RESEARCH PAPER

Socioeconomic deprivation and provision of acute and long-term care after stroke: the South London Stroke Register cohort study

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ABSTRACT

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Background and aims Socioeconomic deprivation (SED) is associated with increased mortality after stroke. however, its associations with stroke care remains uncertain. We assessed the SED impacts on acute and long-term stroke care, and examined their ethnic differences and secular trends.

Methods We used data from 4202 patients with first-ever stroke (mean age 70.1 years, 50.4% male, 20.4% black), collected by a population-based stroke register in South London, England from 1995 to 2010. Carstairs deprivation score was measured for each patient, taking the 1st as the least deprived and the 2nd to 5th guintiles as SED, and was related to 20 indicators of care in multivariate logistic regression models.

Results Patients with SED had 29% and 35% statistically significant reductions in odds of being admitted to hospital and having swallow tests, respectively. The multivariate adjusted odds ratio (OR) for receiving five indicators of acute stroke care was 0.81 (95% CI 0.72 to 0.92). It was 0.76 (0.58 to 0.99) in black patients and 0.82 (0.71 to 0.96) in white patients; and 0.70 (0.58 to 0.84) in patients with stroke occurring before 2001 and 0.89 (0.75 to 1.05) since 2001. SED was further associated with receipt of some stroke care during 5 years of follow-up. including atrial fibrillation medication (0.63, 0.48 to 0.83), and in black patients physiotherapy and occupational therapy (0.32, 0.11 to 0.92). **Conclusions** Stroke healthcare inequalities in England exist for some important indicators, although overall it has improved over time. The impact of SED may be stronger in black patients than in white patients. Further efforts are required to achieve stroke care equality.

INTRODUCTION

Stroke is the leading cause of adult disability and is the second highest cause of death in the world.¹ Previous research has shown that people in low socioeconomic groups have a significantly increased risk of stroke.² There is also substantial evidence for the existence of a socioeconomic gradient in recurrences and mortality.²⁻⁵ However, the reasons for these findings of poor prognosis of stroke among patients with socioeconomic deprivation (SED) are not well understood. Some but not all studies have shown an association of SED with poor provision of acute care after stroke.² ⁶ This

uncertainty also holds true in high-income countries.⁵ ^{7–9} Furthermore, the SED impact on longterm stroke care remains unclear.² ⁶ Although more people of minority ethnic populations live in Western countries and are more likely to experience socioeconomic disadvantage than their white counterparts, few studies have been done to investigate socioeconomic inequalities among ethnic minorities on accessing stroke care. While many governments have campaigned to reduce health inequality in stroke for decades,⁶ ¹⁰ ¹¹ little is known about whether the SED impact on access to stroke care has changed over time.

In this study we examined the association of SED with the provision of acute and long-term stroke care, using data from a population register covering an inner city multiethnic population in England.¹² We further investigated secular trends and ethnic differences in the impact of SED.

METHODS

Patients and provision of stroke care data collection

The study population was derived from the South London Stroke Register (SLSR).^{12 13} Its method-ology has been fully described before.^{12 14 15} In brief, the SLSR is an ongoing prospective population-based stroke register set up in January 1995, recording all first-ever strokes in patients of all ages living in 22 electoral wards in Lambeth and Southwark (total population at the 2001 census was 271 817), inner city South London.^{13 16} In this study, we included all data collected until 31 December 2010.

We identified patients using multiple sources of notification by specially trained study nurses and fieldworkers, from hospital and community surveil-lances for stroke.¹³ ¹⁶ Patients or their relatives gave written informed consent to participate in the study. Patients' ethnicity was recorded by self definition of ethnic origin (2001 UK census question) stratified into white, black (black Caribbean, black African and black other) and other ethnic groups (South Asians (Indian, Pakistani, Bangladeshi), Chinese, other Asians, etc). According to patients' postcode of residence at the time of stroke, we calculated the Carstairs deprivation index score¹⁷ to measure baseline SED for each patient, as we did in previous studies.¹⁸ ¹⁹ The Carstairs index is an area-based measure of SED derived from decennial census data, using levels of male unemployment,

Table 1 Characteristics of patients with stroke across socioeconomic deprivation groups in SLSR of 1995–2010

