Cerebral Infarcts and Cognitive Performance
Importance of Location and Number of Infarcts

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Background and Purpose—Cerebral infarcts increase the risk for cognitive impairment. The relevance of location and number of infarcts with respect to cognitive function is less clear.

Methods—We studied the cross-sectional association between number and location of infarcts and cognitive performance in 4030 nondemented participants of the Age Gene/Environment Susceptibility-Reykjavik Study. Composite scores for memory, processing speed, and executive function were created from a neuropsychological battery. Subcortical, cortical, and cerebellar infarcts were identified on brain MRI. We performed linear regression analyses adjusted for demographic and vascular risk factors, depression, white matter lesions, and atrophy.

Results—Compared to participants with no infarcts, those with infarcts in multiple locations (n=287, 7%) had slower processing speed (β=−0.19; P<0.001) and poorer memory (β=−0.16; P<0.001) and executive function (β=−0.12; P=0.003). Compared to no infarcts, the presence of either subcortical infarcts only (n=275; β=−0.12; P=0.016) or cortical infarcts only (n=215; β=−0.17; P=0.001) was associated with poorer memory performance. Compared to no infarcts, a combination of cortical and subcortical infarcts (n=45) was associated with slower processing speed (β=−0.38; P<0.001) and poorer executive function (β=−0.22; P=0.02), whereas a combination of cerebellar and subcortical infarcts (n=89) was associated with slower processing speed (β=−0.15; P=0.04). Infarcts in all 3 locations was associated with slower processing speed (β=−0.33; P=0.002).

Conclusions—Having infarcts in >1 location is associated with poor performance in memory, processing speed, and executive function, independent of cardiovascular comorbidities, white matter lesions, and brain atrophy, suggesting that both the number and the distribution of infarcts jointly contribute to cognitive impairment. (Stroke. 2009;40:677-682.)

Key Words: cognition ■ epidemiology ■ MRI

Cerebral infarcts, common in older adults, increase the risk for cognitive impairment and dementia.1,2 Depending on where they are located, infarcts may disrupt cerebral circuitry and impact function on specific cognitive abilities while sparing others. Frontal–subcortical circuits are associated with memory, information processing, and executive function.3–5 Cerebro-cerebellar circuits are associated with motor control and complex higher cognitive functions, including executive function and memory.6,7 In addition to location, number of infarcts may also be related to cognitive dysfunction.1,2 Previous reports have found that, compared to having a single infarct, having ≥2 infarcts is associated with worse cognitive performance.1,2 Although there is evidence suggesting both number and location of infarcts are independently clinically meaningful with respect to cognitive dysfunction, number and location of infarcts have not been examined together. It is possible that the combination of multiple infarcts in multiple locations is a stronger predictor of cognitive impairment than either infarct alone. Characterizing the cognitive effects of these 2 infarct parameters can contribute to our understanding of brain structure–function associations and improve our identification of persons at particularly high risk for cognitive impairment.

Here we examine the role of infarct number and location on performance in 3 cognitive domains: processing speed, memory, and executive function. Data are from the population-based Age Gene/Environment Susceptibility-Reykjavik Study (AGES-Reykjavik).

Materials and Methods
The AGES-Reykjavik study is aimed at investigating the contributions of environmental factors, genetic susceptibility, and gene-
environment interactions to aging of the neurocognitive, cardiovascular, musculoskeletal, body composition, and metabolic systems. Details on the study design and the baseline AGES-Reykjavik assessments have been described elsewhere. Briefly, participants are from the cohort of men and women born in 1907 to 1935, living in Reykjavik, and who were followed-up as a part of the Reykjavik Study initiated in 1967 by the Icelandic Heart Association. In 2002, cohort members were reinvited to participate in AGES-Reykjavik. Here we report on 5764 participants who completed the AGES-Reykjavik examination, which included a structured survey instrument, cognitive testing, and brain MRI.

AGES-Reykjavik was approved by the Icelandic National Bioethics Committee (VSN 00-063), the Icelandic Data Protection Authority, and by the Institutional Review Board of the US National Institute on Aging, National Institutes of Health. Informed consent was signed by all participants.

