Gait in Elderly With Cerebral Small Vessel Disease

Karlijn F. de Laat, MD; Anouk G.W. van Norden, MD; Rob A.R. Gons, MD; Lucas J.B. van Oudheusden, MSc; Inge W.M. van Uden, MD; Bastiaan R. Bloem, MD, PhD; Marcel P. Zwiers, PhD; Frank-Erik de Leeuw, MD, PhD

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.1–6 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.5–10

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...
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. 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. 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) parkinsonism according to the international diagnostic criteria; (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, neuroleptics, L-dopa, or dopa-antineuroleptics; (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, 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 (WFU Pickatlas, Version 2.3), was computed. They included the frontal, parietal, occipital, temporal lobes, and subcortical (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 infratentorial pseudolacunae. In a random sample of

### Table 1. Characteristics of the Study Population

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>N=431</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Demographic and clinical characteristics</strong></td>
<td></td>
</tr>
<tr>
<td>Age at admission, years</td>
<td>63.8 (8.9)</td>
</tr>
<tr>
<td>Age at enrollment, years</td>
<td>65.2 (8.9)</td>
</tr>
<tr>
<td>Disease duration, years*</td>
<td>1.4 (1.1)</td>
</tr>
<tr>
<td>Female, no.</td>
<td>195 (45.2)</td>
</tr>
<tr>
<td>Height, m</td>
<td>1.7 (0.1)</td>
</tr>
<tr>
<td>Mini Mental State Examination</td>
<td>28.2 (1.6)</td>
</tr>
<tr>
<td>Barthel Index</td>
<td>19.7 (0.7)</td>
</tr>
<tr>
<td><strong>Neuroimaging characteristics</strong></td>
<td></td>
</tr>
<tr>
<td>TBV, mL</td>
<td>1098.9 (119.5)</td>
</tr>
<tr>
<td>White matter volume, mL</td>
<td>467.5 (64.9)</td>
</tr>
<tr>
<td>WML volume, mL</td>
<td>6.5 (3.2–17.7)</td>
</tr>
<tr>
<td>Frontal</td>
<td>2.1 (0.9–6.1)</td>
</tr>
<tr>
<td>Parietal</td>
<td>0.2 (0.0–1.1)</td>
</tr>
<tr>
<td>Occipital</td>
<td>0.6 (0.3–1.1)</td>
</tr>
<tr>
<td>Temporal</td>
<td>0.4 (0.1–1.6)</td>
</tr>
<tr>
<td>Sublobar</td>
<td>2.5 (1.2–4.5)</td>
</tr>
<tr>
<td>Limbic</td>
<td>0.4 (0.2–1.1)</td>
</tr>
<tr>
<td>Infratentorial</td>
<td>0.2 (0.1–0.6)</td>
</tr>
<tr>
<td>Lacunar infarcts, no.</td>
<td>132 (30.6)</td>
</tr>
<tr>
<td>Frontal</td>
<td>47 (10.9)</td>
</tr>
<tr>
<td>Parietal</td>
<td>23 (5.3)</td>
</tr>
<tr>
<td>Occipital</td>
<td>14 (3.2)</td>
</tr>
<tr>
<td>Temporal</td>
<td>11 (2.6)</td>
</tr>
<tr>
<td>Sublobar (basal ganglia/internal capsule)</td>
<td>74 (17.2)</td>
</tr>
<tr>
<td>Limbic</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Infratentorial</td>
<td>25 (5.8)</td>
</tr>
</tbody>
</table>