<table-container>ParticulationPercentPercentPercentPercentAge (says, median, IQR26. (5-51.)7.17.16.014.9.90.02Age (says, median, IQR25.10.14.9.90.010.01Bals say, 16%, 16%177.15.86.10.010.01Bils (says, 16%, 16%)20.120.120.10.010.010.010.010.01Bils (says, 16%)177.522.50.012.00.010.</table-container>		Socioeconomic deprivation (Carstairs score—quintile)					
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Ape (parts), median, (QR) 72.6 (61.5-00.1) 72.3 (61.9-41.3) 0.602 Male aco, (Ys) 1707 50.8 410 48.9 0.539 Bindish, (Ys)*	Variable	n	Per cent	n	Per cent	p Value	
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White 2417 71,9 581 601 0.019 Black 655 135 202 240 Other 201 6.0 41 4.9 Viet of troke, n(%)	Ethnicity, n (%)*						
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With others in private accommodation 1568 46.7 420 49.9 Nursing home or other 671 20.0 182 21.6 Unknown 68 2.0 1.4 1.7 B1 prior to stroke, n (%) 20 74.1 622 74.0 0.695 15-19, mid disability 477 14.2 117 13.9 0-14, moderate-severe disability 224 6.7 55 6.5 Unknown 170 5.1 47 5.6 5 Vorke subtype, n (%) 171 73.5 6.31 75.0 0.575 Infarction 2471 73.5 6.31 75.0 0.575 Subarachnoid haemorrhage (SAH) 170 5.1 42.2 5.0 0.129 Glagor cora scale score, n (%) 173 1.4 2.4 2.9 0.129 <ld>clagor cora scale score, n (%) 126 3.8 4.1 4.9 1.29 <ld>clagor cora scale score, n (%) 126 3.8 4.1 4.9</ld></ld>	Alone in private accommodation	1054	31.4	225	26.8	0.055	
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B1 prior to stroke, n (%) 2490 74.1 622 74.0 0.695 15-19, mild disability 274 6.7 75.5 6.5 Unknown 170 5.1 47 5.6 Unknown 170 5.1 47 5.6 Stroke subtype, n (%) infarction 2471 73.5 631 75.0 0.575 Primary intracerebral haemorrhage 434 12.9 106 12.6 Subarachnoid haemorrhage (SAH) 170 5.1 42 5.0 Unknown 137 4.1 24 2.9 Glasgow coma scale score, n (%) 213 7.7 211 25.1 213 (mapired consciousness) 932 27.7 211 25.1 Unknown 126 3.8 41 4.9 Incontinence, n (%) 23 6.6 6.4 7.6 Yes 1407 41.9 355 42.2 Unknown 223 6.6 6.4 7.6 Speech deficit, n (%) 7.2 2.0 0.53 Yes 1197 35.6 30.4 36.2 None 159 47.4 48 53.3 None 152 22.1<	Unknown	68	2.0	14	1.7		
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0-14, moderate-severe disability 224 6.7 55 6.5 Unknown 170 5.1 47 5.6 Stroke subtype, n (%)	15–19, mild disability	477	14.2	117	13.9		
Unknown1705.1475.6Stroke subtype, n (%)	0–14, moderate-severe disability	224	6.7	55	6.5		
Stroke subtype, n (%) Infarction 2471 73.5 631 75.0 0.575 Primary intracerbral haemorrhage 434 12.9 106 12.6 Subarachonid haemorrhage (SAH) 170 5.1 42 5.0 Unclassified 149 4.4 38 4.5 Unknown 137 4.1 24 2.9 ≧13 2303 685 589 7.0.0 0.129 <13 (impaired consciousness)	Unknown	170	5.1	47	5.6		
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Primary intracerebral haemorrhage 434 12.9 106 12.6 Subarachnoid haemorrhage (SAH) 170 5.1 42 5.0 Unclassified 149 4.4 38 4.5 Unknown 137 4.1 24 2.9 Glasgow coma scale score, n (%) 2 2.1 25.1 ≥13 2303 68.5 589 7.0 0.129 <13 (impaired consciousness)	Infarction	2471	73.5	631	75.0	0.575	
Subarahnoid haemorrhage (SAH) 170 5.1 42 5.0 Unclassified 149 4.4 38 4.5 Unknown 137 4.1 24 2.9 § 137 4.1 24 2.9 § 13 2303 68.5 589 7.0.0 0.129 <13 (mpaired consciousness)	Primary intracerebral haemorrhage	434	12.9	106	12.6		
Unclassified1494.4384.5Unknown1374.1242.9Glasgow coma scale score, n (%) \geq 13230368.558970.00.129<13 (impaired consciousness)	Subarachnoid haemorrhage (SAH)	170	5.1	42	5.0		
Unknown1374.1242.9Glasgow coma scale score, n (%) ≥ 13 230368.558970.00.129<13 (impaired consciousness)	Unclassified	149	4.4	38	4.5		
Glasgow coma scale score, n (%) ≥ 13 2303 68.5 589 70.0 0.129 <13 (impaired consciousness)	Unknown	137	4.1	24	2.9		
≥13 2303 68.5 589 7.0 0.129 <13 (impaired consciousness)	Glasgow coma scale score, n (%)						
