Effect of Area-Based Deprivation on the Severity, Subtype, and Outcome of Ischemic Stroke

Stella Aslanyan, MD; Christopher J. Weir, PhD; Kennedy R. Lees, MD, FRCP; John L. Reid, DM, FRCP, FRSE; Gordon T. McInnes, MD, FRCP, FFPM

Background and Purpose—Markers of low socioeconomic status (deprivation) are associated with stroke and its causes. In the United Kingdom, area-based deprivation measures are available routinely through links with postal codes. We hypothesized that deprivation is associated with ischemic stroke risk factors, severity, subtype, and outcome.

Methods—We studied 2026 patients, each with at least 2 years of outcome follow-up by record linkage after first admission with ischemic stroke to an acute stroke unit. Baseline factors recorded routinely were age, sex, medical history, blood pressure, and stroke severity and subtype. Deprivation was assessed by the Womersley score (WS) and Murray score (MS).

Results—Higher WS and MS were associated with stroke at younger age (eg, WS linear regression coefficient \( r = -0.26 \); 95% confidence interval [CI], -0.51 to -0.01 per additional point), smoking (odds ratio [OR], 1.12; 95% CI, 1.08 to 1.17), and claudication (OR, 1.09; 95% CI, 1.01 to 1.17); WS was associated with higher systolic blood pressure \( r = 0.13 \); 95% CI, 0.02 to 0.24); and MS was associated with severe stroke. Deprivation was not associated with case fatality in univariate analysis or after correction for all baseline factors. Deprivation was associated with readmission to hospital as a result of any vascular event in univariate analysis (hazard ratio [HR], 1.05; 95% CI, 1.02 to 1.09) and after correction for all baseline factors (HR, 1.06; 95% CI, 1.02 to 1.10).

Conclusions—Tackling health inequalities in stroke should focus on stroke primary prevention by tackling deprivation, including promoting changes in lifestyle. (Stroke. 2003;34:2523-2629.)

Key Words: deprivation ■ outcome ■ stroke, ischemic

Acutestroke and disability among stroke survivors are enormous burdens on the social and healthcare systems. Ischemic stroke, the most common subtype of stroke, is frequent in older individuals and often leads to prolonged hospitalization, disability, or death. It is likely that aging of the population will increase the number of ischemic stroke events in the future. Therefore, its prevention and treatment have considerable public health significance.

Factors that influence health can be categorized into 3 broad groups: (1) factors intrinsic to the individual (genetic factors, biological and ethnic diversity, early life expectancy, lifestyles, and health behavior), (2) factors related to the health service (access to health services and their quality and use), and (3) external factors such as physical environment (air pollution, climate) or socioeconomic status (SES) (local amenities, occupation, income, and housing quality). External factors are closely related to personal behavior and lifestyle.

Deprivation is defined as “a state of observable and demonstrable disadvantage relative to the local community or a wider society or nation to which an individual, family or group belongs.” Low SES is associated with the risk of stroke.

However, specific SES indicators are not always recorded in medical studies. Among the measures of deprivation, area-based measures have an advantage because postal codes available in the United Kingdom have been shown to be useful markers of social class. Various scoring systems have been independently created to measure area-based deprivation on the basis of different data and were intended to serve different healthcare-related purposes. The Womersley score (WS) has been shown to be appropriate as a categorical measure of deprivation, and the Murray (MS) score has shown to be a continuous measure in assessments of deprivation in the Greater Glasgow National Health Service (GGNHS) area.

There are 136 postal code sectors in GGNHS. GGNHS has the largest number and proportion of people living in severely deprived areas (Carstairs deprivation category, 6 or 7) compared with the rest of Scotland. More than half of the Scottish population living in severely deprived areas and 80% of those living in deprivation category 7 areas reside within GGNHS boundaries.
We aimed to investigate the relationship between area-based deprivation, assessed by the WS and MS, and stroke baseline severity, subtype, and outcome. We hypothesized that deprivation is associated with (1) more severe stroke, (2) stroke subtype, (3) placement of patients at 1 and 3 months and 2 years, (4) all-cause mortality, (5) higher rates of hospital readmission resulting from any vascular event (RVE) (any ischemic or hemorrhagic vascular event in any vascular territory), and (5) a longer stay in the acute stroke unit.

