that of the controls.

In healthy persons, no relationship between velocity and asymmetry indices were found. Hemiparetic patients with high swing AI produced low ordinary ($r=-0.57$, $p<0.01$) and fastest ($r=-0.46$, $p<0.05$) velocities. However, the ability of patients to produce a range of velocities was not associated with the range of asymmetry indices calculated from their speed trials (**Figure 5**).

To understand the relationship between postural sway and ambulation in chronic stroke, we searched for correlations between posturographic and gait parameters. No significant relationship was found between gait and posture in the control group. In hemiparetic patients, the increase in the mean lateral displacement was associated with lower fastest velocity ($r=-0.68$, $p<0.05$). The larger mean sagittal displacement was associated with increasing stance AI during slowest walking ($r=0.77$, $p<0.01$). These relationships were present in both EO and EC conditions.

We analyzed the impact of other factors on the ambulation parameters in our patients and found that the site of the lesion and the time after the onset of stroke were insignificant.

DISCUSSION

The present study reveals that patients with residual hemiparesis due to chronic unilateral supratentorial ischemic infarction walk slower, more asymmetrically, and sway more laterally favoring their nonaffected leg, than healthy persons. Similar patterns of standing and locomotion were observed previously in hemiparetic patients, some of them

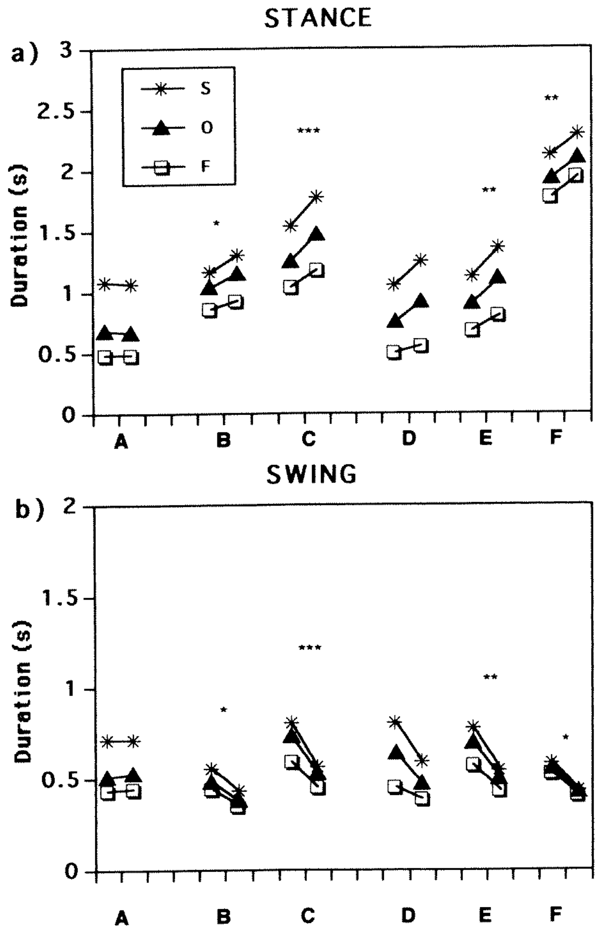

Figure 1.
Mean durations of stance (a) and swing (b) phases of gait obtained during slowest (s), ordinary (o), and fastest (f) speeds in control group (A) and patient subgroups with left-sided infarction (B), right-sided infarction (C), and different ranges of walking velocity: ≥0.73 m/s (D), 0.72–0.26 m/s (E), and ≤0.26 m/s (F). The data from different speeds are shown above each other in the respective order. Horizontal lines connect right and left leg values in controls, and the values of affected (AS) and nonaffected (NS) sides in patients. Significant differences were found between AS and NS, * $p<0.05$, ** $p<0.01$, *** $p<0.001$.

