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Background and Purpose—Gait disorders are common in the elderly and are related to loss of functional independence and death. White matter lesions (WMLs) may be related, but only a minority of individuals with WMLs has gait disorders. Probably other factors are involved, including location and the independent effect of frequently coinciding lacunar infarcts, the other aspect of cerebral small vessel disease. The aim of our study was to investigate the effect of both the severity and location of both WMLs and lacunar infarcts on gait.

Methods—Four hundred thirty-one independently living, nondemented elderly aged between 50 and 85 years with cerebral small vessel disease were included in this analysis and underwent MRI scanning. The number and location of lacunar infarcts were rated and WML volume was assessed by manual segmentation with automated delineating of different regions. Gait was assessed quantitatively with an electronic walkway as well as the semiquantitatively Tinetti and Timed-Up-and-Go test.

Results—WMLs and lacunar infarcts were both independently associated with most gait parameters with stride length as the most sensitive parameter related to WMLs. WMLs in the sublobar (basal ganglia/internal capsule) and limbic areas and lacunar infarcts in the frontal lobe and thalamus were related to a lower velocity.

Conclusions—Cerebral small vessel disease is related to gait disturbances. Because small vessel disease may, in part, be preventable, it should be regarded as a potentially important target for postponing gait impairment. **(*Stroke*. 2010;41: 1652-1658.)**

Key Words: cerebral small vessel disease ■ gait ■ lacunar infarcts ■ MRI ■ white matter lesions

Gait disturbances are common in the elderly and are associated with loss of functional independence, institutionalization, and death.[1] Proper gait is the result of automated spinal motor programs under supraspinal control. The integrity of connections between the cortical areas and between these areas and the spinal cord can be disrupted by small vessel disease (SVD), including white matter lesions (WMLs) and lacunar infarcts. WMLs are recognized as a possible cause of gait impairment,[2–7] but not all individuals with WMLs have gait disturbances.[2] It may be that the severity of WMLs or the frequent coinciding lacunar infarcts play a role. Remarkably, there was no statistical control for the presence of lacunar infarcts in most of these studies and they did not investigate the independent effect of these lesions on gait.[3–6] Moreover, the site of SVD in relation to gait could be another important factor, but was usually not taken into account. Only few studies with small sample size investigated the effect of WML location on gait.[8–10]

Quantitative gait analysis, providing insight into the different components of gait, has been performed in patients with extensive SVD with severe gait disorders.[2] However, most studies among independently living elderly with SVD used semiquantitative clinical rating scales,[3,6] a composite gait score,[7] or measured only gait velocity.[3,6] Hence, it is not clear how SVD alters the different parameters of the gait pattern in the early stages of the disease and which parameter is a sensitive indicator for underlying SVD.

The aim of our study was therefore to investigate the individual contribution of WML volume and lacunar infarcts, overall and by specific location, on different gait parameters using quantitative gait analysis.

Subjects and Methods

Study Population

The Radboud University Nijmegen Diffusion tensor and MRI Cohort (RUN DMC) study is a prospective cohort study that was designed to investigate risk factors and cognitive, motor, and mood consequences of functional and structural brain changes in the elderly with cerebral SVD.

In subjects with cerebral SVD, symptoms are due to either complete (lacunar infarcts) or incomplete infarction (WMLs) of

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subcortical structures that might lead to acute symptoms as transient ischemic attacks or lacunar syndromes or subacute manifestations as cognitive, motor, and/or mood disturbances.[11] Because the onset of cerebral SVD is often insidious, clinically heterogeneous and with mild symptoms, it has been suggested that the selection of subjects with cerebral SVD in clinical studies should be based on the more consistent brain imaging features.[12] Accordingly, in 2006, consecutive patients from the department of neurology between October 2002 and November 2006 were selected for participation.

Inclusion criteria were: (1) age between 50 and 85 years; (2) cerebral SVD on neuroimaging; and (3) acute (n=219) or subacute (n=284) clinical symptoms of SVD as assessed by standardized structured assessments. Patients who were eligible because of a lacunar syndrome were included only >6 months after the event to avoid acute effects on the outcomes.