Subjects and Methods
We studied patients who were consecutively admitted to the Western Infirmary Acute Stroke Unit in Glasgow. Patient data are collected routinely by medical personnel and stored in a database. Institutional guidelines are followed for informing patients about the recording and use of their information. Only anonymous information was examined for the purposes of this study as permitted by local ethics and European data protection guidelines.

We included patients admitted from 1991 to 1998, allowing at least 2 years of follow-up. Sample inclusion criteria were (1) age >18 years, (2) first admission to the acute stroke unit and clinical diagnosis of a new ischemic attack, (3) exclusion of tumor or hemorrhage by cerebral imaging, and (4) categorization by Oxfordshire Community Stroke Project (OCSP) classification\(^1\) as total anterior circulation infarction, partial anterior circulation infarction, posterior circulation infarction, or lacunar infarction confirmed by CT or MRI.

The postal code itself is given by a string of letters and numbers and can be interpreted hierarchically; eg, G11 6SB is Glasgow postal code area G, postal code district 11, postal code sector 6, and postal code unit SB. The postal code sector has a population of ~5000 on average, a size sufficient to provide fairly reliable estimation of the rates of common health events.\(^3\) Both deprivation scores used in this study (WS and MS) are based on postal code sector. Some codes are grouped together; eg, all the G1 and G2 areas were merged because each has a small population. Both were created by use of GGNHS area census data on 29 variables (related to the size and ownership or occupancy of housing, car ownership, occupation, social status, and age) using different statistical techniques. The WS is categorical and has 8 clusters, 1 (most affluent) through 8 (most deprived). The MS was associated with younger age, higher systolic BP, smoking, and claudication. The MS was associated with younger age, smoking, claudication, and reported alcohol consumption.

Comparing the linear models with generalized additive models using analysis of variance (ANOVA) assessed the linearity of the relationships between the deprivation scores and dependent variables.

Results
We identified 2748 consecutive admissions from May 1991 to June 1998 with ischemic stroke. Only 2183 (79%) were from the GGNHS area, 2036 (93%) of these being the first admission to the stroke unit of patients >18 years of age. Our sample comprised the 2026 patients who had known stroke outcome.

Figure 1 shows the distribution of the deprivation scores; they have similar shape and are highly correlated (Spearman nonparametric correlation coefficient, 0.79; 95% confidence interval [CI], 0.78 to 0.81). Descriptive statistics on patient age, sex, BP, and medical history and univariate associations with deprivation scores are presented (Table 1). Similar trends were seen with the WS and MS. The WS was associated with younger age, higher systolic BP, smoking, and claudication. The MS was associated with younger age, smoking, claudication, and reported alcohol consumption.

Table 2 presents the distribution of stroke severity, subtype, and outcome.

WS was not associated with baseline mNIHSS (Table 3). MS, however, was associated with the baseline mNIHSS score in both univariate and multivariate models. Interaction between deprivation scores and age in modeling baseline stroke severity was not significant, but similar analysis of the
1495 patients ≥65 years of age showed stronger relationships between deprivation scores and stroke severity.

Deprivation was not associated with stroke subtype, length of stay in the stroke unit, or patient placement at 1 or 3 months or 2 years (dead or in care versus at home) in either univariate or multivariate models (results not presented). From stroke outcome measures, we found a statistically significant association only between deprivation and all-cause mortality and RVE (Table 4). Deprivation assessed by both WS and MS was associated with RVE in all models. The scores were associated with mortality in all models except univariate and after correction for all factors; neither was WS associated with mortality after correction for age, sex, and stroke severity. Figure 2 illustrates the univariate relationship between WS and all-cause mortality and RVE presented as Kaplan-Meier curves. There is no association between mortality and deprivation and a clear gradual increase in RVE with an increase in deprivation.