<13 (impaired consciousness) 932 27.7 211 25.1 Unknown 126 3.8 41 4.9 Incontinence, n (%) 1731 51.5 422 50.2 0.553 Yes 1407 41.9 355 42.2 0.553 Yes 1407 41.9 355 42.2 0.553 Unknown 223 6.6 64 7.6 7.6 Speech deficit, n (%) 7 7.3 149 0.136 0.136 None 1308 30.9 230 27.4 0.405 Wallow impairment, n (%) 752 52.1 448 53.3 Ves 1197 35.6 304 36.2 0.405 None 1752 52.1 448 53.3 0.405 None 1752 52.1 448 53.3 0.405 Motor deficit, n (%) 759 4.7 35 4.2 0.778 None 2634 78.4	≥13	2303	68.5	589	70.0	0.129	
Unknown1263.8414.9Incontinence, n (%)No173151.542250.20.553Yes140741.935542.2Unknown2236.6647.6Speech deficit, n (%)777Yes174351.946154.90.136None130830.923027.4Unknown58017.314917.7Swallow impairment, n (%)735.630436.20.405None119735.630436.20.405None175252.144853.30.405None deficit, n (%)735.630436.20.405None1594.7354.24.2	– (impaired consciousness)	932	27.7	211	25.1		
Incontinence, n (%) 1731 51.5 422 50.2 0.553 Yes 1407 41.9 355 42.2 Unknown 223 6.6 64 7.6 Speech deficit, n (%) 743 51.9 461 54.9 0.136 None 1308 30.9 230 27.4 7.6 Unknown 580 17.3 149 17.7 7.7 Swallow impairment, n (%) 752 52.1 448 53.3 7.4 None 1752 52.1 448 53.3 7.5 7.6 None 1752 52.1 448 53.3 7.5 7.6 Motor deficit, n (%) 71 72.3 89 10.6 7.78 None 568 16.9 142 16.9 7.78 None 568 16.9 142 16.9 7.78 Unknown 159 4.7 35 4.2 7.78	Unknown	126	3.8	41	4.9		
No 1731 51.5 422 50.2 0.553 Yes 1407 41.9 355 42.2 Unknown 223 6.6 64 7.6 Speech deficit, n (%) 7 7 7 Yes 1743 51.9 461 54.9 0.136 None 1308 30.9 230 27.4 7 Unknown 580 17.3 149 17.7 7 Swallow impairment, n (%) 7 7 7 7 7 Yes 1197 35.6 304 36.2 0.405 None 1752 52.1 448 53.3 7 Unknown 412 12.3 89 10.6 7 Motor deficit, n (%) 78.4 664 79.0 0.778 None 568 16.9 142 16.9 7 Unknown 159 4.7 35 4.2 7	Incontinence, n (%)						
Yes140741.935542.2Unknown2236.6647.6Speech deficit, n (%) </td <td>No</td> <td>1731</td> <td>51.5</td> <td>422</td> <td>50.2</td> <td>0.553</td>	No	1731	51.5	422	50.2	0.553	
Unknown2236.6647.6Speech deficit, n (%) </td <td>Yes</td> <td>1407</td> <td>41.9</td> <td>355</td> <td>42.2</td> <td></td>	Yes	1407	41.9	355	42.2		
Speech deficit, n (%) Yes 1743 51.9 461 54.9 0.136 None 1308 30.9 230 27.4 Unknown 580 17.3 149 17.7 Swallow impairment, n (%) 7 7 7 7 Yes 1197 35.6 304 36.2 0.405 None 1752 52.1 448 53.3 149 16.9 Unknown 412 12.3 89 10.6 7 7 Motor deficit, n (%) 7 7 568 16.9 0.778 0.778 None 568 16.9 142 16.9 16.9 17 Unknown 159 4.7 35 4.2 16.9 16	Unknown	223	6.6	64	7.6		
Yes174351.946154.90.136None130830.923027.4Unknown58017.314917.7Swallow impairment, n (%)735.630436.20.405Yes119735.630436.20.405None175252.144853.314Unknown41212.38910.615Present263478.466479.00.778None56816.914216.9142Unknown1594.7354.216.9	Speech deficit, n (%)						
None130830.923027.4Unknown58017.314917.7Swallow impairment, n (%) </td <td>Yes</td> <td>1743</td> <td>51.9</td> <td>461</td> <td>54.9</td> <td>0.136</td>	Yes	1743	51.9	461	54.9	0.136	
Unknown58017.314917.7Swallow impairment, n (%)Yes119735.6304Sone175252.144853.3Unknown41212.389Motor deficit, n (%)Present263478.466479.00.778None56816.9142Unknown1594.7354.2	None	1308	30.9	230	27.4		
Swallow impairment, n (%) Yes 1197 35.6 304 36.2 0.405 None 1752 52.1 448 53.3 0.405 Unknown 412 12.3 89 10.6 Motor deficit, n (%) Present 2634 78.4 664 79.0 0.778 None 568 16.9 142 16.9 0.778 Unknown 159 4.7 35 4.2	Unknown	580	17.3	149	17.7		
Yes 1197 35.6 304 36.2 0.405 None 1752 52.1 448 53.3 Unknown 412 12.3 89 10.6 Motor deficit, n (%)	Swallow impairment, n (%)						
None 1752 52.1 448 53.3 Unknown 412 12.3 89 10.6 Motor deficit, n (%) 78.4 664 79.0 0.778 Present 2634 78.4 664 79.0 0.778 None 568 16.9 142 16.9 Unknown 159 4.7 35 4.2	Yes	1197	35.6	304	36.2	0.405	
Unknown 412 12.3 89 10.6 Motor deficit, n (%) </td <td>None</td> <td>1752</td> <td>52.1</td> <td>448</td> <td>53.3</td> <td></td>	None	1752	52.1	448	53.3		
Motor deficit, n (%) 2634 78.4 664 79.0 0.778 Present 2634 16.9 142 16.9 None 568 16.9 142 16.9 Unknown 159 4.7 35 4.2	Unknown	412	12.3	89	10.6		
Present 2634 78.4 664 79.0 0.778 None 568 16.9 142 16.9 Unknown 159 4.7 35 4.2	Motor deficit, n (%)						
None 568 16.9 142 16.9 Unknown 159 4.7 35 4.2	Present	2634	78.4	664	79.0	0.778	
Unknown 159 4.7 35 4.2	None	568	16.9	142	16.9		
	Unknown	159	4.7	35	4.2		

