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Post-stroke hemiparesis: Does chronicity, etiology, and lesion side are associated with gait pattern?

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ABSTRACT

Background: Studies that evaluate gait rehabilitation programs for individuals with stroke often consider time since stroke of more than six months. In addition, most of these studies do not use lesion etiology or affected cerebral hemisphere as study factors. However, it is unknown whether these factors are associated with post-stroke motor performance after the spontaneous recovery period.

Objective: To investigate whether time since stroke onset, etiology, and lesion side is associated with spatiotemporal and angular gait parameters of individuals with chronic stroke.

Methods: Fifty individuals with chronic hemiparesis (20 women) were evaluated. The sample was stratified according to time since stroke (between 6 and 12 months, between 13 and 36 months, and over 36 months), affected cerebral hemisphere (left or right) and lesion etiology (ischemic and hemorrhagic). The participants were evaluated during overground walking at self-selected gait speed, and spatiotemporal and angular gait parameters were calculated.

Results Differences between gait speed, stride length, hip flexion, and knee flexion were observed in subgroups stratified based on lesion etiology. Survivors of a hemorrhagic stroke exhibited more severe gait impairment. Subgroups stratified based on time since stroke only showed intergroup differences for stride length, and subgroups stratified based on affected cerebral hemisphere displayed between-group differences for swing time symmetry ratio.

Conclusion: In order to recruit a more homogeneous sample, more accurate results were obtained and an appropriate rehabilitation program was offered, researchers and clinicians should consider that gait pattern might be associated with time since stroke, affected cerebral hemisphere and lesion etiology.

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Introduction

Stroke is the leading cause of chronic disability worldwide[1–3] and individuals with stroke usually consider improvements in walking function as their main goal in rehabilitation.[4] However, despite substantial gait recovery in the first months after the brain injury, gait abnormalities persist in a large percentage of these individuals.[4]

Motor recovery after a stroke is the result of spontaneous mechanisms and motor learning.[5–7] Spontaneous recovery occurs in the first month after the lesion and reaches a plateau between three and six months (spontaneous recovery period).[8,9] Motor learning is not limited by time and is associated with the individual's daily motor performance and rehabilitation programs prescribed after the stroke.[10] These mechanisms may occur simultaneously enhancing motor recovery in the early months after stroke.[10]

Studies that evaluate the effectiveness of gait rehabilitation programs for individuals with stroke often consider time since stroke onset of more than six months,[11–13] due to the difficulty in distinguishing whether the improvement observed after intervention results from spontaneous mechanisms or motor learning.[14] In addition, most of these studies do not specify a maximum time since stroke as a factor to investigate,[15–17] and often do not use lesion etiology or the affected cerebral hemisphere as inclusion criteria. Research that does not consider these facts could increase sample diversity – which could influence treatment effects[18–20] and limit their application in clinical environments.

Considering the initial motor impairment during the acute and subacute phase, individuals who experienced hemorrhagic stroke have been shown to exhibit better motor recovery than those who suffer from ischemic stroke.[21,22] This appears to be associated with the reabsorption caused by hemorrhagic stroke as well as resolution of ischemia in the penumbra area that surrounds the hematoma.[22–24] In regard to the affected cerebral hemisphere, individuals with right hemisphere damage are able to achieve standing balance quicker in the first months,[25] and better mobility levels[26] when compared to those with damage to the left hemisphere. However, to date is unknown whether these factors are associated with

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post-stroke motor performance after the spontaneous recovery period. Gait analysis in this population is particularly important, since gait abnormalities are one of the most limiting factors in the execution of daily activities after a stroke, restricting independence, and social life more than six months after stroke onset.

Earlier studies[27,28] investigated the association between lesion characteristics and gait pattern in individuals with stroke, however, they assessed those with time since stroke of up to six months and investigate gait parameters, such as foot contact,[27] endurance,[28] and gait velocity. Moreover, these studies used costly techniques to identify lesion characteristics, such as computer tomography[27] or magnetic resonance imaging[28] procedures that are not commonly applied in low- and middle-income countries. These factors could limit extrapolating these results to a clinical environment.