The linear models presented in the study did not differ significantly from nonlinear, generalized additive models (ANOVA, \( P > 0.05 \)).

### Table 1. Distribution of Baseline Factors and Their Univariate Associations With Deprivation Scores

<table>
<thead>
<tr>
<th>Baseline Factors</th>
<th>Distribution</th>
<th>WS Per Additional Point</th>
<th>MS Per Increase by 1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male sex, %</td>
<td>48</td>
<td>1.03 (0.99–1.07)</td>
<td>1.08 (0.95–1.22)</td>
</tr>
<tr>
<td>Age, mean (SD),* y</td>
<td>71 (12)</td>
<td>−0.26 (−0.51–−0.01)†</td>
<td>−1.17 (−1.93–−0.40)†</td>
</tr>
<tr>
<td>Systolic BP, mean (SD),* mm Hg</td>
<td>126 (5)</td>
<td>0.13 (0.02–0.24)†</td>
<td>0.32 (−0.02–0.66)</td>
</tr>
<tr>
<td>Diastolic BP, mean (SD),* mm Hg</td>
<td>89 (13)</td>
<td>−0.13 (−0.39–−0.12)</td>
<td>−0.47 (−1.25–−0.31)</td>
</tr>
</tbody>
</table>

*Linear regression coefficients.
†\( P < 0.05 \).

**Discussion**

We found that patients living in more deprived areas tend to have ischemic stroke at a younger age, higher baseline systolic BP, and higher rates of stroke risk factors (smoking and claudication). An apparent association between MS and alcohol use was inconclusive as our data did not describe the extent of alcohol use.

Age-standardized first-stroke admission rates increased progressively with deprivation during 1992 to 1996.\(^{16}\) Accurate population estimates within each postal code sector and rates of referral to our hospital were not available. This study showed, however, that stroke admissions to our hospital occurred at a younger age in patients from deprived areas, implying that cumulative event rates may be even higher.

Patients living in more deprived areas had more severe stroke, with the association being stronger in older patients (≥65 years of age) both in univariate analysis and after correction for other factors that may influence stroke severity. For example, an increase in deprivation from MS = 1.5 to 1.5

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**Table 2. Distribution of Stroke Severity, Subtype, and Outcome**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Distribution</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline severity, mNIHSS, median (IQR)</td>
<td>3 (1–7)</td>
</tr>
<tr>
<td>TACI, %</td>
<td>21</td>
</tr>
<tr>
<td>PACI, %</td>
<td>36</td>
</tr>
<tr>
<td>POCI, %</td>
<td>11</td>
</tr>
<tr>
<td>LACI, %</td>
<td>32</td>
</tr>
<tr>
<td>Stay in stroke unit, median (IQR), d</td>
<td>4 (2–6)</td>
</tr>
<tr>
<td>Placement at, % (dead or in care versus at home)</td>
<td></td>
</tr>
<tr>
<td>30 d</td>
<td>70</td>
</tr>
<tr>
<td>90 d</td>
<td>59</td>
</tr>
<tr>
<td>2 y</td>
<td>65</td>
</tr>
<tr>
<td>Mortality, %</td>
<td>48</td>
</tr>
<tr>
<td>Readmission because of any vascular event, %</td>
<td>31</td>
</tr>
</tbody>
</table>

TACI indicates total anterior circulation infarction; PACI, partial anterior circulation infarction; POCI, posterior circulation infarction; and LACI, lacunar infarction.
will lead to an increase in stroke severity assessed by the mNIHSS of 1 point (all patients) or of 1.3 points (older patients). We did not find any association between deprivation and ischemic stroke subtype, patient placement at 1 or 3 months and 2 years, or length of stay in a stroke unit.