Exclusion criteria were: (1) dementia; and (2) parkinson(ism) according to the international diagnostic criteria[13–15]; (3) life expectancy of <6 months; (4) intracranial space-occupying lesion; (5) (psychiatric) disease interfering with cognitive testing or follow-up; (6) recent or current use of acetylcholine–esterase inhibitors, neuroleptic agents, L-dopa, or dopa-a(nta)gonists; (7) WML mimics (eg, multiple sclerosis and irradiation induced gliosis); (8) prominent visual or hearing impairment; (9) language barrier; and (10) MRI contraindications or known claustrophobia.

From 1004 invited individuals by letter, 727 were eligible after contact by phone and 525 agreed to participate. In 22 subjects, exclusion criteria were found during their visit to our research center, yielding a response of 71.3% (503 of 705). These 503 individuals had symptoms of transient ischemic attack or lacunar syndrome (n=219), cognitive disturbances (n=245), motor disturbances (n=97), depressive symptoms (n=100), or a combination thereof. A motor disturbance was defined, in accordance with operationalization as used in other large-scale studies on cerebral SVD and gait,[16] as follows: a reported history of ≥1 falls during the past year or a self-reported slowing of gait. All participants signed an informed consent form. The Medical Review Ethics Committee of region Arnhem-Nijmegen approved the study.

Measurement of Gait

Quantitative gait analysis was performed with a 5.6-m electronic portable walkway (GAITRite; MAP/CIR Inc, Havertown, Pa). We measured velocity (m/s), consisting of the stride length (m; the distance between the heel points of 2 consecutive footprints of the same foot) and cadence (number of steps per minute), stride width (cm; the distance between the midpoint of a footprint to the line of progression of the opposite foot), double support percentage (percentage of the gait cycle time during which both feet are on the floor), and variability of stride length, stride time, and stride width, 2 times at a self-selected speed. Variability was expressed as coefficients of variation: SD/mean×100%.

Semiquantitative assessment consisted of the modified Tinetti test with 17 items: 9 for body balance (score 0 to 16) and 8 for gait (score 0 to 12) with a maximum score of 28 as well as the Timed-Up-and-Go (TUG) test, which was executed three times. Interrater reliability was calculated in a random sample of 15% with an intraclass correlation coefficient of 0.99.

MRI Scanning and Processing

Imaging was performed on a 1.5-Tesla scanner (Magnetom Sonata; Siemens Medical Solutions, Erlangen, Germany). The protocol included 3-dimensional T1 magnetization-prepared rapid gradient-echo imaging (time repetition/time echo/time interval 2250/3.68/850 ms; flip angle 15°; voxel size 1.0×1.0×1.0 mm) and fluid-attenuated inversion recovery sequences (time repetition/time echo/time interval 9000/84/2200 ms; voxel size 1.0×1.2×5.0 mm, plus an interslice gap of 1 mm). All scans were performed on the same scanner.

White matter signal hyperintensities on fluid-attenuated inversion recovery scans, which were not, or only faintly, hypointense on T1-weighted images, were considered WMLs, except for gliosis surrounding infarcts. WMLs were manually segmented on the fluid-attenuated inversion recovery images by 2 trained raters. All imaging analyses were performed by raters blinded to clinical information. Also, the WML volume of predefined volumes of interest, taken from an inversely normalized (parameters taken from the T1 normalization) Talairach-based atlas[17] (WFU Pickatlas, Version 2.3), was computed. They included the frontal, parietal, occipital, temporal lobes, and sublobar (basal ganglia, thalamus, internal and external capsule, insula) and limbic (cingulate gyrus) area, brain stem, and cerebellum. Lacunar infarcts were defined as areas with a diameter >2 mm and <15 mm with low signal intensity on T1 and fluid-attenuated inversion recovery ruling out enlarged perivascular spaces and infraputaminal pseudolacunes.[18] In a random sample of