MRI Scanning and Reading Protocol

High-resolution MR images were acquired on a 1.5-T Signa Twin-speed system (General Electric Medical Systems). The image protocol consisted of the following pulse sequences: a proton density-/T2-weighted fast-spin echo sequence (time to echo 1, 22 ms; time to echo 2, 90 ms; repetition time, 3220 ms; echo train length, 8; flip angle, 90°; field of view, 220 mm; matrix 256×256), a fluid-attenuated inversion recovery (FLAIR) sequence (time to echo, 100 ms; repetition time, 8000 ms; inversion time, 2000 ms; flip angle, 90°; field of view, 220 mm; matrix 256×256), a T2*-weighted gradient echo-type planar sequence (time to echo, 50 ms; repetition time, 3050 ms; flip angle, 90°; field of view, 220 mm; matrix, 256×256). The acquisition of these sequences were performed with 3-mm-thick interleaved slices. Additionally, images were acquired with a T1-weighted 3-dimensional spoiled gradient echo sequence (time to echo, 8 ms; repetition time, 21 ms; flip angle, 30°; field of view 240 mm; matrix 256×256, slice thickness 1.5 mm). All images were acquired to give full brain coverage and slices were angled parallel to the anterior commissure–posterior commissure line to give reproducible image views in the oblique axial plane.

A parenchymal defect (infarct) was defined as a defect of the brain parenchyme with a signal intensity that is isointense to that of cerebrospinal fluid on all pulse sequences (ie, FLAIR, T2-weighted, proton density-weighted). Cortical infarct-like lesions were defined as parenchymal defects involving or limited to the cortical ribbon and surrounded by an area of high signal intensity on FLAIR images. Subcortical infarct-like lesions were defined as parenchymal defects not extending into the cortex that are surrounded by an area of high signal intensity on FLAIR images with a minimal size diameter of 4 mm. Defects in the subcortical area without a rim or area of high signal intensity on FLAIR and without evidence of hemosiderin on the T2*-weighted gradient echo-type echo planar scan were labeled as large Virchow-Robin spaces. Large Virchow-Robin spaces were excluded from the definition of subcortical infarcts for this analysis. There were no size criteria for defects in the cerebellum. Infarcts that spanned 2 areas were assigned to the location with the largest measured (mm) diameter of the defect regardless of orientation.

Image analyses were performed in a 2-step procedure. An experienced neuroradiologist (O.K.) examined the scan for clinical abnormalities that needed immediate attention. At the same time, the neuroradiologist recorded directly into a shared database the slice location of observed cortical and cerebellar infarcts. Trained raters with access to the shared database identified subcortical infarcts and characterized all of the infarcts in more radiological detail.

White matter lesions (WML) are considered present in the case of signal intensity higher than normal white and gray matter on both T2-weighted and FLAIR images. The load of WML in the subcortical and periventricular regions is separately rated according to a scale with known properties. Briefly, the size of the lesion is measured at the largest diameter and categorized into small (≤3 mm), medium (4–10 mm), and large (>10 mm) lesions. The total load of subcortical WML of the whole brain was calculated as the weighted sum of the number and size of lesions. Pertinent WML are graded in the frontal caps, occipital-parietal caps, and bands based on size of the lesions: 0 (absent), 1 (0–5 mm), 2 (6–9 mm), and 3 (≥10 mm). A total load of periventricular WML was calculated as the sum of lesion scores.

Total brain volume and volumes of gray and white matter, cerebrospinal fluid, and white matter hyperintensities were computed automatically with an algorithm based on the Montreal Neurological Institute pipeline. The AGES-Reykjavik/Montreal Neurological Institute pipeline has been modified to accommodate full brain coverage, including the cerebellum and brain stem, multispectral images (T1-weighted 3-dimensional spoiled gradient echo, FLAIR, and proton density–T2-weighted fast-spin echo sequences), high throughput, and minimal editing. A parameter of global brain atrophy was derived from the ratio of the total brain volume and the intracranial volume.