Patients from deprived areas had higher rates of RVE. This association was found for both scores after full correction for possible confounding factors. The corrected hazard ratio represents a 6% increase in readmission rates per 1-category increase in WS. Overall, the readmission risk between the 2 extremes of WS differs by 56%. Therefore, we conclude that deprivation has an independent effect on readmission to the hospital because of any vascular event. Our data indicate that if Glasgow had the same deprivation profile as Lothian (the second-largest urban area in Scotland, with lower rates than Glasgow of people living in deprived areas), 6.5 hospital admissions resulting from any vascular event would be avoided per 1000 person-years of follow-up after stroke.

Mortality was associated with residence area deprivation only after correction for age and sex and remained significant after correction for all confounding factors except smoking. After correction for smoking, the deprivation effect on mortality was statistically nonsignificant. Thus, studies that correct for only age and sex should be interpreted with caution because the results may be confounded by underlying differences in other factors such as smoking and stroke baseline severity. The point estimate and lower CI limits of deprivation are close to unity; therefore, the significance of any associations between mortality and deprivation may be considered marginal and not clinically significant.

Stroke-standardized mortality ratios rose progressively with increases in deprivation category only for the population under 65 years of age in 1991 to 1998. This discrepancy between age groups may be attributed to the earlier death of individuals at risk and the increasing mortality from causes other than cerebrovascular disease in people >65 years of age. Other possible explanations include inaccuracy of coding of stroke and underestimation of stroke cases, artifact from the relative insensitivity of deprivation measures in the elderly, and a cohort effect, with older people belonging to a generation for whom deprivation is less of a risk factor for stroke. Mortality rates and hazard ratios presented in this study relate to case fatality because they are based on a hospital-based cohort. To investigate whether the reported age discrepancies were due to differences in case fatality, we conducted Cox proportional-hazards modeling of mortality for the subgroup of our sample <65 years of age (n=567). The results were not substantially different from the results for the entire sample. In addition, there was no statistically significant association between deprivation and stroke severity in that subgroup. Therefore, we conclude that the discrepancies in mortality presented are due to stroke occurring at younger ages in people who live in deprived areas and not to differences in stroke severity or case fatality.

There are limitations in using area-based deprivation scores as a measurement of SES. The scores are based on the assumptions about factors that may represent deprivation; for instance, car ownership may be needed in some households that commute to work or have bigger families and therefore may not represent access to material resources but rather a drain on resources. Areas are not internally homogeneous, and sectors containing a mixture of deprived and less deprived households will have a middle ranking. The scores from postal code sectors with small populations are more

### TABLE 3. Linear Regression Coefficients (95% CI) of the WS (Per Additional Point) and the MS (Per Increase by 1) When Predicting mNIHSS for the Sample and Subgroup of Patients ≥65 Years of Age

<table>
<thead>
<tr>
<th>Model Information</th>
<th>All Patients</th>
<th>≥65 y</th>
<th>All Patients</th>
<th>≥65 y</th>
</tr>
</thead>
<tbody>
<tr>
<td>Univariate</td>
<td>0.04 (−0.04–0.13)</td>
<td>0.10 (−0.00–0.21)</td>
<td>0.26* (0.00–0.53)</td>
<td>0.45* (0.13–0.77)</td>
</tr>
<tr>
<td>Plus age and sex</td>
<td>0.06 (−0.02–0.15)</td>
<td>0.13* (0.02–0.23)</td>
<td>0.33* (0.07–0.59)</td>
<td>0.56* (0.24–0.88)</td>
</tr>
<tr>
<td>Plus all factors</td>
<td>0.06 (−0.02–0.15)</td>
<td>0.13* (0.02–0.24)</td>
<td>0.34* (0.08–0.60)</td>
<td>0.55* (0.23–0.87)</td>
</tr>
</tbody>
</table>

*P<0.05.