The aim of this study was to investigate whether time since stroke onset, etiology, and lesion side are associated with the spatio-temporal and angular gait parameters of individuals with chronic stroke. We hypothesized that gait parameters may vary among stroke subgroups even more than six months after stroke onset. Understanding this diversity may help researchers in determine inclusion/exclusion criteria seeking interindividual variability, reduced sample heterogeneity, and more accuracy results. It could help clinicians select gait intervention target specificities of each subgroup. Furthermore, it can assist both clinicians and researchers to better interpret quantitative gait data, given that gait pattern may exhibit differences according with lesion characteristics even after spontaneous period recovery.

Method

The study was conducted in accordance with the Declaration of Helsinki and was approved by Research Ethics Committee of Onofre Lopes Universitary Hospital (Protocol number 0364. 0. 000. 294 - 11). All participants gave their written informed consent. STROBE statement recommendations were followed for the methodological design of the study.

Sample characterization

The sample consisted of 50 individuals with chronic unilateral hemiparesis after an ischemic or hemorrhagic (more than six months with the lesion), stroke recruited by non – probability convenience sampling. Sample size was determined from the number of individuals who agreed to participate and met the eligibility criteria.

The following inclusion criteria were adopted: (1) time since stroke onset of more than six months; (2) single stroke episode; (3) absence of orthopedic or pulmonary pathologies, or other neurological impairment that could compromise gait; and (4) walking ability between Functional Ambulatory Category (FAC)[29] levels 3 and 5.

Assessment procedures

Anthropometric, demographic, and clinical data such as age, time since stroke onset, lesion etiology, affected cerebral hemisphere, gait ability FAC,[29] classification of neurological status according to the National Institutes of Health Stroke Scale – NIHSS[30,31] and gait assistive device use were collected from the participants.

Gait analysis was performed using the Qualisys Motion Capture System® (Qualisys Medical AB, 411 13 Gothenburg, Sweden) with eight infrared cameras (Qualisys Oqus 300) interconnected in series for data acquisition during gait assessment at frequency of 120 Hz.

To that end, spherical passive reflective markers between 15- and 19-mm wide were placed on the anterior superior illiac crest, greater trochanter, lateral and medial epicondyle, medial and lateral malleolus, first metatarsal head, fifth metatarsal head, and calcaneus (reference markers). Moreover, tracking markers were placed no collinearly in clusters attached to the middle third of the lateral surface of the thighs, shank, and at the base of the sacrum between the posterior iliac spines. The reference markers of the malleolus, calcaneus, and fifth metatarsal head were also considered tracking markers.

Static collection was carried out to identify the segments, with participants in the orthostatic position for 5 s. Next, reference markers were removed to perform dynamic collections, where subjects were instructed to walk at a comfortable, self-selected speed along an eight-meter walkway. They wore their usual footwear, and used assistive device, if necessary. This task was repeated until 10 gait cycles were recorded by the camera capture system.[12,32]

Data reduction

Data were collected using Qualisys Track Manager 2.6 software, and processing was conducted with Visual 3D software (Visual 3D Standard; C-Motion), whereby the participants' biomechanical model was built based on the coordinates (x, y, z) of the reference markers. Dynamic collections were then associated with the biomechanical model, to determine spatio-temporal variables and angular displacement of the hip, knee, and ankle during each trial.

A low-pass Butterworth filter with a frequency of 6 Hz was used to eliminate the noise created by the movement of markers during data collection. Angular displacements of the hip, knee, and ankle were analyzed. To delineate the gait cycles, four consecutive initial contact (IC) were observed; two performed with the paretic foot (P) and two with the non-paretic foot (NP). The final period of foot contact during the stance phase of gait (toe off) was also determined.

The following outcomes were analyzed: gait speed (m/s), cadence (steps/min), stride length (m), paretic limb (P), and non-paretic limb (NP) single support time (% gait cycle), double support time (s), P and NP swing time (% gait cycle), step length symmetry ratio and simple support time symmetry ratio both calculated as the ratio between paretic and non-paretic limbs parameters,[33] maximum hip flexion (°), maximum hip extension (°), maximum knee flexion (°), and maximum ankle dorsiflexion (°).