Every 6 months the intraobserver variability for each observer and every 3 months the interobserver variability for the whole group of observers were assessed. The intraobserver weighted κ statistics were 0.89 for global WML and 0.92 for parenchymal defects; the interobserver weighted κ statistics were 0.71 for global WML and 0.66 for parenchymal defects.

Tests of Cognitive Function

The cognitive test battery included multiple tests of 3 cognitive domains. Similar to other population-based studies, composites scores for memory (MEM), processing speed (SP), and executive function (EF) were constructed based on a theoretical grouping of tests. The MEM composite includes California Verbal Learning Test immediate and delayed recall. The SP composite includes the Digit Symbol Substitution Test (DSST), Figure Comparison, and the Stroop Test parts 1 and 2. The EF composite include: Digits Backward, the CANTAB spatial working memory task, and the Stroop Test part 3.

All tests were normally distributed in the cohort and inter-rater reliability was excellent (Spearman correlations for specific cognitive tests range from 0.96–0.99). Composite measures were computed for each test by converting raw scores to standardized z scores and averaging them across the tests in each composite. A confirmatory factor analysis, previously reported, showed that the fit of the composites was adequate.

Diagnosis of Dementia

Dementia case ascertainment was a 3-step process. The Mini-Mental State Examination and the DSST were administered to all participants. Individuals who screened positive based on a combination of these tests (<24 on the Mini-Mental State Examination or <18 on the DSST) were administered a second, diagnostic test battery. Based on performance on the Trails B and the Rey Auditory Verbal Learning test (AVLT), a subset of these individuals (Auditory Verbal Learning test ≤18 or Trails B ≥8 for the ratio of time taken for Trails B/Trails A corrected for the number correct: [(time Trails B/time correct Trails B)/time Trails A/time correct Trails A]) went on to a third step. This step included a neurological examination and a proxy interview about medical history and social, cognitive, and daily functioning relevant to the diagnosis. A consensus diagnosis of dementia based on the Diagnostic and Statistical Manual (DSM)-IV guidelines was made by a panel that included a geriatrician, neurologist, neuropsychologist, and neuroradiologist. There were 316 cases of dementia diagnosed in the first 5764 AGES-Reykjavik participants.

Potential Confounders

Based on previous reports, we controlled for a number of demographic (age, sex, and education) and health-related confounding variables associated with infarcts and cognitive impairment. High depressive symptomatology was classified as a score of ≥6 on the 15-item Geriatric Depression Scale. We adjusted for the following vascular risk factors: hypertension (self-reported doctor’s diagnosis of hypertension, use of hypertensive medications, systolic blood pressure ≥140 or diastolic blood pressure ≥90); myocardial infar-
tion defined as a self-reported doctor’s diagnosis or detected on ECG, and smoking status assessed via questionnaire (ever smoker/never smoker). We also adjusted for periventricular and subcortical WML and brain atrophy.

**Analytic Sample and Strategy**

Of the 5764 participants, 281 were excluded based on standard MRI contraindications and an additional 834 had incomplete MRI scans (because of claustrophobia, equipment failure, refusal or choosing contraindications and an additional 834 had incomplete MRI scans). Of the 4614 with MRI scans, 4030 were non-demented and had complete cognitive data. Compared to those in the same location, and 7.1% (n=677). The prevalence of cortical infarcts in the AGES-Reykjavik population was 10% (n=413), of subcortical infarcts was 11% (n=447), and of cerebellar infarcts was 17% (n=677).