*Patients' ethnicity was recorded by self definition of ethnic origin (2001 UK census question) stratified into white, black (black Caribbean, black African and black other) and other ethnic groups.

BI, Barthel index; SLSR, South London Stroke Register.

overcrowding, car ownership and proportion in social classes IV and V (partly skilled and unskilled) in a small area.^{17 20} The index was derived using 2001 census data for each lower layer super output area covered by the register.^{17 20} The higher the score, the

more deprived. The Carstairs deprivation index has been validated and widely used in health-related studies in the UK. $^{17-19}$

The diagnosis of stroke, using the WHO clinical definition, was verified by a study clinician, and patients were examined

within 48 h of being notified to the SLSR where possible. We obtained the clinical details at the time of maximal impairment. These included information on motor deficit, swallowing (using the 3 oz (85 ml) water swallow test), speech, visual impairments and urinary incontinence. Classification of stroke subtype (ischaemic stroke or primary intracerebral haemorrhage) was based on results from at least one of CT or MRI.

We examined a range of indicators of the processes of care after an acute stroke, and the indicators for provision of rehabilitation therapy (physiotherapy (PT) assessment within 72 h, occupational therapy (OT) within 7 days, and speech and language therapy (SALT) within 7 days) for those with deficits for PT/OT defined as visual field defects, motor deficits and sensory deficits, and for SALT, dysarthria, dysphagia and failed swallow test. Since 2005 we also investigated other interventions, including thrombolysis within 3 h of symptom onset if ischaemic stroke; receipt of aspirin at any time within the 1st week of stroke or within 48 h if ischaemic stroke; enteral feeding (nasogastric or percutaneous endoscopic gastrostomy), after a failed swallow test result; and provision of intravenous fluids.

Follow-up data were collected by validated postal or face-to-face instruments with patients and/or their carers. Patients were assessed at 3 months and annually after stroke. We examined follow-up by a specialist or general practitioner (GP) (available data from 2002), four indicators of rehabilitation therapy provision for those with recorded deficits (PT/OT and SALT \leq 1 year) and 11 indicators of appropriate management of clinical risk factors (atrial fibrillation, hypertension, hypercholesterolaemia, diabetes, antiplatelet treatment for ischemic stroke).

The study was approved by the ethics committees of Guy's and St Thomas' Hospital Trust, King's College Hospital, Queen's Square, and Westminster Hospital (London).