Statistical analysis

Subgroups were stratified based on lesion etiology (G_I = ischemic and G_H = hemorrhagic); affected cerebral hemisphere (G_R = right or G_L = left), and time since stroke (G_1 = between 6 and 12 months; G_2 = between 13 and 36 months; and G_3 = over 36 months). Subgroups based on etiology and affected cerebral hemisphere were compared using independent samples t-tests and subgroups

Table 1. General characteristics of the individuals.

Characteristics	Mean	± SD	*N*	%
Age (years)	55.68	8.59		
Time since stroke (months)	32.5	26.47		
Height (m)	1.63	0.07		
Weight (kg)	70.74	11.85		
FAC	4.32	0.55		
NIHSS	3.92	2.47		
Sex				
Female			20	40
Male			30	60
Lesion etioly				
Ischemic			39	78
Hemorrhagic			11	22
Cerebral hemisphere affected				
Left			18	36
Right			32	64
Gait devices use				
No			18	36
Yes			32	64

Note: SD = standard deviation, m = meters, kg = kilogram, FAC = Functional Ambulatory Category, NIHSS = National Institutes of Health Stroke Scale.

based on time since stroke were compared applying one-way analysis of variance (ANOVA). When ANOVA was significant, Bonferroni adjustments for multiple comparisons were used to identify differences between the subgroups.

Multiple linear regression analysis was conducted to identify potential predictors of spatio-temporal and angular gait parameters, adjusting the models for age and gender. An alpha level of 0.05 was adopted for all statistical tests, which were conducted using SPSS 17.0 software.

Results

The sample was composed of 50 individuals with chronic hemiparesis, aged between 40 and 70 years (55.68 ± 8.59), and time since stroke between 6 and 144 months (32.5 ± 26.47). The clinical, demographic, and anthropometric characteristics of the participants are described in Table 1.

A comparison of subgroups, stratified based on lesion etiology (G_H = 11 and G_I = 39), *t*-tests, revealed intergroup differences in speed (p = 0.006, statistical power = 0.96), stride length (p = 0.017, statistical power = 0.87), NP stance and swing time (p = 0.001, statistical power = 0.94 and 0.95, respectively), maximum hip flexion (p = 0.021, statistical power = 0.69), and maximum knee flexion (p = 0.004, statistical power = 0.82). Survivors of a hemorrhagic stroke exhibited more severe gait disorders.

The subgroups stratified based on affected cerebral hemisphere (G_L = 18 and G_R = 32) showed differences between subgroups only in swing time symmetry ratio (p = 0.016, statistical power = 0.71) (Table 2); individuals with left hemisphere damage had a greater asymmetry. ANOVA compared subgroups stratified based on time since stroke (G_1 = 10, G_2 = 26 and G_3 = 14), revealing differences between subgroups for stride length (p = 0.049), Bonferroni adjustments for multiple comparisons showed a trend toward greater stride length in G_3, albeit, not statistically significant (p = 0.062, statistical power = 0.90) (Table 3).

Multiple linear regression analysis results are shown in Table 4. Lesion etiology was associated with the largest number of gait indicators, according to B indicator analysis, and having suffered a hemorrhagic stroke meant worse results. The affected hemisphere and time since stroke showed no association with the gait parameters analyzed.

Discussion

This study investigated whether time since stroke onset, etiology and lesion side can are associated with spatio-temporal and angular gait parameters of individuals with chronic hemiparesis. The results show that primarily lesion etiology can be associated with some of the gait parameters of individuals with chronic hemiparesis during overground walking at a self-selected speed. Individuals who survived hemorrhagic stroke showed reduced speed, stride length, maximum hip flexion, and maximum knee flexion. In addition, those with time since stroke of more than 36 months had a longer stride length and subjects who exhibited a

Table 2. Comparison between subgroups stratified based on lesion etiology (GI and GH) and *cerebral hemisphere affected (GL and GR)*.