### TABLE 4. Hazard Ratios (95% CI) of the WS (Per Additional Point) and the MS (Per Increase by 1) at the Different Stages of Correction for Baseline Factors

<table>
<thead>
<tr>
<th>Model Information</th>
<th>Readmission Due to Any Vascular Event</th>
<th>All Cause Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>WS</td>
<td>MS</td>
</tr>
<tr>
<td>Univariate</td>
<td>1.05* (1.01–1.09)</td>
<td>1.21* (1.08–1.35)</td>
</tr>
<tr>
<td>Plus age and sex</td>
<td>1.05* (1.02–1.09)</td>
<td>1.21* (1.01–1.36)</td>
</tr>
<tr>
<td>Plus severity</td>
<td>1.06* (1.02–1.10)</td>
<td>1.23* (1.10–1.38)</td>
</tr>
<tr>
<td>Plus BP+stroke subtype + medical history†</td>
<td>1.06* (1.02–1.10)</td>
<td>1.24* (1.10–1.39)</td>
</tr>
<tr>
<td>Plus smoking</td>
<td>1.06* (1.02–1.10)</td>
<td>1.23* (1.10–1.38)</td>
</tr>
</tbody>
</table>

*P<0.05.

†History of high BP, transient ischemic attack, stroke, myocardial infarction, angina pectoris, diabetes, high lipid level, atrial fibrillation, alcohol use, claudication, and family history of stroke.
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Figure 2. Kaplan-Meier curves of all-cause mortality and readmission to hospital as a result of any vascular event stratified by Womersley score. —, 1-2 (least deprived); — —, 3-4; — — —, 5-6; — — — —, 7-8 (most deprived).

susceptible to small variations. The ecological fallacy is an important potential limitation of area-based measurement because it is based on the false assumption that inferences can be made about individual phenomena on the basis of population observations. Not all deprived individuals live in deprived areas; conversely, many of the less deprived live in very deprived areas. Finally, scores are based on census data updated only every 10 years. Changes in scores may reflect both the true change of the deprivation of the area and the change in relative values of the components. The scores in our study are original: WS is based on 1981 census data and MS is based on 1991 census data. Patients of our sample had stroke from 1991 to 1998, up to 7 to 17 years after the census recording.

Important potential limitation of area-based measurement is the possibility of selection bias. For example, transient ischemic attack patients might have access to outpatient treatment at a cerebrovascular clinic adjacent to the acute stroke unit; during the time period of our study, 213 such patients would have met the inclusion criteria of this study. They had the same median WS and were not significantly less deprived on the MS. In addition, more affluent patients may rarely attend a private hospital, or deprived patients may conceivably have dismissed stroke symptoms. Overall, we believe that our sample is not severely biased against deprivation.

This study has several strengths. Detailed patient information allows correction for factors such as history of high BP, transient ischemic attack, stroke, myocardial infarction, angina pectoris, diabetes, high lipid level, claudication, atrial fibrillation, stroke family history, smoking, alcohol consumption, BP, stroke severity, and stroke subtype. This study has reliable follow-up because of record linkage (only 1% of missing outcome data) and good-quality data collected prospectively and routinely by trained personnel. Area-based deprivation is currently the only available measure of SES recorded and stored on a regular basis and therefore enables research with a large sample size.

In conclusion, area-based deprivation is associated with stroke risk factors and with stroke at younger age. Deprivation has an independent effect on baseline stroke severity and RVE. Deprivation does not independently affect case fatality, and the inequality is linked mostly to smoking and stroke severity. Tackling health inequalities in stroke should focus on primary prevention of stroke by improving the SES of the disadvantaged population through promoting changes in lifestyle.

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References

In industrialized countries, people with a low level of education and those who have unskilled manual work are clearly at higher risk for being afflicted by stroke than people with university education or nonmanual employment.1–5 As convincingly demonstrated in a study from Glasgow in this issue of Stroke, this is at least partly due to a more unfavorable risk factor profile (such as higher blood pressure and more smoking) among people living in deprived areas.

It comes as no surprise that there is a social patterning of risk factors for stroke.6,7 An important question is if this is entirely due to personal factors or if societal factors also contribute. Multilevel analyses have shown that the area/community a person is living in has an influence on risk factors for cardiovascular disease that goes above and beyond the individual level of education.8

Once a stroke has occurred, are affluent and deprived people treated equally? The Glasgow investigators report that early case fatality does not differ by degree of deprivation, a finding similar to what has been observed in the Scandinavian countries.2,3 However, a Canadian study showed reduced early survival after stroke in low-income people.9 Worse long-term survival after stroke in people of low social class has been reported from Finland10 and, now, from Scotland.