Statistical analysis

All analyses were performed with the STATA statistical package for Windows (V11.2, STATA Corporation, College Station, Texas, USA). Patients were divided into two groups in terms of SED status: those in the second to fifth quintiles of Carstairs scores defined as having SED and others in the first quintile as the least deprived for analysis. This is because source population of the SLSR had a higher mean Carstairs index than the general population where we have taken participants having the first tertile of Carstairs score as a reference group in the data analysis.¹⁹ We examined differences in patients' characteristics between the two SED groups, using Kruskal-Wallis method for

continuous variables and the χ^2 test for categorical variables. Multivariate adjusted logistic regression models were employed to investigate the associations of SED with short-term and longterm stroke care. We calculated ORs and their 95% CIs for provision of acute and long-term stroke care among patients with SED. We further performed stratified data analyses for black patients and white patients and for strokes occurring in 1995-2000 (ie, the earlier periods) and 2001-2010 (the later periods), a period of policy-led drives to improve the quality of stroke care in the UK. To increase the statistical power, we pooled ORs for similar indicators of stroke care in ethnicity and in the stroke occurring years, and where needed, in all patients for 3 months to 5 years follow-up, according to the standard methods which we used before.²¹ If heterogeneity of within-indicator and between-indicator variation in those selected indicators was significant, a random effect model was used; otherwise, a fixed effect model used. We tested for an interaction on between SED and period of stroke occurrence, and patients' ethnicity using a one-sided p value.²²

RESULTS

From 1 January 1995 to 31 December 2010, 4212 people with first-ever stroke were registered, of whom 4202 had the Carstairs index calculated for analysis. Their mean age at onset was 70.1 years (IQR 61.6–81.1 years), 50.4% were male and 20.4% were black patients. Table 1 shows baseline characteristics of these patients. Compared with those with the least deprived, patients with SED were more likely to be white and with stroke having occurred in the years of 2007–2010. We did not find significant differences in age, sex, living conditions, Barthel index prior to stroke, stroke subtype, Glasgow coma score, incontinence, speech deficit, swallow impairment and motor deficit between two SED groups (table 1).

Acute stroke care

Table 2 reports the specific interventions among patients after stroke. Compared with those who were the least deprived, patients with SED had significantly less chance of being admitted to hospital. There was a borderline significant association of SED with reduced swallow test, and the associations of SED with stroke unit admission, >50% of stay on stroke unit and brain imaging were not statistically significant.

After adjustment for covariables, we observed the significant associations with hospital admission and swallow tests (table 3). A small reduction in OR was found for stroke unit admission, >50% stay and brain imaging, but none of these reached

Table 2 Interventions in acute stroke in SLSR of 1995–2010*

	Socioeconomic dep	privation (Carstairs score—	-quintile)		
	Deprived (2nd–5th N=3191	Q)	Least deprived (1st Q) N=799		
Variable	n	Per cent	n	Per cent	p Value
Hospital admission, n (%)	2783/3191	87.8	721/799	90.2	0.019
Stroke unit admission,†n (%)	1518/2710	56.0	402/708	56.8	0.715
>50% of stay on stroke unit†	1135/2454	46.3	295/635	46.5	0.926
Brain imaging, n (%)	2827/3044	92.9	719/771	93.0	0.710
Swallow test,†n (%)	2524/2768	91.2	665/713	93.3	0.074

Values are numbers of patients with process/total number of patients with data on process measure (%) unless stated otherwise.

*Patients with a subarachnoid haemorrhage were excluded in this analysis because they have differing needs for acute care and are typically managed in neurosurgical wards following different protocols. All data analysis excluding patients with subarachnoid haemorrhage (total n=3990 remained).

†Limited to patients admitted to hospital.

SLSR, South London Stroke Register.

Table 3 OR for receiving	provisio	n of acute strok	e care in SL	SR of 15	95-2010*										
	Hospitä	al admission		Stroke L	unit admission†		>50% o	f stay on stroke ı	unit†	Brain im	aging		Swallow	v test†	
Socioeconomic deprivation	OR‡	95% CI	p Value	OR‡	95% CI	p Value	OR‡	95% CI	p Value	OR‡	95% CI	p Value	OR‡	95% CI	p Value
All patients															
Deprivation															
No	1.00			1.00			1.00			1.00			1.00		
Yes	0.71	(0.54 to 0.94)	0.017	0.87	(0.71 to 1.07)	0.182	0.86	(0.68 to 1.08)	0.194	0.93	(0.55 to 1.58)	0.791	0.65	(0.45 to 0.95)	0.023
Subgroup data analysis															
By ethnicity															
Black§	0.52	(0.26 to 1.04)	0.064	0.76	(0.48 to 1.18)	0.216	0.81	(0.51 to 1.28)	0.358	0.58	(0.10 to 3.39)	0.543	1.08	(0.48 to 2.45)	0.845
White§	0.74	(0.53 to 1.03)	0.076	0.89	(0.69 to 1.14)	0.338	06.0	(0.68 to 1.19)	0.461	0.80	(0.43 to 1.52)	0.500	0.62	(0.39 to 0.97)	0.039
By period of stroke															
1995-2000§	0.60	(0.41 to 0.89)	0.010	0.76	(0.57 to 1.02)	0.067	0.71	(0.47 to 1.08)	0.112	0.83	(0.42 to 1.63)	0.585	0.50	(0.22 to 1.14)	0.100
2001-2010§	0.79	(0.52 to 1.20)	0.274	0.97	(0.73 to 1.30)	0.854	0.97	(0.73 to 1.30)	0.854	0.98	(0.37 to 2.62)	0.965	0.68	(0.44 to 1.04)	0.078
*All data analysis excluding patit tLimited to patients admitted to #OR adjusted for age, sex, ethnic §OR was for patients having soci BI, Barthel index; SAH, Subarachr	ents with 5 hospital. city, living 6 oeconomic noid haemo	AH. conditions before str : deprivation in com orrhage; SLSR, South	oke, period of parison with th t London Strok	stroke, Bl F ose having e Register.	rior to stroke, stroke not.	e subtype, Glas	sgow come	a scale sore (≥13), i	ncontinence, s	peech defi	cit and motor deficit				