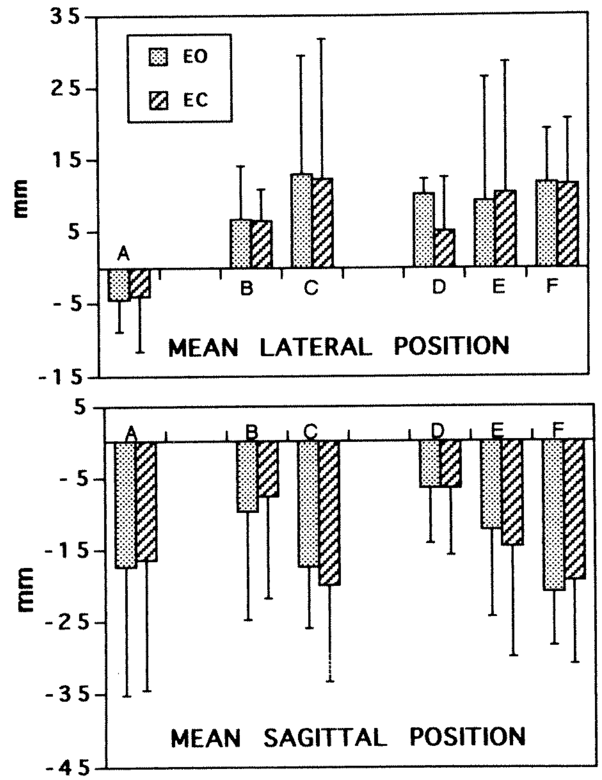

Figure 2.
Mean ± SD of the mean lateral (Mld) and the mean sagittal (Msd) displacement in control group (A, n = 12), and patient subgroups with left-sided infarction (B, n = 5), right-sided infarction (C, n = 7), and in the subgroups according to the range of walking velocity obtained by patients: ≥ 0.73 m/s (D, n = 2); 0.72–0.26 m/s (E, n = 6); and ≤0.26 m/s (F, n = 4). EO refers to eyes open condition and EC refers to eyes closed condition.

with different types and locations of stroke (10,12,19). The balance disturbances and the alterations of gait varied among the patients: they were associated with the degree of peroneal muscle paresis (evaluated by MMT) and were more pronounced in the subgroup with right-sided infarction and those with a small range of walking velocity. The differences among the patients were found quantitative rather than qualitative in nature. The foot-ground contact patterns of the hemiparetic gait changed in a stereotyped manner, resulting in a prolonged swing duration on the AS and a prolonged stance duration on the NS. Changes in the walking speed were indicative of changes in stance, swing and total double support time confirming previous findings (16,17).

The major emphasis of this study was the walking asymmetry and its impact in speed performance. Previously, asymmetries in durations of swing and stance phases were expressed as a simple ratio between affected and nonaffected sides or as a percentage of each phase from the duration of the whole stride (7,8,11). In addition, Wall and Turnbull (10) evaluated the pattern of gait in patients with residual hemiplegia using an asymmetry ratio between the single support time of AS and NS. However, all these studies did not evaluate the

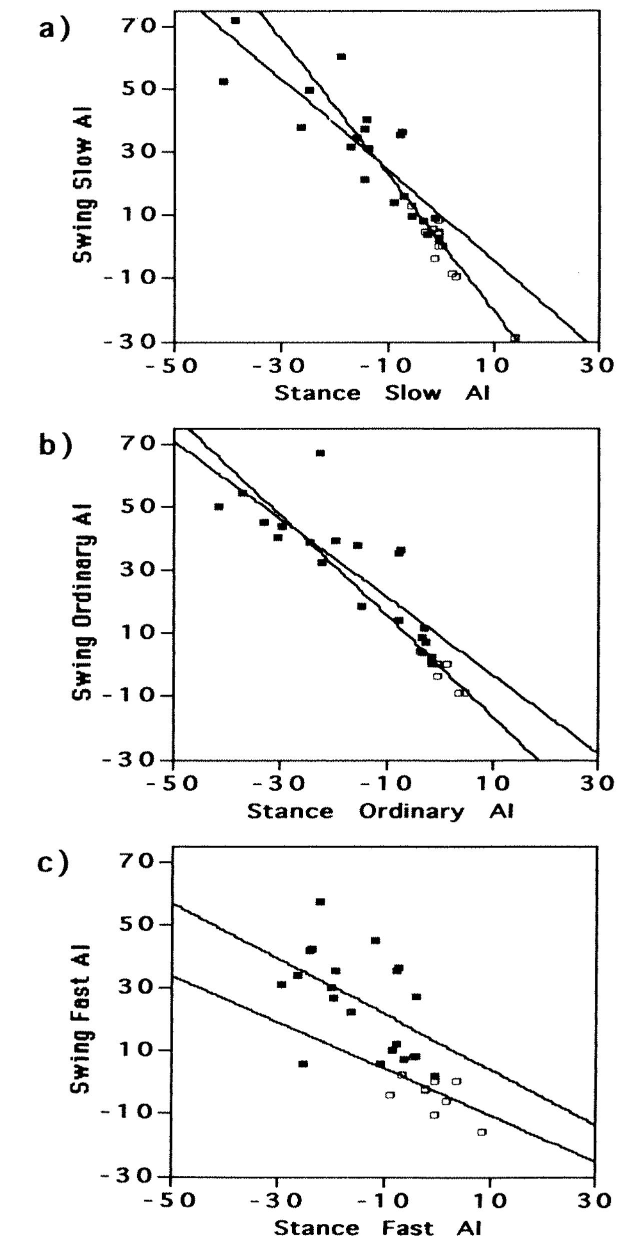

Figure 3.
Relationship between swing and stance asymmetry indices at slowest (a), ordinary (b), and fastest (c) speeds in control subjects (empty squares) and all patients (filled squares).