Table 1. Characteristics of the Study Population

Characteristics	N=431
Demographic and clinical characteristics	
Age at admission, years	63.8 (8.9)
Age at enrollment, years	65.2 (8.9)
Disease duration, years*	1.4 (1.1)
Female, no.	195 (45.2)
Height, m	1.7 (0.1)
Mini Mental State Examination	28.2 (1.6)
Barthel Index	19.7 (0.7)
Neuroimaging characteristics	
TBV, mL	1098.9 (119.5)
White matter volume, mL	467.5 (64.9)
WML volume, mL	6.5 (3.2–17.7)
Frontal	2.1 (0.9–6.1)
Parietal	0.2 (0.0–1.1)
Occipital	0.6 (0.3–1.1)
Temporal	0.4 (0.1–1.6)
Sublobar	2.5 (1.2–4.5)
Limbic	0.4 (0.2–1.1)
Infratentorial	0.2 (0.1–0.6)
Lacunar infarcts, no.	132 (30.6)
Frontal	47 (10.9)
Parietal	23 (5.3)
Occipital	14 (3.2)
Temporal	11 (2.6)
Sublobar (basal ganglia/internal capsule)	74 (17.2)
Limbic	0 (0)
Infratentorial	25 (5.8)
Gait characteristics	
Gait velocity, m/s	1.3 (0.3)
Stride length, m	1.4 (0.2)
Cadence, steps/min	112.1 (10.8)
Stride width, cm	10.8 (3.1)
Double support percentage	25.8 (4.2)
Stride length variability, %	1.7 (1.2–2.5)
Stride time variability, %	1.6 (1.2–2.3)
Stride width variability, %	17.1 (12.4–24.0)
Tinetti test	28.0 (28.0–28.0)
TUG test, seconds	8.7 (7.7–10.3)
TUG test, no. of steps	12.4 (3.4)

Data represent no. (%), mean (SD) or median (interquartile range).
*Defined as age at enrollment−age at admission.

Table 2. Association Between Cerebral SVD and Gait

	Gait Parameters										
	GAITRite Parameters								Clinical Rating Scales		
Cerebral SVD	Gait Velocity, m/s	Stride Length, m	Cadence, Steps/min	Stride Width, cm	DSP, %	CV Stride Length,* %	CV Stride Time,* %	CV Stride Width,* %	Tinetti Test	TUG Test,* Seconds	TUG Test, No. of Steps
WML volume, mL											
Model 1	−0.21‡	−0.22‡	−0.08	0.25‡	0.19‡	0.15†	0.13†	−0.12†	−0.23‡	0.16†	0.18‡
Model 2	−0.14†	−0.16‡	−0.03	0.20‡	0.10	0.10	0.06	−0.09	−0.10	0.08	0.12†
Lacunar infarcts, no.											
Model 1	−0.20‡	−0.20‡	−0.13†	0.19‡	0.23‡	0.14†	0.16†	−0.11†	−0.32‡	0.20‡	0.17‡
Model 2	−0.14†	−0.13†	−0.12†	0.10†	0.19‡	0.10	0.14†	−0.07	−0.28‡	0.17†	0.12†

Data are standardized β values.

Model 1 represents the relation between WML volume in milliliters or no. of lacunar infarcts and gait adjusted for age, sex, height, and TBV; Model 2 is with additional adjustment for no. of lacunar infarcts or WML volume.

*For skewed variables, the logarithm is presented.

†$P<0.05$.

‡$P<0.001$.

DSP indicates double support percentage; CV, coefficients of variation.

10%, interrater variability for WMLs yielded an intraclass correlation coefficient of 0.99; intra- and interrater reliability for lacunar infarctions a weighted κ of 0.80 and 0.88.

Automated segmentation on the T1 images was conducted using Statistical Parametric Mapping (SPM5; www.fil.ion.ucl.ac.uk/spm/) to obtain gray and white matter and cerebrospinal fluid maps.[19] Total brain volume (TBV) was calculated as the sum of total gray and white matter volumes.