	Lesion etiology subgroups			*Affected cerebral hemisphere subgroups*		
	Ischemic	Hemorrhagic		Left	Right	
Outcomes	G_I (N = 39)	G_H (N = 11)	*P**	G_L (N = 18)	G_R (N = 32)	*P**
Age (years)	55.02 ± 8.81	58.0 ± 7.65	0.315	53.44 ± 9.09	56.93 ± 8.16	0.170
Time since stroke (months)	29.61 ± 20.26	42.73 ± 41.55	0.332	23.27 ± 17.23	37.68 ± 29.46	0.064
Speed (m/s)	0.49 ± 0.15	0.35 ± 0.12	0.006§	0.49 ± 0. 17	0.44 ± 0.48	0.378
Cadence (steps/min)	163.73 ± 30.23	147.46 ± 35.74	0.136	170.39 ± 37.77	154.39 ± 26.98	0.088
Stride length (m)	0.76 ± 0.19	0.59 ± 0.16	0.017§	0. 73 ± 0.18	0.71 ± 0.2	0.847
Double-Stance time (s)	0.61 ± 0.22	0.90 ± 0.57	0.014	0.62 ± 0. 29	0.70 ± 0. 37	0.336
P Stance time (% gait cycle)	63.411 ± 4.471	66.82 ± 6.43	0.124	63.25 ± 4.76	64.67 ± 5.28	0.351
NP stance time (% gait cycle)	74.38 ± 4.59	80.33 ± 5.52	0.001§	75.43 ± 5.96	75.83 ± 5.09	0.804
P swing time (% gait cycle)	36.59 ± 4.47	33.18 ± 6.43	0.124	36.74 ± 4.76	35.33 ± 5.28	0.351
NP swing time (% gait cycle)	25.62 ± 4.59	19.67 ± 5.52	0.001§	24.56 ± 5.96	24.16 ± 5.09	0.804
Maximum hip flexion (°)	20.17 ± 9.56	11.92 ± 11.88	0.021§	18.72 ± 8.62	18.14 ± 11.64	0.856
Maximum hip extension (°)	−5.18 ± 10.68	−10.41 ± 9.09	0.146	−6.45 ± 8.89	−6.25 ± 11.43	0.949
Maximum knee flexion (°)	37.28 ± 14.43	20.923 ± 19.66	0.004§	33.09 ± 14.58	34.01 ± 18.36	0.856
Ankle dorsiflexion (°)	−2.78 ± 7.26	−1.68 ± 6.78	0.655	−3.57 ± 6.09	−1.95 ± 7.63	0.443
Swing time symmetry ratio	1.35 ± 0.36	1.49 ± 0.59	0.323	1.6 ± 0.4	1.3 ± 0.4	0.016§
Step length symmetry ratio	1.28 ± 0.46	1.27 ± 0.26	0.971	1.3 ± 0.6	1.3 ± 0.3	0.77

Note: P = paretic, NP = nonparetic, m = meters, min = minutes, s = seconds, °=degrees.
§$p < 0.05$.
*Student's *t*-test for independent samples.

Table 3. Comparison between groups based on time since stroke.