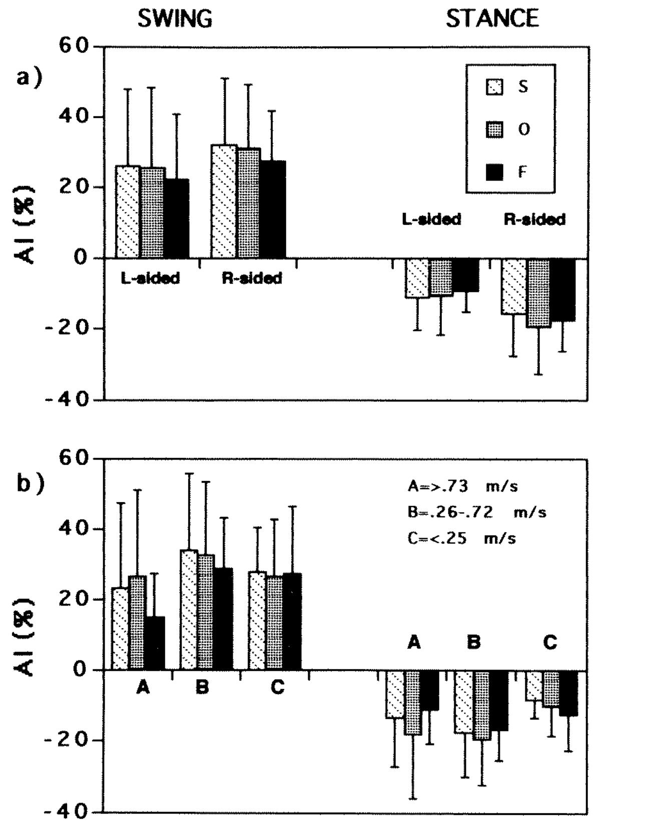

Figure 4.
Asymmetry index (AI, mean ± SD) of durations of swing and stance phases in patient subgroups with left-sided and right-sided infarction (a), and patient subgroups with different ranges of walking velocity (b). The positive values of Y-axis indicate a direction of AI toward the affected side; the negative values indicate the direction of AI toward the nonaffected side; s = slowest walking speed; o = ordinary walking speed; f = fastest walking speed.

magnitude of asymmetry and the question if asymmetry is related to speed performance. The present study indicates that the asymmetries in stride phases were present in all patient subgroups regardless of their speed performance. Also, the magnitude of asymmetry in hemiparetic ambulation was significantly higher than in controls. Large swing asymmetry indicated slower overall walking velocity. However, the ability of patients with stroke to ambulate using various self-selected walking speeds was not related to the highest number of walking asymmetry. This was demonstrated by patients who had a large swing asymmetry but were able to ambulate with a number of speeds using lower velocities. Increased postural stability, increased muscle strength of the paretic limb, and increased symmetry of walking contributed for shifting the velocity performance to higher velocity level, although the range of velocity may remain unaltered.

A more symmetrical weightbearing in hemiplegia is known to be related to greater ambulatory independence (22,23). Our study demonstrates that the hemiparetic postural sway during quiet standing, even in those patients who had a walking

