A Population-Based Study on Brain Atrophy and Motor Performance in Elderly Women

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Background. Brain atrophy is a common neuroimaging finding in healthy elderly individuals as well as in patients with movement-related disorders. The relationship between brain atrophy and motor changes has not been frequently reported. This study investigates this relationship.

Methods. A population-based sample of women (N = 238), aged 70, 74, and 78 years, living in Göteborg, Sweden, participated in this study. Motor performance was measured by a laboratory test, the Postural-Locomotion-Manual test, which precisely measures the subject’s mobility of lower and upper extremities using an optoelectronic technique. Cortical and central atrophy were rated on computerized tomographic (CT) scans of the brain.

Results. In bivariate analysis, temporal lobe atrophy, high sylvian fissure ratio, and high bicaudate ratio were correlated with impaired mobility. The association between temporal lobe atrophy and high sylvian fissure ratio and poor mobility remained after controlling for age, smoking, coronary heart disease, diabetes mellitus, hypertension, and white matter lesions on CT scans.

Conclusions. Our results suggest that temporal lobe atrophy, which is often seen on brain imaging in elderly persons, might be an important brain abnormality related to motor impairments in elderly women. Further studies to investigate this relationship and its underlying mechanisms are needed.

Motor impairment is common in elderly persons, but its underlying mechanisms are still incompletely understood. The development of computerized tomography (CT) and magnetic resonance imaging (MRI) makes it possible to study the relationship between the morphological changes of the brain and motor impairment. Brain atrophy is a common finding on CT and MRI scans in healthy elderly subjects (1–3), as well as in patients with movement-related disorders, such as Parkinson’s disease (4), Alzheimer’s disease (5–7), and normal pressure hydrocephalus (8). However, few studies have previously reported on the relationship between brain atrophy and motor changes (9–14), and they included small samples or limited measurements of brain atrophy.

The present study explores the relationship between brain atrophy on CT scans and motor changes measured by a laboratory test, the Postural-Locomotion-Manual (PLM) test (15–17), which objectively and precisely measures the subject’s mobility of lower and upper limbs, in a population-based sample of women aged 70, 74, and 78 years living in Göteborg, Sweden.

Methods
This study is part of the Women’s Health Study (18,19) and the H70 Study (20,21), which are longitudinal studies on representative samples in Göteborg, Sweden. The Women’s Health Study is a population study of women that started in 1968, and the H70 Study is a longitudinal gerontological and geriatric population study that started in 1971.

In 1992/93, all 591 women aged 70, 74, and 78 years in the Women’s Health Study and the H70 Study were invited to participate in a PLM test and CT scanning of the brain. Two hundred and fifty-three women underwent both a PLM test and a CT scan. Nonresponse was mainly due to refusal and difficulties in performing the examinations. Nonparticipants were more dependent in instrumental activities of daily living (IADLs; p < .001) (22), reported more difficulties in walking indoors (p = .003) and walking outdoors (p = .020), and had a higher prevalence of dementia according to the Diagnostic and Statistical Manual of Mental Disorders, Revised Third Edition, criteria (23) (6.3% vs 2.0%, p = .003) than participants. However, there were no differences regarding age, prevalence of hypertension, coronary heart disease, or diabetes mellitus between participants and nonparticipants. Five women with dementia and 10 with a history of stroke or infarcts on CT were excluded, leaving 238 women (125 aged 70 years, 88 women aged 74 years, and 25 women aged 78 years) for the present study.

PLM Test
An optoelectronic measuring technique was used in the PLM test. Six markers were placed on the right side of the head, shoulder, elbow, hip, and ankle, and on the left foot of each individual. The seventh marker was placed on the test object, a metal handle fastened to a cylindrical horizontal
plate weighing 550 g. The camera system emitted infrared light, which was reflected by the markers and registered in a computer. The positions of the markers were measured 50 times each second (Figure 1).

The participants were asked to pick up the object on the floor at their feet, walk forward 150 cm, and place it on a shelf at the height of their chins. Once the object was placed on the shelf, the subjects immediately went back to the starting point carrying the object. Each individual performed the PLM motor act from the floor to the shelf multiple times as fast as possible during 30 seconds, and mean values of all PLM motor acts were calculated. The PLM movement included three phases: namely, raising the body (the postural [P] phase), walking forward (the locomotion [L] phase), and the goal-directed arm movement (the manual [M] phase). The movement time (MT) in the PLM test was defined as the time from the moment when the object was lifted from the floor until it was placed on the shelf, and was regarded as an indicator of the overall motor performance. As the three PLM phases were partly performed simultaneously, a simultaneity index (SI) was calculated to show the degree of simultaneity of the three PLM phases (the sum of the P, L, and M phase duration divided by the MT). A higher value of SI indicated better motor coordination of the three phases. For each motor act from the floor to the shelf, the MT, the duration of each movement phase (P, L, and M phases), and SI were automatically determined by the computer. The PLM test has been described in more detail in previous studies (15–17).