Other Measurements

We considered age, sex, height, and TBV as possible confounders. We used the Mini Mental State Examination score (range 0 to 30) to assess global cognitive status. Functional independence was assessed using the Barthel Index (range 0 to 20).

Statistical Analysis

The mean quantitative GAITRite and semiquantitative TUG measures were calculated as the mean of the 2 and 3 walks. When 1 trial was missing, the remaining measures were used (n=3 for quantitative and n=2 for semiquantitative gait assessment).

The relation between SVD and gait was analyzed using 3 different approaches. First, we used age, sex, height, and TBV adjusted multiple linear regression to investigate the relation between WML volume or number of lacunar infarcts and gait. Subsequently, we adjusted for either the number of lacunar infarcts (with WMLs as independent variable) or WML volume (with lacunar infarcts as independent variable). This was also done by location of the WMLs and presence of lacunar infarcts. In positively skewed distributions, log transformation was used. Regression coefficients were presented as standardized β values. Second, a possible independent dose–effect relation of WMLs and number of lacunar infarcts with gait velocity was investigated by means of analysis of covariance with WMLs in quintiles or number of lacunar infarcts in 3 groups and the previously mentioned covariates. Third, the risk of impaired gait (velocity <1 m/s[20] or TUG test >12 seconds[21]) by the severity of WMLs (in quintiles) or the number of lacunar infarcts was assessed with logistic regression analysis. Because no impaired TUG test was observed in the first quintile, the first and second quintiles together were considered as the reference group.

All analyses were performed using SPSS Version 16.0.

Results

Characteristics

All gait variables were available for 488 participants. Fifteen individuals could not participate because of walking aids (n=4), current levodopa use (n=1), drop foot (n=1), lower extremity amputation (n=1), joint fusion (n=2), severe arthritis (n=1), severe vascular problems of the lower extremity (n=2), or a functional gait disturbance (n=3). Territorial infarcts were considered confounders and therefore individuals with these infarcts were excluded (n=56). One subject had to be excluded because of technical problems with the calculation of the TBV, yielding a final sample size of 431 subjects.

Table 1 represents the characteristics of these 431 subjects. The mean age was 65.5 years with 43% being women. Eighty-four subjects were classified as having a motor disturbance. Of these 84, none had a mild or severe hemiplegia (Medical Research Council Scale Grade ≤4). Furthermore, of all subjects with a history of a transient ischemic attack or lacunar syndrome, 4 participants were still having a mild hemiplegia of 1 of their legs at the time of enrollment. All analyses were repeated excluding these 4 subjects. This did not markedly change the magnitude of the association. We therefore present the results for the group as a whole. Of all the participants, 11.6% had a gait velocity <1 m/s and 10.4% a TUG test >12 seconds.

Severity of SVD and Gait

WMLs and lacunar infarcts were both independently associated with most of the spatiotemporal gait parameters (Table 2) and borderline significant with variability in stride length (β=0.10; P=0.055 and β=0.10; P=0.066). Regarding the clinical rating scales, there was an association between WMLs and the number of steps needed during the TUG test but not with the duration of the test. Lacunar infarcts were related with both tests (Table 2). In analyses restricted to subjects without lacunar infarcts, WMLs were only associated with a shorter stride length (β=−0.13; P=0.006) and not with other parameters or clinical rating scales.

The Figure shows a dose–effect relation among the severity of WMLs, lacunar infarcts, and gait velocity (P trend=0.003). Those with severe (fifth quintile) WMLs were 4 times more