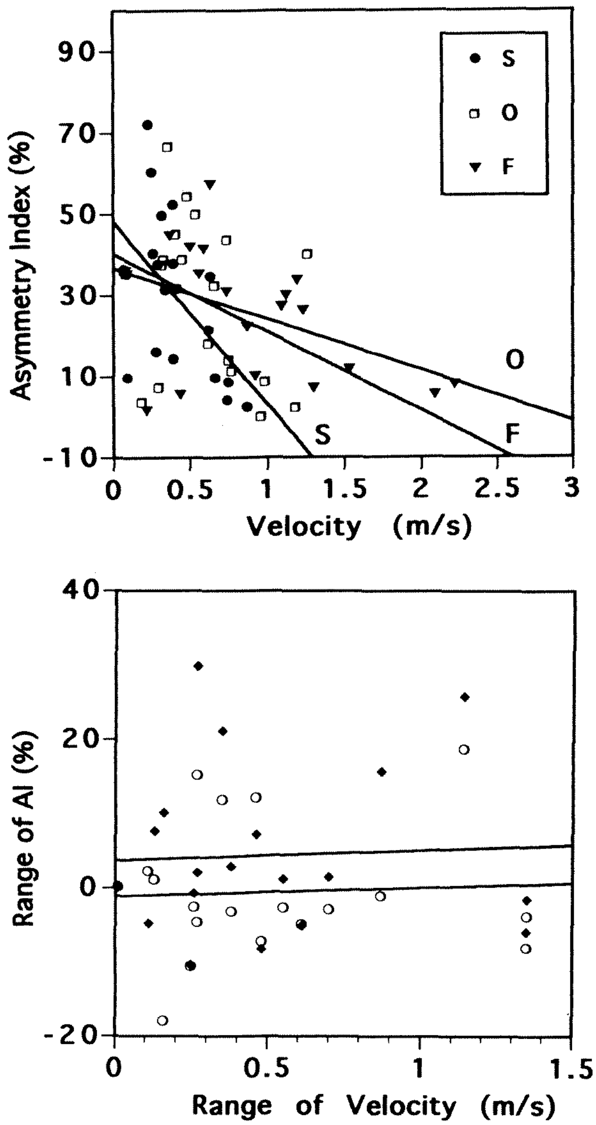

Figure 5.
The relationship between swing asymmetry index and walking velocity of the hemiparetic patients in slowest (s), ordinary (o) and fastest (f) speeds at the top. The relationship between the range of asymmetry indices and the range of velocities of each patient at the bottom.

velocity range close to that in healthy persons, was reflected in interlimb coordination and in the symmetry of limb movements during walking. The larger mean lateral sway toward the NS was related to slower fastest walking speed, while the larger sagittal backward sway was related to increased stance phase asymmetry during slowest walking. Control subjects showed nearly symmetrical patterns of walking during all speeds and no association between posturographic and gait parameters. These findings suggest a strong affiliation between postural sway and hemiparetic ambulation and the increase in mean lateral sway during quiet standing of hemiparetic patients may be an indication for the restricted velocity performance during walking.

The effect of lesion site on the postural stability and gait after stroke is difficult to judge in our relatively small patient group. However, a tendency to increased postural sway and gait abnormalities in the subgroup with right-sided infarction was found compared to the left-sided infarction group. Although 62 percent of the right-sided infarction group used additional support of orthotic devices, known to improve walking performance (24–26), these patients tended to have higher postural sway, and walk more slowly and more asymmetrically than did the patients with left-sided infarction. Previous studies on the effects of lateralized cerebral damage have shown that right hemisphere lesions are associated with greater sensorimotor deficits than left hemisphere lesions (27).

The neurobiological basis of spontaneous recovery after stroke, and particularly the restoration of the ambulatory function, are not completely understood (28). The greater part of walking recovery after a stroke occurs within the first 3–6 months (29), although recovery can continue over a period of years in some patients (30). The present study shows that the hemiparetic postural stability and gait changes occur in a stereotyped manner. One explanation for this behavior may be related to the preserved central pattern generators in the spinal cord (31) which may operate in a stereotyped manner under the residual descending supraspinal influence. The ability of hemiparetic patients to ambulate with different walking speeds is a complex phenomenon that results from the relationships between motor functions, morphological lesions, and structural and functional reorganization of the brain, and is associated with the recovery after stroke (32,33).