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conventional statistical significance level. We pooled OR for receiving these five indicators of acute care and found a significantly reduced OR of 0.81 (95% CI 0.72 to 0.92) in all patients.

Stratified data analysis for ethnicity and for the period on which stroke occurred showed that there may be a stronger SED impact in black patients than in white patients (except for swallow test), and in the earlier period compared with the later period (table 3). The pooled-OR for receiving five indicators was 0.76 (0.58 to 0.99) in black patients with SED and 0.82 (0.71 to 0.96) in white patients, interaction p=0.314, and 0.70 (0.58 to 0.84) in SED patients with stroke occurring before 2001 and 0.89 (0.75 to 1.05) with stroke occurring since 2001, interaction p=0.030 (figure 1).

Data on receipt of interventions in acute care showed no statistically significant differences among two SED groups in receiving these interventions including thrombolysis, aspirin use started at any time in acute phase and aspirin use started 48 h after stroke, (see online supplementary table S1). Multivariate adjusted analysis also suggested no significant association of SED with each intervention and with their combinations. However, in black patients, after pooling the ORs from the six interventions combined (thrombolysis, anytime aspirin, fed by enteral route, intravenous fluids, PT/OT and SALT), we found a statistically significant impact of SED (pooled-OR 0.48, 0.32 to 0.72), which was stronger that in white patients (0.89, 0.70 to 1.13), interaction p=0.005.

Long-term stroke care from hospital discharge to 5 years follow-up

Of the total sample, 1097 (26.0%) died before 3 months, 2037 (48.4%) had a 3-month assessment and 1078 (25.6%) were lost to follow-up (declined, not traced in time, or moved away). Two thousand six hundred and sixty patients survived up to 1 year and reached the 1 year follow-up point and 1917 were then reassessed. The matched figures in years 2, 3, 4 and 5 were 2287 (1310), 1932 (1363), 1658 (1139) and 1406 (872), respectively. There were no statistically significant differences in SED between patients reassessed and not reassessed, for example, p=0.279 at 5 year follow-up.

In the follow-up, we collected data on access to specialist/GP at 3 months and 1 year. Multivariate adjusted OR for patients with SED receiving specialist/GP was not significantly low, at 3 months 0.75 (0.52 to 1.10) and at 1 year 0.79 (0.36 to 1.73). However, the OR at 3 months was significantly reduced among those whose stroke occurred in the later period (0.56, 0.31 to 0.98). No interaction effect was found for ethnicity and for period in which stroke occurred.

Table 4 shows number, percentage and OR for receiving atrial fibrillation medication in eligible patients over 5 years follow-up. The association of SED with receiving less atrial fibrillation medication was found at 3 months and 5 years, but not significantly at years 2–4. The pooled data from 3 months to years 1, 2, 3 and 5 showed an OR of 0.63 (0.48 to 0.83). In the stratified data analysis for ethnicity and for period on which stroke occurred, we only observed a significant OR of 0.40 (0.19 to 0.82) at 3 months for stroke occurring in the later period.

In data on prescribing medications for antihypertension, lowering blood cholesterol and glucose, and antiplatelet in eligible patients, we did not observe that they were significantly related to SED, except for medication for lowering blood cholesterol at 3 months (multivariate adjusted OR 0.51, 0.32 to 0.83) and for lowering glucose at 2 years (0.34, 0.15 to 0.80). The stratified data analysis for ethnicity and for stroke years showed no



Figure 1 Combined OR for receiving five indicators of care after stroke in patients with socioeconomic deprivation (SED) by ethnicity and by the period in which stroke occurred.

significant association of SED with these medications at each time follow-up, except for lowering glucose medication (in black patients, 0.33, 0.11 to 0.95 at 3 months and 0.16, 0.03 to 0.96 at 2 years).