**Table 1. Characteristics of Nondemented Subjects by Infarct Number and Number of Locations**

<table>
<thead>
<tr>
<th>Infarct characteristics</th>
<th>No Infarcts, N=2818</th>
<th>Single Infarct, N=623</th>
<th>Multiple Infarcts, Single Location, N=302</th>
<th>Multiple Infarcts, Multiple Locations, N=287</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sociodemographic factors</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age, y</td>
<td>75.5 (5.2)</td>
<td>76.7 (5.5)†‡</td>
<td>77.5 (5.3)†‡</td>
<td>78.3 (5.3)‡</td>
</tr>
<tr>
<td>Education, % primary</td>
<td>22.5</td>
<td>20.7</td>
<td>22.9</td>
<td>21.4</td>
</tr>
<tr>
<td>Sex, % female</td>
<td>63.3</td>
<td>52.6†‡</td>
<td>44.6*</td>
<td>38.0*</td>
</tr>
<tr>
<td>Depression</td>
<td>5.7</td>
<td>5.3</td>
<td>8.1</td>
<td>9.2†</td>
</tr>
<tr>
<td>Cardiovascular risk factors, %</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Self-reported stroke</td>
<td>3.0</td>
<td>9.3§</td>
<td>10.6*</td>
<td>22.0*</td>
</tr>
<tr>
<td>Hypertension</td>
<td>77.9</td>
<td>83.1</td>
<td>86.8†‡</td>
<td>89.2†‡</td>
</tr>
<tr>
<td>Ever smoke</td>
<td>54.2</td>
<td>61.5</td>
<td>64.2</td>
<td>60.3†‡</td>
</tr>
<tr>
<td>Infarct characteristics</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cortical, %</td>
<td>0</td>
<td>23.4</td>
<td>22.8</td>
<td>69.0</td>
</tr>
<tr>
<td>Subcortical, %</td>
<td>0</td>
<td>33.7</td>
<td>21.5</td>
<td>59.9</td>
</tr>
<tr>
<td>Subcortical WML load, % top quartile</td>
<td>21.6</td>
<td>32.3*</td>
<td>34.5*</td>
<td>43.4*</td>
</tr>
<tr>
<td>Periventricular WML load, % top quartile</td>
<td>18.4</td>
<td>26.1‡</td>
<td>31.9*</td>
<td>33.2*</td>
</tr>
<tr>
<td>Brain parenchymal fraction</td>
<td>0.73 (0.04)</td>
<td>0.72 (0.04)*</td>
<td>71 (0.04)*</td>
<td>0.70 (0.04)*</td>
</tr>
<tr>
<td>N of infarcts, median (25th, 75th)</td>
<td>—</td>
<td>1.0 (1.0, 1.0)</td>
<td>2.0 (2.0, 3.0)</td>
<td>2.0 (2.0, 5.0)</td>
</tr>
<tr>
<td>Size of largest infarct, mm, median (25th, 75th)</td>
<td>—</td>
<td>5.0 (6.0, 12.0)</td>
<td>9.0 (6.0, 15.0)</td>
<td>15.0 (6.0, 24.0)</td>
</tr>
</tbody>
</table>

*P<0.001, †P<0.01, ‡P<0.05, §P<0.01 for age-adjusted comparison with no infarct group.

Data are shown as mean (SD) for continuous variables and % for categorical variables.

*P<0.001, †P<0.01 for age-adjusted comparison with no infarct group.

Results

Of the 4030 nondemented subjects with full cognitive and MRI data, 69.9% (n=2818) had no infarcts, 15.5% (n=623) had a single infarct, 7.5% (n=302) had multiple infarcts in the same location, and 7.1% (n=287) had multiple infarcts in multiple locations. Compared to participants with no infarcts, the other 3 infarct groups were older, less likely to be female, more likely to have a self-reported stroke, and lower average brain parenchymal volume (Table 1). In addition, participants with multiple infarcts were more likely to be hypertensive compared to those with no infarcts and their largest infarct was, on average, larger than the largest infarct of those with a single infarct (Table 1). Performance on the individual cognitive tests varied by infarct group; results are provided in supplemental Table I.

The prevalence of cortical infarcts in the AGES-Reykjavik population was 10% (n=413), of subcortical infarcts was 11% (n=447), and of cerebellar infarcts was 17% (n=677).
Table 2. Infarct Number and Location and Cognitive Performance in Nondemented Subjects Adjusted for Demographic and Cardiovascular Factors, Location of Infarcts, and WML