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REFERENCES

1. Bobath B. Adult hemiplegia: evaluation and treatment. London: Heinemann Medical Books, 1978.
2. Brunnstrom S. Recording gait pattern of adult hemiplegic patients. J Am Phys Ther Assoc 1964:44:11-8.
3. Dimitrijevic MR, Faganel J, Sherwood AM, McKay BW. Activation of paralysed leg flexor and extensors during gait in patients after stroke. Scand J Rehabil Med 1981:13:109-15.
4. Marks M, Hirschberg G. Analysis of the hemiparetic gait. Ann NY Acad Sci 1958:74:59-77.
5. Perry J. The mechanics of walking in hemiplegia. Clin Orthop 1969:63:23-31.
6. Twitchell TE. The restoration of motor function following hemiplegia in man. Brain 1951:54:443-81.
7. Brandstater ME, deBruin H, Gowland C, Clark BM. Hemiplegic gait: analysis of temporal variables. Arch Phys Med Rehabil 1983:64:583-7.
8. Dewar ME, Judge G. Temporal asymmetry as gait quality indicator. Med Biol Eng Comput 1980:18:689-93.
9. Sackley CM. Falls, sway, and asymmetry of weight-bearing after stroke. Int Disab Studies 1991:13:1-4.
10. Wall JC, Turnbull GI. Gait asymmetries in residual hemiplegia. Arch Phys Med Rehabil 1986:67:550-3.
11. Wall J, Ashburn A. Assessment of gait disability in hemiplegics. Scand J Rehabil Med 1979:11:95-103.
12. Winstein CJ, Gardner MS, McNeal DR, Barto PS, Nicholson DE. Standing balance training: effect on balance and locomotion in hemiparetic adults. Arch Phys Med Rehabil 1989:70:755-62.
13. Bohannon RW, Larkin PA. Lower extremity bearing under various standing conditions in independently ambulatory patients with hemiparesis. Phys Ther 1985:65:1323-5.
14. Drillis R. Quantitative recording and biomechanics of pathological gait. Ann NY Acad Sci 1958 1981:74:86-109.
15. Seliktar R, Susak Z, Najenson T, Solzi P. Dynamic features of standing and their correlation with neurological disorders. Scand J Rehabil Med 1978:10:59-64.
16. Andriacchi TP, Ogle JA, Galante JO. Walking speed as a basis for normal and abnormal gait measurements. J Biomech 1977:10:261-8.
17. Wagenaar RC, Beek WJ. Hemiplegic gait: a kinematic analysis using walking speed as a basis. J Biomech 1992:25:1007-15.
18. Wagenaar RC. Functional recovery after stroke. Amsterdam: University Press, 1990.
19. Dettmann MA, Linder MT, Sepic SB. Relationships among walking performance, postural stability, and functional assessments of the hemiplegic patients. Am J Phys Med 1987:66:77-90.
20. Mendell JR, Florence J. Manual muscle testing. Muscle Nerve 1990:13:S16-S20.
21. Norlin R. Corrective surgery and gait analysis in cerebral-palsied children. Linköping University Medical Dissertations, Linköping, Sweden, 1984:187.
22. Caldwell C, MacDonnald D, MacNiel K, McFarland K, Turnbull G, Wall F. Symmetry of weight distribution in normals and stroke patients using digital weight scale. Physiother Pract 1986:2:109-16.
23. Gruendel TM. Relationship between weight-bearing characteristics in standing and ambulatory independence in hemiplegics. Physiother Can 1992:44:16-7.
24. Lehmann JF. Biomechanics of ankle-foot orthosis. Prescription and design. Arch Phys Med Rehabil 1979:60:200-7.
25. Lehmann JF, Conton SM, Price R, deLateur BJ. Gait abnormalities in hemiplegia: their correction by ankle-foot orthosis. Arch Phys Med Rehabil 1987:68:763-71.
26. Nakamura R. Recovery of gait in hemiparetic stroke patients with reference to training program. In: Shimamura M, ed. Neurobiological basis of human locomotion. Tokyo: Japan Scientific Societies Press, 1991:425-35.
27. Hom J, Reitan RM. Effect of lateralized cerebral damage upon contralateral and ipsilateral sensorimotor performances. J Clin Neuropsychol 1982:4:249-68.
28. Goldstein LB, Davis JN. Restorative neurology. Drugs and recovery following stroke. Stroke 1990:25:19-24.
29. Skilback CE, Wade DT, Hewer RL, Wood VA. Recovery after stroke. J Neurol Neurosurg Psychiatry 1983:46:5-8.
30. Bach-y-Rita P. Central neuron system lesions: sprouting and unmasking in rehabilitation. Arch Phys Med Rehabil 1981:62:413-7.
31. Brooks VB. Locomotion. In: The neuronal basis of motor control. New York: Oxford University Press, 1986:181-201.
32. Chollet F, DiPiero V, Wise RJS, Brooks DJ, Dolan RJ, Frackowiak RSJ. The functional anatomy of motor recovery after stroke in humans: a study with positron emission tomography. Ann Neurol 1991:29:63-71.
33. Weiller C, Chollet F, Friston KJ, Wise RJS, Frackowiak RSJ. Functional reorganization of the brain in recovery from striatocapsular infarction in man. Ann Neurol 1992:31:463-72.