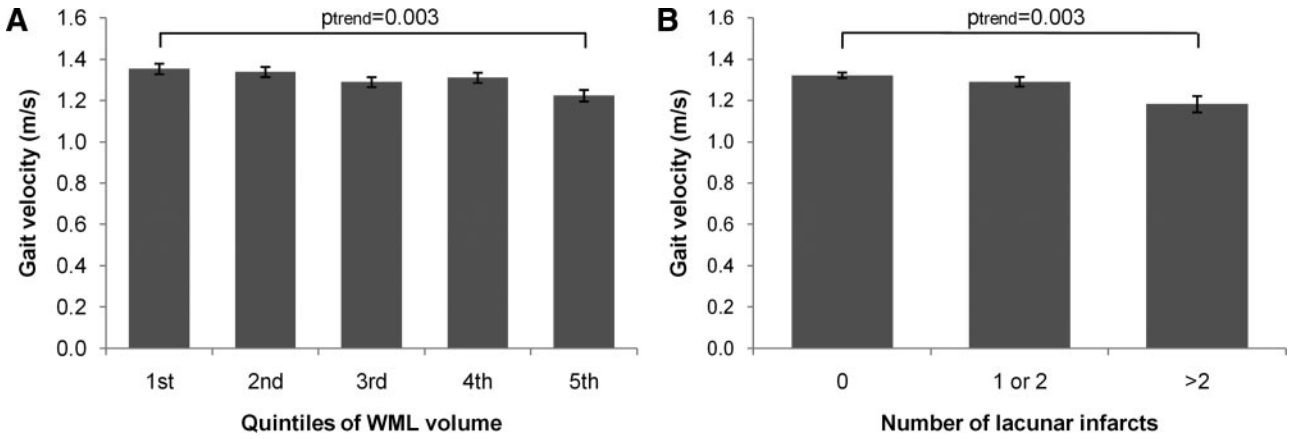

Figure. Dose–effect relation of cerebral SVD and gait velocity. Adjusted for age, sex, height, TBV, and number of lacunar infarcts (A) or WMLs (B; with SEs).

likely to have an abnormal gait velocity or an abnormal TUG test than the reference group. Subjects with >2 lacunar infarcts had an OR of 4.5 (95% CI, 1.7 to 12.0) for impaired gait velocity and 3.1 (95% CI, 1.1 to 8.7) for an impaired TUG test (Table 3).

Location of SVD and Gait

Because gait velocity is dependent on stride length and cadence, we examined these 3 parameters separately. WMLs in all regions were associated with gait velocity with the strongest association in the sublobar and limbic regions and to a lesser extent in the frontal lobe, due to a reduction in stride length. Lacunar infarcts in the frontal lobe were related to both a shorter stride length ($\beta=-0.08$; $P=0.042$) and lower step frequency ($\beta=-0.11$; $P=0.025$), resulting in a lower gait velocity. Those subjects with thalamic infarcts walked slower ($\beta=-0.13$; $P=0.005$) due to a shorter stride length. Cadence was also related with the presence of lacunar infarcts in the brain stem ($\beta=-0.13$; $P=0.005$; Table 4).

Discussion

Both WMLs and lacunar infarcts were independently associated with several gait parameters. WMLs particularly in the sublobar (basal ganglia, thalamus, internal and external capsule, insula), limbic area, and frontal lobe seemed to be related to a lower gait velocity, due to a shorter stride length, and lacunar infarcts in the thalamus and frontal lobe to a lower gait velocity, due to, respectively, a shorter stride length and both a reduced stride length and cadence.

Some methodological issues need to be considered. First, we report on cross-sectional data, which prevent causal inference. Furthermore, in the examination of the influence of the location of SVD, a potential confounder could be the presence of SVD in other regions related to gait, which are highly correlated with each other. We tried to overcome this problem by analyzing the relations in 2 separate models, 1 with and 1 without taking into account the possible confounding effect of SVD in the other regions. As a major advantage of our study, WML volume was obtained by manual segmen-

Table 3. Association Between Severity of Cerebral SVD and Impaired Gait

Severity of Cerebral SVD		Impaired Gait Velocity*		Impaired TUG Test†	
		OR (95% CI)	No./Total‡	OR (95% CI)	No./Total‡
WML volume in quintiles (range in mL)					
First+second	(0.5–5.1)	1.0 (reference)	5/172	1.0 (reference)	4/172
Third	(5.1–8.9)	4.1 (1.3–12.5)§	12/87	2.7 (0.8–9.8)	8/87
Fourth	(8.9–20.6)	2.0 (0.6–6.6)	10/86	2.5 (0.7–8.9)	11/86
Fifth	(20.6–139.7)	4.3 (1.3–14.1)§	23/86	4.4 (1.2–15.8)§	22/86
Lacunar infarcts					
0		1.0 (reference)	23/299	1.0 (reference)	22/299
1 or 2		1.4 (0.6–3.1)	13/93	1.0 (0.4–2.4)	11/93
>2		4.5 (1.7–12.0)§	14/39	3.1 (1.1–8.7)§	12/39