	Groups based on time since stroke (months)			
	6–12	13–36	> 36	
Outcomes	G_I ($N = 10$)	G_{II} ($N = 26$)	G_{III} ($N = 14$)	P*
Age (years)	49.5 ± 10.21	57.07 ± 8.73	57.50 ± 4.73	0.035
Speed (m/s)	0.43 ± 0.11	0.43 ± 0.17	0.53 ± 0.13	0.129
Cadence (steps/min)	172.47 ± 26.47	153.92 ± 36.59	162.92 ± 23.78	0.279
Stride length (m)	0.64 ± 0.16	0.69 ± 0.19	0.82 ± 0.20	0.049§
Double-stance time (s)	0.61 ± 0.14	0.74 ± 0.44	0.58 ± 0.21	0.288
P stance time (% gait cycle)	64.82 ± 3.54	65.06 ± 5.38	62 ± 5.17	0.177
NP stance time (% gait cycle)	75.95 ± 2.45	76.00 ± 6.16	74.91 ± 5.51	0.821
P swing time (% gait cycle)	35.17 ± 3.54	34.93 ± 5.38	37.99 ± 5.17	0.177
NP swing time (% gait cycle)	24.04 ± 2.45	23.99 ± 6.16	25.08 ± 5.51	0.821
Maximum hip flexion (°)	19.89 ± 10.55	18.00±9.57	17.88 ± 12.84	0.879
Maximum hip extension (°)	−4.66 ± 12.93	−7.19 ± 10.31	−5.91 ± 9.52	0.805
Maximum knee flexion (°)	32.82 ± 12.90	33.93 ± 14.74	33.83 ± 23.43	0.965
Ankle dorsiflexion (°)	−2.29 ± 7.82	−2.80 ± 7.94	−2.21 ± 5.10	0.964
Swing time symmetry ratio	1.46 ± 0.26	1.42 ± 0.41	1.25 ± 0.52	0.401
Step length symmetry ratio	1.54 ± 0.77	1.24 ± 0.25	1.16 ± 0.25	0.07

Note: P = paretic, NP = nonparetic, m = meters, min = minutes, s = seconds.
§$p < 0.05$.
*ANOVA *one way.*

Table 4. Linear regression results for the gait outcomes based on time since stroke onset, side, and etiology of lesion.

	Lesion etiology		Affected cerebral hemisphere		Time since stroke (months)			
	Ischemic stroke		Left stroke		6–12		13–36	
Gait outcomes*	*B*	*p*	*B*	*p*	*B*	*p*	*B*	*p*
Speed (ms)	0.13	0.03	0.029	0.54	−0.071	0.29	−0.084	0.11
Cadence (steps/min)	8.47	0.46	11.21	0.25	−2.29	0.87	−19.6	0.07
Stride length (m)	0.16	0.03	−0.005	0.93	−0.078	0.37	−0.057	0.39
Double-stance time (s)	−0.23	0.13	0.01	0.94	−0.05	0.78	0.18	0.20
P stance time (% gait cycle)	−2.44	0.30	0.44	0.825	−0.76	0.79	0.76	0.73
P swing time (% gait cycle)	2.44	0.30	−0.44	0.82	0.76	0.79	−0.76	0.73
NP stance time (% gait cycle)	−5.53	0.02	1.86	0.33	−0.84	0.76	2.07	0.32
NP swing time (% gait cycle)	5.53	0.02	−1.86	0.33	0.84	0.75	−2.07	0.32
Maximum hip flexion (°)	7.37	0.04	−0.11	0.97	−2.65	0.54	−3.77	0.27
Maximum hip extension (°)	5.85	0.15	0.93	0.79	−2.53	0.68	−4.63	0.22
Maximum knee flexion (°)	14.61	0.01	−4.17	0.37	−3.04	0.65	−2.07	0.66
Ankle dorsiflexion (°)	−0.87	0.73	−1.65	0.45	0.62	0.84	−0.30	0.9

Note: *Adjusted for age and sex.

left cerebral lesion walked more asymmetrically in terms of swing time. As such, the hypothesis that analyzed gait parameters may vary between subgroups was partially confirmed based primarily on the lesion etiology of the subgroup.

Hemorrhagic stroke have been associated with higher mortality rates, in both acute phase[34] and chronic phase,[35] and more severe functional limitations and gait impairments in the acute phase[36] compared to ischemic stroke, although there is a higher prevalence of the latter.[37,38] Although the literature describes different clinical outcomes between ischemic and hemorrhagic stroke survivors, to the best of our knowledge this is the first study to examine whether lesion etiology can influence spatio-temporal and angular gait parameters in this population. Given that, gait is a unique daily activity, associated with control of bilateral lower limbs movement and coordination, understanding whether the strategies used by subgroups of individuals with stroke to compensate their unilateral impairment can be helpful in planning a more specific rehabilitation programs. The results showed survivors of a hemorrhagic stroke suffer greater gait impairments in the variables analyzed, even after adjustments for age and sex. In addition to being statistically significant, these between-group differences are clinically meaningful, as reported in earlier studies,[39–41] demonstrating statistical power of more than 0.70 at a significance level of 0.05. These differences may be associated with the type of lesion, since hemorrhagic variety is generally more severe and affect a larger brain area.[34] These results suggest that lesion etiology is an important factor that could help in planning clinical interventions to achieve at long-term results and more effective rehabilitation programs. Moreover, it also highlights the importance of considering lesion etiology during research through subgroup analysis. This approach is currently uncommon, but could avoid the wide range of treatment effects. It is not suggested that individuals with hemorrhagic stroke be excluded from these studies, however, participants' stratification according to lesion etiology should be considered in data analysis and interpretation.