<table>
<thead>
<tr>
<th>Location of Infarcts</th>
<th>MEM</th>
<th>SP</th>
<th>EF</th>
</tr>
</thead>
<tbody>
<tr>
<td>N of Infarcts</td>
<td>N</td>
<td>β (95% CI)</td>
<td>N</td>
</tr>
<tr>
<td>No infarcts</td>
<td>2818</td>
<td>Reference</td>
<td>Reference</td>
</tr>
<tr>
<td>Single infarct</td>
<td>623</td>
<td>−0.10 (−0.17, −0.04)</td>
<td>−0.07 (−0.14, −0.01)</td>
</tr>
<tr>
<td>Multiple infarcts, single location</td>
<td>302</td>
<td>−0.07 (−0.16, 0.02)</td>
<td>−0.04 (−0.13, 0.05)</td>
</tr>
<tr>
<td>Multiple infarcts, multiple locations</td>
<td>287</td>
<td>−0.22 (−0.32, −0.13)</td>
<td>−0.16 (−0.26, −0.07)</td>
</tr>
</tbody>
</table>

Location of Infaracts
In the fully adjusted models, participants with cerebellar infarcts only did not perform different from those with no infarcts on MEM, SP, or EF (Table 3). Participants with subcortical infarcts only and those with cortical infarcts only had significantly poorer MEM performance compared to those with no infarcts (Table 3, cortical P=0.016; cortical P=0.001) but did not perform different on SP or EF.

Compared to participants with no infarcts, those who had both cortical and subcortical infarcts had significantly slower SP (P<0.001) and poorer EF (P=0.024) performance (Table 3). The join effect on SP performance of having lesions in both the subcortical and cortical regions (P<0.038) was greater than the additive effect of having infarcts in either the cortical or subcortical regions (ie, −0.05 + −0.07= −0.12; see Table 3), suggesting some synergism between cortical and subcortical infarcts on SP performance. Similar evidence for synergism of cortical and subcortical infarcts was observed for EF performance (Table 3). Because of sample size, we did not formally test for interaction between cortical and subcortical infarcts.

Presence of both subcortical and cerebellar infarcts was associated with significantly slower SP (P=0.04; Table 3). Participants with infarcts in all 3 areas had significantly lower performance on all 3 cognitive composites.

Table 3. Infarct Location and Cognitive Performance in Nondemented Subjects Adjusted for Demographic and Cardiovascular Factors, Cerebral Infarcts and WML

<table>
<thead>
<tr>
<th>Location of Infarcts</th>
<th>Memory</th>
<th>Processing Speed</th>
<th>Executive Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>N of Infarcts</td>
<td>N</td>
<td>β (95% CI)</td>
<td>N</td>
</tr>
<tr>
<td>No infarcts</td>
<td>2818</td>
<td>Reference</td>
<td>Reference</td>
</tr>
<tr>
<td>Cortical infarcts only</td>
<td>215</td>
<td>−0.22 (−0.32, −0.11)</td>
<td>−0.17 (−0.28, −0.07)</td>
</tr>
<tr>
<td>Subcortical infarcts only</td>
<td>275</td>
<td>−0.14 (−0.24, −0.05)</td>
<td>−0.12 (−0.21, −0.02)</td>
</tr>
<tr>
<td>Cerebellar infarcts only</td>
<td>435</td>
<td>0.01 (−0.08, 0.08)</td>
<td>0.02 (−0.05, 0.10)</td>
</tr>
<tr>
<td>Cortical and subcortical</td>
<td>45</td>
<td>−0.29 (−0.52, −0.07)</td>
<td>−0.22 (−0.44, 0.01)</td>
</tr>
<tr>
<td>Cortical and cerebellar</td>
<td>115</td>
<td>−0.18 (−0.32, −0.04)</td>
<td>−0.13 (−0.28, 0.01)</td>
</tr>
<tr>
<td>Subcortical and cerebellar</td>
<td>89</td>
<td>−0.21 (−0.37, −0.05)</td>
<td>−0.14 (−0.31, 0.02)</td>
</tr>
<tr>
<td>Infarcts in 3 locations</td>
<td>38</td>
<td>−0.31 (−0.55, −0.06)</td>
<td>−0.23 (−0.47, 0.01)</td>
</tr>
</tbody>
</table>

AGES-Reykjavik, N=4030.
*P<0.05; † adjusted for age, education, sex, and depression; ‡ additional adjustments for subcortical and periventricular white matter lesions, brain atrophy, diabetes, smoking status, hypertension, MI, and total cholesterol; §P<0.01; ¶P<0.001.
Discussion