CT Evaluation

All CT scans were performed without contrast enhancement and with 10-mm continuous slices on Philips Tomoscan 310 (Philips, Eindhoven, The Netherlands) or on General Electric 8800 equipment (General Electric, Milwaukee, WI). The scans were examined by two experienced radiologists blinded to the subjects’ clinical characteristics.

The cortical atrophy of occipital, parietal, frontal, and temporal lobes was categorized using a 3-point scale according to the extent of sulcal widening (normal, moderate, and severe) (24,25). Ventricular and sylvian fissure size were measured using a transparent metric ruler as described by de Leon and colleagues (24). The following linear distances were measured: (a) the bifrontal span of the lateral ventricles, (b) the width of the lateral ventricles at the head of the caudate nucleus, (c) the minimum width of the bodies of the lateral ventricles at the waist, (d) the greatest width of the third ventricle, and (e) the sum of the greatest widths of the left and right sylvian fissures. Ratios for (a), (b), (c), and (e) were determined by dividing the obtained values by the internal diameter of the skull at the level of the measurement, giving the following ratios: bifrontal ratio, bicaudate ratio, cella media ratio, and sylvian fissure ratio.

White matter lesions were defined as low-density areas in the periventricular or subcortical white matter. Decreased density was subjectively rated as no, mild, moderate, or severe, in relation to the attenuation of normal white matter (26,27). The rating procedures were carried out separately for brain atrophy and white matter lesions.

Chronic Medical Conditions

The presence of chronic conditions was ascertained from each subject’s medical history, a physical examination performed by a physician, and laboratory examinations. Coronary heart disease was defined as meeting one or more of the following criteria: angina pectoris (28), history of myocardial infarction, or pathological Q waves and/or ST depression on an electrocardiogram (29). Hypertension and diabetes mellitus were defined as having a history of treatment.

Statistical Analysis

Differences between participants and nonparticipants regarding dependence in IADLs, difficulties in walking indoors and outdoors, and prevalence of dementia, hypertension, coronary heart disease, or diabetes mellitus were investigated by Mantel-Haenszel test with age as the background factor.

Spearman rank correlation coefficients were used to investigate the association between PLM variables and atrophy of temporal, frontal, occipital, and parietal lobes. Pearson correlation coefficients were performed to study the relationship between PLM variables and bifrontal ratio, bicaudate ratio, cella media ratio, third ventricle width, and sylvian fissure ratio. Some PLM variables, the bicaudate ratio, and the sylvian fissure ratio had skewed distributions. They were, therefore, transformed into more symmetric distributions by taking logarithms in Pearson correlation coefficients. Partial correlation coefficients were used to further explore the associations between brain atrophy and the PLM test, after adjustment for age, smoking, diabetes mellitus, hypertension, coronary heart disease, and white matter lesions on CT scans.

Differences of the PLM test and brain atrophy between women with and without diseases were studied by a permutation t test or χ² test.

Two-tailed tests of significance were used in all calculations; p < .05 was considered statistically significant.

RESULTS

Among 238 women aged 70, 74, and 78 years, 44% had temporal lobe atrophy, 33% had frontal atrophy, 34% had...
occipital atrophy, and 34% had parietal atrophy on CT. Five percent of the women had diabetes mellitus, 26% had hypertension, 22% had coronary heart disease, and 24% had white matter lesions on CT; 13% were tobacco smokers.

Table 1 shows unadjusted correlation coefficients between the PLM test and CT scan variables. Temporal lobe atrophy correlated with prolonged MT ($r = .20, p = .002$), L phase ($r = .19, p = .003$), and M phase ($r = .20, p = .002$) in the PLM test. A high sylvian fissure ratio correlated with a long MT ($r = .17, p = .013$) and M phase ($r = .15, p = .018$). A high bicaudate ratio correlated with a long L phase ($r = .15, p = .024$).

Partial correlation coefficients between the PLM test and brain atrophy are presented in Table 2, after adjustment for age, smoking, diabetes mellitus, hypertension, coronary heart disease, and white matter lesions on CT. The adjusted association was generally weaker than that in the bivariate analyses. However, temporal atrophy was still significantly associated with a prolonged MT ($r = .17, p = .010$), L phase ($r = .16, p = .018$), and M phase ($r = .19, p = .004$), and a high sylvian fissure ratio was associated with a long MT ($r = .13, p = .045$) and M phase ($r = .14, p = .029$).