In the first months after stroke, individuals with right cerebral hemisphere impairments display worse Tomei mobility scores when compared to those affected in the left hemisphere.[26] However, our results show differences in gait pattern between these subgroups six months after stroke onset, were observed only for swing time symmetry ratio. These results corroborate

those reported by Correa et al.,[42] who found no difference in stride duration and length, or gait speed and cadence between individuals with cerebral lesions in the right or left hemisphere possibly due to the similarities in electromyographic parameters and ground reaction between the two groups. Furthermore, similarities in most of the gait parameters analyzed may be associated with neuronal network reorganization resulting from motor experience and specific rehabilitation after stroke.

Although there were intergroups similarities in terms of the affected hemisphere, individuals with left hemisphere lesion were more asymmetric. This finding disagrees with the results of Chen et al.[43] who showed that individuals with chronic stroke and right cerebral hemisphere lesion are slower and more asymmetric than those with a lesion in the left hemisphere. These differences could be associated with the sample characteristics, since Chen et al.[43] only evaluated subjects with unilateral infarcts affecting less than one-third of the MCA territory, which could limit extrapolating the results. On the other hand, the results of the present study can be associated with the poorer mobility levels observed during the first months after stroke in individuals with left hemisphere damage, as previously described.[25,26] This could restrict gait and improve paretic limb weakness resulting in a more compensatory gait pattern.[44] However, the present investigation did not analyze mobility level in the first few months after stroke, precluding confirmation of this hypothesis. Additionally, affected hemisphere seems to primarily influence interlimb coordination parameters such as gait symmetry which may increase the energy cost of walking, and restrict mobility levels and daily functional performance.[45–47]

Studies have shown that individuals with stroke can improve gait parameters beyond six months after stroke onset, when a spontaneous motor recovery plateau is reached.[48] These improvements may result from compensatory mechanisms adopted over the years,[49–52] as well as motor gains generated by intense, repetitive, and specific rehabilitation programs prescribed after the stroke. Thus, the longer stride length observed in the present study in individuals with time since stroke of more than 36 months (G_{III}) could result from compensatory mechanisms adopted during their lifetime and/or the more rehabilitation experience in this group. Furthermore, these individuals may have always had a longer step length and did not actually improve this variable over time. However, since this is a cross-sectional study, this hypothesis cannot be confirmed, which represents a study limitation.

Other limitations include: the small sample size, the difference in the number of individuals in each subgroup for multiple comparisons, the relatively young sample which can limits extrapolation to a larger population, the absence of rehabilitation protocol control in the years after the stroke and the absence of volume and extent of brain damage analysis. We suggest that future studies with a larger sample be conducted, to investigate other clinical factors that could better explain the differences in gait pattern observed in individuals with stroke.

Conclusion

Parameters such as lesion side, time since stroke onset and primarily lesion etiology can influence gait pattern in individuals with chronic stroke and be used to differentiate gait parameters, underscoring their importance in research and clinical environments. Studies considering these factors could result in the recruitment of a more homogeneous sample and more accurate results. In a clinical environment, these factors could help prescribing the best rehabilitation program to achieve short- and long-term gait improvements.

Adherence to ethics and reporting requirements

Ethical principles with respect to clinical research involving human beings adhered to those proposed by the Declaration of Helsinki, and the study was approved by the Institutional Ethics Committee of Onofre Lopes University Hospital under protocol number 0364.0.000.294-11.

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