There was no significant association of SED with receiving PT/OT and SALT at 3 months and 1, 2, 3, 4 and 5 years after stroke. However, in the stratified data analysis we found that the significantly reduced OR was for black patients at year 3 to receive PT/OT (0.32, 0.11 to 0.92).

DISCUSSION

In this multiethnic population of patients with stroke with longterm follow-up, we found significant associations of SED with many provisions of acute and long-term stroke care. More indicators of stroke care seemed to be inversely related to SED in black patients than in white patients. There were more inequalities in stroke care in those whose stroke occurred before 2001.

To our knowledge the current study is the first to compare SED impact on stroke care between white patients and black patients who live in high-income countries. The SLSR data includes the largest proportion of black and minority ethnicity patients in the world. Although living in the same society in the UK, black people generally have lower levels of education and occupational class and are poorer than white people. These offered a unique opportunity for us to identify the association of SED with stroke care. A second strength is that our study covered a long time period, with meticulous follow-up of the patients with stroke. This allowed us to examine the impact of SED on longer-term stroke care (which has been lacking in study, before) and its secular trend, evaluating health policies. Our study has some limitations. Although the study included a large proportion of black and minority ethnicity patients, when we stratified data analysis for ethnicity the number of events was small, giving a wider CI in adjusted OR, which needs to be

taken with caution when interpreting the findings. However, we employed meta-analysis techniques to pool data to further examine the association of SED with stroke care; the ethnic differences in the SED impacts were tested for significance. Thus the overall findings are robust. We had limited data on some indicators of stroke care in terms of collection from 2005, for example, receiving intervention for acute stroke, and we could not examine differences in the impact of SED between the early and the later periods. We do not have data on personal or family income for each patient, while data of educational level and occupational class include substantial missing values. We could therefore not analyse their associations with provision of stroke care. We will carry out more studies to investigate these, including breaking down Carstairs index into several components (eg, education, occupation) and further follow-up the cohort.

Previous studies reported some significant associations between SED and stroke care, mainly acute care. In Nordic countries, Langagergaard *et al*⁵ found that low-income patients and disabled pensioners were less likely to receive seven specific processes of care (including stroke unit care, scan, antiplatelet or anticoagulation, assessment by physiotherapist or OT) after stroke. The FINMONICA stroke register (a Finnish contribution to the World Health Organization's MONICA project (MONItoring of trends and determinants of CArdiovascular disease)⁷ reported that patients from high-income groups were more likely to be treated at a university hospital, be examined by a neurology specialist and have CT or MRI. In North America, patients in the lowest-income group were less likely to receive inhospital rehabilitation treatments and waited longer for carotid endarterectomy,²³ while patients from higher socio-economic groups were more likely to receive post acute stroke rehabilitation.²⁴ In China, patients with stroke with lower income and those without medical insurance were less likely to receive antithrombotic therapy.²⁵

 Table 4
 Associations between socioeconomic deprivation and access to atrial fibrillation medical follow-up in SLSR of 1995–2010

	Atrial f	ibrillation medicat	tion*					
	Yes		No			Multivar	iate adjusted	
Socioeconomic deprivation	n	Per cent	n	Per cent	p Value	OR†	95% CI	p Value
At 3 months								
Deprivation								
No	45	35.2	47	18.5				
Yes	83	64.8	207	81.5	0.000	0.46	0.27 to 0.79	0.005
At 1 year								
Deprivation								
No	37	30.8	53	22.5				
Yes	83	30.7	183	77.5	0.086	0.75	0.42 to 1.31	0.311
At 2 years								
Deprivation								
No	22	25.3	37	20.6				
Yes	65	74.7	143	79.4	0.382	0.93	0.45 to 1.94	0.856
At 3 years								
Deprivation								
No	27	29.0	33	22.3				
Yes	66	71.0	115	77.7	0.239	0.69	0.33 to 1.43	0.316
At 4 years								
Deprivation								
No	19	25.0	33	23.7				
Yes	57	75.0	106	76.3	0.837	0.79	0.36 to 1.72	0.554
At 5 years								
Deprivation								
No	21	32.8	19	16.7				
Yes	43	67.2	95	83.3	0.013	0.39	0.17 to 0.88	0.024

*Based on patients having atrial fibrillation.

tOR—adjusted for age, sex, ethnicity, living conditions before stroke, period of stroke, BI (each analysis changed at its data collection), stroke subtype, Glasgow coma scale sore (≥13), incontinence, speech deficit and motor deficit.