The study sample was further divided into two groups: a disease group ($n = 126$; with one or more of the following diseases: hypertension, diabetes mellitus, coronary heart disease, and white matter lesions on CT) and a nondisease group ($n = 112$; without any of those diseases). Women with diseases showed a longer MT ($p = .034$) and L phase ($p = .037$) and lower SI ($p = .044$) than women without diseases. Brain atrophy variables were not different between the two groups. The relationship between brain atrophy and the PLM test was studied again in women without those diseases. Temporal lobe atrophy was still associated with a long MT ($r = .21, p = .033$), L ($r = .23, p = .018$), and M phase ($r = .24, p = .012$), after adjustment by age.

**DISCUSSION**

Temporal lobe atrophy, high sylvian fissure ratio, and high bicaudate ratio on CT were associated with impaired mobility in this population-based sample of women without dementia, history of stroke, or infaracts on CT. After controlling for age, smoking, coronary heart disease, diabetes mellitus, hypertension, and white matter lesions on CT scans, temporal lobe atrophy and high sylvian fissure ratio, which is an indicator of cortical atrophy and often occurs together with temporal lobe atrophy (1), were still associated with poor mobility in the PLM test.

Studies on the relationship between cerebral atrophy and mobility are few, and often comprise small samples or include limited measurements of brain atrophy. A previous study (9) showed that frontal atrophy on MRI was greater in patients with impaired gait and balance than in age- and sex-matched control subjects, but other types of cortical atrophy were not considered in this study. An association between general sulcal widening on MRI and poor balance has been shown in the Cardiovascular Health Study (10). An association between larger ventricles and impaired gait or balance in elderly individuals has also been reported previously (10–13). Our results that cerebral atrophy may lead to motor impairment in elderly individuals are thus supported by other studies. Our findings that temporal lobe atrophy and high sylvian fissure ratio were especially related to motor impairment are supported by a previous study, in which the temporal horn width of the lateral ventricles and sylvian fissure width were associated with gait disturbance, independent of white matter lesions on CT in a sample of 130 patients (14).

The underlying mechanisms by which temporal lobe atrophy may contribute to motor impairment are unclear, but there are some possible explanations. First, the occipitotemporal stream, which projects from the striate cortex to the inferotemporal cortex, is one of the two major processing streams of visual information (30–31). It plays a critical role in visually guided movement (32–34). Temporal lobe atrophy may disturb this visual pathway and thus interfere with visuomotor control. Second, atrophy of medial temporal lobes on MRI and CT is an early sign of Alzheimer’s disease (5–7). Motor dysfunction, such as slow speed, poor equilibrium, and poor limb coordination, also occurs in the early stage of Alzheimer’s disease (35–37). Although dementia was an exclusion criterion, it is possible that some nondemented individuals had subtle pathological changes of Alzheimer’s disease. Whether the association between temporal lobe atrophy and motor impairments found in our

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Table 1. Bivariate Correlation Coefficients Between the PLM Test and Brain Atrophy in 238 Women Aged 70, 74, and 78 Years

<table>
<thead>
<tr>
<th>Variable</th>
<th>MT</th>
<th>P</th>
<th>L</th>
<th>M</th>
<th>SI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Temporal atrophy</td>
<td>0.20**</td>
<td>0.09</td>
<td>0.19**</td>
<td>0.20**</td>
<td>−0.08 **</td>
</tr>
<tr>
<td>Frontal atrophy</td>
<td>0.07</td>
<td>0.00</td>
<td>0.03</td>
<td>0.11</td>
<td>−0.02 **</td>
</tr>
<tr>
<td>Occipital atrophy</td>
<td>0.03</td>
<td>−0.04</td>
<td>0.02</td>
<td>0.07</td>
<td>0.04</td>
</tr>
<tr>
<td>Parietal atrophy</td>
<td>0.10</td>
<td>0.09</td>
<td>0.06</td>
<td>0.09</td>
<td>−0.02</td>
</tr>
<tr>
<td>Sylvian fissure ratio</td>
<td>0.17*</td>
<td>0.02</td>
<td>0.12</td>
<td>0.15*</td>
<td>−0.13</td>
</tr>
<tr>
<td>Bifrontal ratio</td>
<td>0.06</td>
<td>0.07</td>
<td>0.02</td>
<td>0.05</td>
<td>−0.06</td>
</tr>
<tr>
<td>Bicaudate ratio</td>
<td>0.12</td>
<td>0.05</td>
<td>0.15*</td>
<td>0.00</td>
<td>−0.08</td>
</tr>
<tr>
<td>Cellia media ratio</td>
<td>0.04</td>
<td>−0.10</td>
<td>0.03</td>
<td>0.01</td>
<td>−0.11</td>
</tr>
<tr>
<td>Third ventricle width</td>
<td>0.10</td>
<td>0.03</td>
<td>0.08</td>
<td>0.03</td>
<td>−0.09</td>
</tr>
</tbody>
</table>

*Note: PLM = Postural-Locomotion-Manual; MT = movement time; P = postural phase; L = locomotion phase; M = manual phase; SI = simultaneity index.