We examined the association of location and number of cerebral infarcts to the pattern of cognitive function in a population-based sample of nondemented older adults. We found single infarcts, specifically cortical infarcts, were associated with poor memory performance. We also found compared to participants with no infarcts, those with multiple infarcts in the same location did not perform more poorly on tests of MEM, SP, or EF. Importantly, we found that multiple infarcts in multiple locations were associated with poor performance in all 3 cognitive abilities. Specifically, the combination of subcortical and cortical infarcts was associated with slower SP and poorer EF. These associations were independent of the presence of WML, brain atrophy, depressive symptomatology, and cardiovascular risk factors. In addition, we found a higher prevalence of cerebellar infarcts than has been previously reported.2,5,26

This study has several strengths. Findings are based on a large population-based cohort, which is well-characterized. This allowed us to adjust for several factors that have not been assessed in previous studies. We adjusted for other brain lesions, which could explain the association of infarcts to cognitive performance. Further, we could exclude those with clinical dementia to reduce the potentially overwhelming effect of whatever pathology underlies the dementia and cannot be detected on MRI. A large sample reduces the impact of lower precision associated with good but not excellent inter-rater reliability. We derived composite cognitive test scores, which are more stable measures of cognitive domains than any one single test. Finally, we examined the association of cognitive performance to the occurrence of cerebral infarcts with a prevalence as low as 1% in the cohort.

However, results should be interpreted with caution. We did not have sufficient power to investigate thoroughly the joint associations of multiple infarcts on multiple locations to cognitive performance. Both depression and cardiovascular risk factors may have an adverse effect on cognitive performance through multiple brain pathologies. Although we adjusted for these factors as well as other brain pathology visible on MRI, there may be residual confounding of these variables attributable to unmeasured risk factors or pathology not visible on our MR images. Finally, these results are cross-sectional and need to be repeated in longitudinal studies.

Previous studies have shown a higher risk of cognitive impairment and dementia in the presence of multiple infarcts compared to single or no infarcts.1,2 We extend these findings to include not just the number of infarcts but the location of the infarcts. We find that those with multiple infarcts in a single location have slightly lower performance compared to those with no infarcts, but those with multiple infarcts in multiple locations had the lowest performance on all 3 abilities.

There are several mechanisms by which multiple infarcts in multiple locations may impact cognitive performance to a greater extent than a single infarct or multiple infarcts in a single location. Compensatory brain responses that contribute to plasticity and repair after infarcts may be inefficient when multiple brain regions are infarcted.27 In the case of a single infarct or infarctions in a single location, function may be retained if neural networks, such as frontal-subcortical circuits,2 enlist secondary areas that are connected to the injured area, restoring function. This compensation may be disrupted with multiple infarcted areas in the brain.

There was a high prevalence of cerebellar infarcts in our cohort (17%), higher than that reported in many other population-based cohorts.2,5,26 This is potentially attributable to differences in the age of the sample5 or spatial resolution (slice thickness and matrix size) of the MR images acquired in our study compared to others.2,26

In conclusion, we found that having multiple infarcts in multiple locations, but not multiple infarcts in a single location, was associated with poor performance in MEM, SP, and EF. These findings suggest number or location alone may not be adequate to describe the functional impairments caused by infarcts. To further clarify the association of infarct location and function, longitudinal studies are needed, as are additional studies with more detailed mapping of infarct location, functional MRI, and tractology as delineated from diffusion tensor imaging.

Sources of Funding

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Disclosures

None.

References


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In the article entitled “Cerebral Infarcts and Cognitive Performance: Importance of Location and Number of Infarcts” by Saczynski et al., the authors would like to alter a sentence under the “Materials and Methods” section. In the second paragraph of the “MRI Scanning and Reading Protocol” subheading, the sentence “Subcortical infarct-like lesions were defined as parenchymal defects not extending into the cortex that are surrounded by an area of high signal intensity on FLAIR images with a minimal size diameter of 3 mm” should list “4 mm” rather than “3 mm” as the diameter. The authors regret this error.

The corrected version can be viewed online at http://stroke.ahajournals.org.