In the Canadian study, the effect of socioeconomic status was not small: each $10 000 increase in median neighborhood income was associated with a 9% reduction in the hazard of death at 30 days. In that study, patients with the lowest incomes were less likely to receive in-hospital rehabilitation and they had to wait much longer for carotid surgery.9 Similar observations have been made in the United States.10

The Glasgow example emphasizes that there may be remarkably large gradients of stroke between communities within one and the same city. Socioeconomic factors explain also many of the differences across ethnic groups, such as the excess stroke mortality among black Americans in the United States.11 The scenario is even bigger: stroke mortality rates between countries vary markedly with economic conditions, as exemplified in the Figure. General economic indicators seem to explain more of the variation of stroke mortality than population levels of classic cardiovascular risk factors do.12

If the risk of stroke is greater in low-income people and their survival after stroke is worse, we would expect higher stroke mortality rates in countries with lower levels of economic development. Indeed, when data from a large number of countries have been compiled, manual classes consistently have higher stroke mortality rates than nonmanual classes. This social gradient is relatively large in the United Kingdom, Ireland, and Finland; intermediate in the United States, France, and Switzerland; and relatively small in Sweden, Norway, Denmark, Italy, and Spain.13 In most countries, inequalities are much larger for stroke mortality than for ischemic heart disease mortality.13

So, what to do? The Glasgow investigators conclude: “Tackling health inequalities in stroke should focus on stroke...”

Correlation between real gross domestic product in US dollars (a measure adjusted for purchasing power) and standardized stroke mortality rates in countries in the European region of WHO (that includes also all former USSR countries). Data from the World Health Organization databases European Health for All and WHOSIS.
primary prevention by tackling deprivation, including promoting changes in lifestyle.”

The most radical interpretation of this message is: Down with the class society!

Another interpretation, lukewarm enough to be attractive to many, is that more health education is needed, with a special focus on deprived people. In fact, it is very common for scientific articles on inequity in health to end with a plea for better health education for the poor and deprived. But does it work?

In rich countries, well-educated people are the first to adopt messages about a healthy lifestyle and they are more prone to change their health behavior (see Shaper et al.14). It would seem that health education is, in itself, driving inequity. A social patterning emerges, so that people with a high level of education have more leisure-time physical activity; are leaner; have lower levels of blood pressure, cholesterol, and fibrinogen; are less often smokers; and are less likely to have diabetes.14-16 Some of this general pattern is shown also in the Glasgow study.

This is one expression of what public health experts call epidemiologic transition. This translates also into major clinical events. As the overall mortality from cerebrovascular disorders declined in Australia over 3 decades, the differences in stroke mortality rates between social classes increased considerably.17

This general model seems to apply not only to individuals but also to nations. The decline in stroke mortality in most Western countries in face of unchanged or even increasing stroke mortality in Russia and other former USSR countries has resulted in a rapidly increasing gap in the burden of stroke between East and West Europe.18

If the concept of epidemiologic transition is valid, there is cause for optimism. The theory tells that there are early and late adopters of lifestyle messages (and a large majority of modestly enthusiastic people in between).19 As messages about healthy lifestyle habits are spreading, they will ultimately also make the late adopters change. A caveat: this development, attractive as it seems, must be an oversimplification. Many “late adopters” of a healthy lifestyle are in fact “early adopters” of new unhealthy habits.

The safest and most desirable road to combat stroke as a public health problem seems to be social and economic development going hand in hand. All information available suggests that improved education and less poverty will, in itself, reduce the risk of stroke. Waiting for such wishes to come true, we as stroke physicians can start to eliminate apparent inequities in the detection and management of cardiovascular risk factors and in the treatment of stroke patients.

References
Editorial Comment—Down With the Class Society!
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