*p < .05; **p < .01.

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Table 2. Partial Correlation Coefficients Between the PLM Test and Brain Atrophy in 238 Women Aged 70, 74, and 78 Years

<table>
<thead>
<tr>
<th>Variable</th>
<th>MT</th>
<th>P</th>
<th>L</th>
<th>M</th>
<th>SI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Temporal atrophy</td>
<td>0.17*</td>
<td>0.08</td>
<td>0.16*</td>
<td>0.19**</td>
<td>−0.06 **</td>
</tr>
<tr>
<td>Frontal atrophy</td>
<td>0.03</td>
<td>0.00</td>
<td>−0.01</td>
<td>0.10</td>
<td>0.01</td>
</tr>
<tr>
<td>Occipital atrophy</td>
<td>0.02</td>
<td>−0.07</td>
<td>−0.03</td>
<td>0.05</td>
<td>0.06</td>
</tr>
<tr>
<td>Parietal atrophy</td>
<td>0.06</td>
<td>0.00</td>
<td>0.04</td>
<td>0.05</td>
<td>0.02</td>
</tr>
<tr>
<td>Sylvian fissure ratio</td>
<td>0.13*</td>
<td>0.01</td>
<td>0.10</td>
<td>0.14*</td>
<td>−0.09 **</td>
</tr>
<tr>
<td>Bifrontal ratio</td>
<td>0.07</td>
<td>0.08</td>
<td>0.03</td>
<td>0.06</td>
<td>−0.05</td>
</tr>
<tr>
<td>Bicaudate ratio</td>
<td>0.09</td>
<td>0.05</td>
<td>0.11</td>
<td>0.00</td>
<td>−0.06</td>
</tr>
<tr>
<td>Cellia media ratio</td>
<td>0.02</td>
<td>−0.11</td>
<td>0.00</td>
<td>0.01</td>
<td>−0.10</td>
</tr>
<tr>
<td>Third ventricle width</td>
<td>0.04</td>
<td>0.02</td>
<td>0.02</td>
<td>0.01</td>
<td>−0.05</td>
</tr>
</tbody>
</table>

Notes: Values are adjusted for age, smoking, diabetes mellitus, hypertension, coronary heart disease, and white matter lesions on computed tomography. PLM = Postural-Locomotion-Manual; MT = movement time; P = postural phase; L = locomotion phase; M = manual phase; SI = simultaneity index.

*p < .05; **p < .01.
study reflects the existence of preclinical Alzheimer’s disease needs further study.

White matter loss is an important confounding factor of brain atrophy. White matter lesions may impair motor function by damaging the long loop reflex, which is essential for adequate gait and balance (38). We have previously found an association between white matter lesions and impaired mobility of the lower extremities in this population (27). However, the association between poor mobility and temporal atrophy and high sylvian fissure ratio still remained after adjustment for white matter lesions.

The strengths of our study include the fact that it was population-based and that mobility was measured by an objective method. Some limitations also need to be considered. First, although the population was fairly representative regarding a number of health factors (19,21), the nonparticipants in the CT and PLM study were more dependent in IADLs and reported more difficulties in walking indoors and outdoors. If anything, this might have underestimated the association between brain atrophy and motor impairment. Second, subjective ratings and linear measurements on CT are rather crude measures of brain atrophy, and we did not formally test the interrater reliability in the present study. However, a good interrater reliability of this method has previously been reported by others (2,24). Third, some brain abnormalities that might cause motor deterioration, such as atrophy of the basal ganglia and cerebellum, were not considered. In addition, even if we excluded women with a history of stroke or infarct on CT, we cannot exclude the possibility that some individuals might have had small infarcts that could not be detected on brain CT scans. Finally, multiple correlation was made in this study, which may lead to false-positive findings. One way to treat this problem, a way we have used, is to give information on how much correlation has been made and report both significant and nonsignificant results. We emphasize that our new findings should be considered only suggestive until further confirmed by other studies. The finding that all significant associations were in the expected direction supports our findings.

In summary, the high prevalence of temporal lobe atrophy (44% in the present study) and its strong association with poor mobility suggest that temporal lobe atrophy might be an important brain abnormality related to motor impairments in elderly individuals. More studies to further investigate this relationship and its underlying mechanisms are needed.

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