Adjusted for age, sex, height, TBV, and no. of lacunar infarcts or WMLs.
*Defined as <1 m/s in gait velocity.
†Defined as a TUG test of >12 seconds.
‡No. represents the absolute no. of subjects with an impaired gait velocity or TUG test in that group.
§$P<0.05$.

Table 4. Association Between Location of Cerebral SVD and Gait

Cerebral SVD	Gait Parameters					
	Gait Velocity, m/s		Stride Length, m		Cadence, Steps/min	
	Model 1	Model 2	Model 1	Model 2	Model 1	Model 2
Frontal lobe						
WML volume, mL	−0.17†	−0.06	−0.19†	−0.09	−0.04	0.05
Lacunar infarcts	−0.11*	−0.11*	−0.09*	−0.08*	−0.11*	−0.11*
Parietal lobe						
WML volume, mL	−0.17†	0.06	−0.19†	0.02	−0.04	0.09
Lacunar infarcts	0.01	0.04	0.04	0.07	−0.03	−0.01
Occipital lobe						
WML volume, mL	−0.12*	0.02	−0.12*	0.04	−0.08	−0.05
Lacunar infarcts	−0.05	−0.03	−0.05	−0.03	−0.03	−0.02
Temporal lobe						
WML volume, mL	−0.16*	0.14	−0.18†	0.06	−0.04	0.18
Lacunar infarcts	0.02	0.04	−0.02	−0.01	0.07	0.08
Sublobar areas						
WML volume, mL	−0.19†	−0.14	−0.18†	−0.09	−0.10*	−0.13
Lacunar infarcts	−0.09*	−0.06	−0.10*	−0.08	−0.04	−0.01
Limbic areas						
WML volume, mL	−0.20†	−0.13	−0.22†	−0.14	−0.08	−0.06
Lacunar infarcts	–	–	–	–	–	–
Infratentorial						
WML volume, mL	−0.18†	−0.03	−0.19†	−0.03	−0.07	−0.05
Lacunar infarcts	−0.05	−0.02	−0.01	0.01	−0.11*	−0.10*
Brain stem						
WML volume, mL	−0.15*	−0.01	−0.16†	−0.00	−0.06	−0.03
Lacunar infarcts	−0.08	−0.06	−0.04	−0.02	−0.14*	−0.13*
Cerebellum						
WML volume, mL	−0.21†	−0.11	−0.22†	−.14	−0.08	−0.06
Lacunar infarcts	−0.03	−0.01	−0.02	−.00	−0.05	−0.04

Data are standardized β values.

Model 1 represents the independent association between WML volume in milliliters, presence of lacunar infarcts, and gait adjusted for age, sex, height, and TBV; Model 2 is with additional adjustment for WMLs and presence of lacunar infarcts in the other regions.

*$P<0.05$.

†$P<0.001$.

tation with automated delineating of different regions. Gait was also assessed in a quantitative manner as well as with semiquantitative rating scales, because these tests are frequently used and easy to apply in everyday clinical practice. Another strong element of our study was the fact that we were able to investigate the effect of SVD on gait independent of possible confounders such as TBV and lacunar infarcts (and WMLs with lacunar infarcts as an independent variable). We intentionally did not correct for vascular risk factors as hypertension or diabetes because they were considered part of the causal chain between SVD and gait. Finally, our study was large with a high response and with all subjects examined by only 2 investigators in a single center.