**Gait characteristics**

<table>
<thead>
<tr>
<th>Gait characteristics</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Gait velocity, m/s</td>
<td>1.3 (0.3)</td>
</tr>
<tr>
<td>Stride length, m</td>
<td>1.4 (0.2)</td>
</tr>
<tr>
<td>Cadence, steps/min</td>
<td>112.1 (10.8)</td>
</tr>
<tr>
<td>Stride width, cm</td>
<td>10.8 (3.1)</td>
</tr>
<tr>
<td>Double support percentage</td>
<td>25.8 (4.2)</td>
</tr>
<tr>
<td>Stride length variability, %</td>
<td>1.7 (1.2–2.5)</td>
</tr>
<tr>
<td>Stride time variability, %</td>
<td>1.6 (1.2–2.3)</td>
</tr>
<tr>
<td>Stride width variability, %</td>
<td>17.1 (12.4–24.0)</td>
</tr>
<tr>
<td>Tinetti test</td>
<td>28.0 (28.0–28.0)</td>
</tr>
<tr>
<td>TUG test, seconds</td>
<td>8.7 (7.7–10.3)</td>
</tr>
<tr>
<td>TUG test, no. of steps</td>
<td>12.4 (3.4)</td>
</tr>
</tbody>
</table>

Data represent no. (%), mean (SD) or median (interquartile range).
*Defined as age at enrollment—age at admission.
10%, interrater variability for WMLs yielded an intraclass correlation coefficient of 0.99; intra- and interrater reliability for lacunar infarctions a weighted $k$ 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. 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 $\beta$ 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 the number of lacunar infarcts was assessed (velocity was investigated by means of analysis of covariance with WMLs in quintiles or the number of lacunar infarcts was assessed (velocity was investigated by means of analysis of covariance with WMLs in quintiles or the 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 infaracts 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.

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 ($\beta=0.10$; $P=0.055$ and $\beta=0.10$; $P=0.066$). 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 ($\beta=-0.13$; $P=0.006$) and not with other parameters or clinical rating scales.

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

### Table 2. Association Between Cerebral SVD and Gait

<table>
<thead>
<tr>
<th>Cerebral SVD</th>
<th>GAITRite Parameters</th>
<th>Clinical Rating Scales</th>
</tr>
</thead>
<tbody>
<tr>
<td>WML volume, mL</td>
<td>Gait Velocity, m/s</td>
<td>Stride Length, m</td>
</tr>
<tr>
<td>Model 1</td>
<td>-0.21‡</td>
<td>-0.22‡</td>
</tr>
<tr>
<td>Model 2</td>
<td>-0.14‡</td>
<td>-0.16‡</td>
</tr>
<tr>
<td>Lacunar infarcts, no.</td>
<td>Model 1</td>
<td>-0.20‡</td>
</tr>
<tr>
<td>Model 2</td>
<td>-0.14‡</td>
<td>-0.13†</td>
</tr>
</tbody>
</table>

Data are standardized $\beta$ 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.
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-

![Figure](https://stroke.ahajournals.org/)

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

<table>
<thead>
<tr>
<th>Severity of Cerebral SVD</th>
<th>Impaired Gait Velocity*</th>
<th>Impaired TUG Test†</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>OR (95% CI)</td>
<td>No./Total‡</td>
</tr>
<tr>
<td>WML volume in quintiles (range in mL)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>First+second (0.5–5.1)</td>
<td>1.0 (reference)</td>
<td>5/172</td>
</tr>
<tr>
<td>Third (5.1–8.9)</td>
<td>4.1 (1.3–12.5)$^\S$</td>
<td>12/87</td>
</tr>
<tr>
<td>Fourth (8.9–20.6)</td>
<td>2.0 (0.6–6.6)</td>
<td>10/86</td>
</tr>
<tr>
<td>Fifth (20.6–139.7)</td>
<td>4.3 (1.3–14.1)$^\S$</td>
<td>23/86</td>
</tr>
<tr>
<td>Lacunar infarcts</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>1.0 (reference)</td>
<td>23/299</td>
</tr>
<tr>
<td>1 or 2</td>
<td>1.4 (0.6–3.1)</td>
<td>13/93</td>
</tr>
<tr>
<td>&gt;2</td>
<td>4.5 (1.7–12.0)$^\S$</td>
<td>14/39</td>
</tr>
</tbody>
</table>

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.
$^\S P < 0.05$. 

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).
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. 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...
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 distur-
bances 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 pro-
vides 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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