Gait velocity is determined by both the stride length and cadence and was found to be associated with both aspects of SVD in our study. No studies discriminated between these 2 components of velocity. We showed that cadence was only reduced in association with lacunar infarcts. In analyses restricted to subjects without lacunar infarcts, WML volume was significantly related with stride length but not with velocity or other parameters. We therefore suggest that stride length is a more sensitive indicator for (subclinical) SVD than velocity. This is also reflected by more steps needed during the TUG test in subjects with a higher WML volume, but without a significant effect on the duration of the test. The same is observed in other gait disorders such as Parkinson disease and normal pressure hydrocephalus.[22] In conclusion, these results suggest that measuring stride length instead of velocity could lead to early detection of gait abnormalities in SVD.

Apart from the lower velocity, due to smaller steps and in the presence of lacunar infarcts also due to a lower step

frequency, we found that gait in subjects with more severe SVD was characterized by a broader base, more shuffling, reflected by increased double support percentage, and with a higher variability in stride time and trend to a higher stride length variability. Rosano et al did not detect an effect of SVD on stride width in contrast to our finding of a clear relation with both aspects of SVD.[4] The association of lacunar infarcts with double support percentage is also a novel finding. Gait variability, which is suggested to be more sensitive to fall risk than other gait parameters,[23] has not been extensively studied in the early stages of SVD. Although the effect of WML on overall gait variability was recently reported,[7] our data provide insight into the different components because we showed an association between variability in stride time and to a lesser extent in stride length and SVD.

Because the presence of ≥3 lacunar infarcts was significantly associated with gait velocity, our findings suggested a threshold rather than a dose–effect. However, those with >4 lacunar infarcts were limited and could therefore not be investigated separately. In contrast, the relation between WML and gait suggested a dose-dependent effect. A similar finding was reported by 2 other studies.[3,7] One study showed a threshold effect, but the median WML volume in this study was lower than in our study.[6] They found a significant lower gait velocity with WML volume >5 to 10 mL like we did. Hence, it seems that gait impairment only occurs when WMLs are moderate or (in the combination) with more lacunar infarcts.

The exact cerebral networks impaired in gait disorders due to SVD have not been identified yet. In previous studies in subjects with gait impairment and extensive SVD, the frontal basal ganglia–thalamocortical circuit was considered to be involved.[2] Our findings support this hypothesis, even during the early stages of SVD because we showed that lacunar infarcts in the frontal lobe were related to a lower gait velocity. Furthermore, we found WMLs in the sublobar region and thalamic infarcts to be related to gait velocity. Rosano et al also suggested that lacunar infarcts in the basal ganglia were related to gait velocity because most of the infarcts in their population were located in the basal ganglia, but their sample size was too small for subanalyses.[4] A remarkable finding in our study was the relevance of WMLs in the limbic area for gait velocity. This is in accordance with a functional MRI study that showed, except for activation of the dorsal premotor cortex and supplementary motor area, also activation of the cingulated motor area and the parahippocampal gyri.[24] These latter 2 areas are part of the limbic area and believed to be required for spatial navigation. At last, we found that lacunar infarcts in the brain stem were associated with a lower cadence. This suggests that cadence is regulated, among others, by regions in the brain stem (and cerebellum) such as the mesencephalic locomotor regions. One other study also demonstrated that persons with WMLs in the brain stem walked more slowly.[10] Our findings support the notion that gait relies on widespread cerebral networks that are vulnerable to the disruption by SVD resulting in impaired gait. Further studies, for example with the aid of diffusion tensor imaging, may be useful to investigate the disruption of these networks by cerebral SVD more precisely.

In conclusion, cerebral SVD contributes to disturbances in gait even at a preclinical level that currently cannot be identified properly with standard semiquantitative clinical tests. Stride length may be a useful measure for the early detection of gait abnormalities due to SVD. Follow-up studies are needed to study possible gait deterioration in those patients. A clinical rule of thumb could be that gait disturbances should only be attributed to SVD in patients with moderate or severe WMLs and/or with the presence of ≥3 lacunar infarcts, especially at specific locations. This provides an explanation why only some patients with SVD have gait disturbances. Because cerebral SVD may, in part, be preventable, it should be regarded as a potentially therapeutic target for postponing gait impairment.

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Disclosures

None.

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