BI, Barthel index; SLSR, South London Stroke Register.

In the current study, we found that there were significant associations between SED and multiple components of acute and long-term stroke care. The most important finding is that there was a stronger impact of SED on provision of stroke care in black patients than their white counterparts. Previous studies²⁶ mainly investigated ethnic differences in stroke care, showing that black patients with stroke received fewer evidence-based care processes. The current study has identified that within black patients SED was associated with poor stroke care, and the association may be stronger than in white patients. This suggests that addressing healthcare inequality in stroke in black patients may be behind that in whites, and strategies for reducing health inequality should target on this population.

Our study has shown that the impact of SED appears to have attenuated with time, which may be associated with changes in health policies, organisation of stroke services and the application of new scientific evidence into clinical practice. It has probably reflected increased efforts in the UK to improve the quality of stroke care through a raft of policy initiatives including the National Stroke Strategy¹⁰ and a rolling national programme of audit of clinical guideline implementation.^{11 27} This is a good achievement in public health. However, significantly reduced ORs for specialist/GP follow-up and for atrial fibrillation medication were observed in the later periods, and black patients appeared to still have healthcare inequality in stroke. Our findings could help improve making of health policies.

We did not observe significant associations of SED with other indicators of stroke care, for example, antihypertensive and antiplatelet medication, SALT, PT/OT (except in black patients). This may suggest that the government has dealt with health inequality in stroke care on these aspects or these are more easily established as routine care for all patients with stroke. An Austrian study also suggested no socioeconomic differences in the administration of thrombolysis, or the rate of prescribing secondary prevention drugs.⁸ Equal access to stroke unit care and an apparent equity in thrombolysis provision among all socioeconomic groups was reported in patients from three Scottish hospitals with universal access to care.⁹ However, the SLSR data showed that patients with SED had less chance of being given medication for atrial fibrillation in acute and longterm phases. It is an interesting finding in the current study. This would have an important implication and may help explain a high level of mortality in patients with stroke with SED. We need to carry out a further study to investigate its reasons and reduce inequality in secondary stroke prevention. Nevertheless, our study is of timely importance for clinicians and health policy makers for reducing healthcare inequality and improving the prognosis of stroke.

Variations in the findings of the association between SED and stroke care provision may reflect different healthcare systems among these countries and improvement on reducing healthcare inequality in stroke over time. It may also be due to different indicators of stroke care and duration of the J Neurol Neurosurg Psychiatry: first published as 10.1136/jnnp-2013-306413 on 13 April 2014. Downloaded from http://jnnp.bmj.com/ on February 24, 2024 by guest. Protected by copyright.

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follow-up. In this long-term follow-up study, using a comprehensive measurement of SED,^{19 20} we found an overall significant association with many indicators. Recently having analysed data from Hospital Episode Statistics in England, Lazzarino *et al*²⁸ also reported that SED (measured using the Carstairs index as well) was related to less chance of being selected for emergency admission for stroke and for a brain scan on the same day of admission in patients scanned at any time in the same hospital. Our data further showed that the impact of SED was greater in black patients. All these suggest that stroke healthcare inequality still remains in England, particularly in an ethnic population. Our study may help explain why patients with SED had a poorer prognosis of stroke than patients who were not in SED.²

Conclusions

This study has shown an overall significant association between SED and reduced access to acute and long-term stroke care in a multiethnic population of patients with stroke in England. Inequalities in stroke care provision were more obviously observed in black patients than in white patients. There were improvements in some indicators over time. Reducing the poverty in black and other minority populations may help reduce health in equality in stroke nationally and internationally. Further investigation is required to understand how these improvements were achieved to address other aspects of stroke care where inequalities in access remain.

Contributors Study concept and design: RC, CDAW. Data collection and supervision: CDAW, CM, AGR. Analysis and interpretation of data: RC, SLC, CDAW. Drafting of the manuscript: RC, CM. Critical revision of the manuscript for important intellectual content: CDAW, AGR, CM, SLC, RC. Obtained funding: CDAW, AGR, CM. Administrative, technical and material support: CDAW, SLC, RC, CM. CDAW is the guarantor of this paper.

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Competing interests None.

Patient consent Obtained.

Ethical approval The study was approved by the ethics committees of Guy's and St Thomas' Hospital Trust, King's College Hospital, Queen's Square, and Westminster Hospital (London).

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Online Table 1. Proportion of patients eligible for and receiving intervention for acute stroke in SLSR of 2005-2010 $^{\$\$}$

	So				
	(0				
Variable	Deprived $(2^{nd} - 5^{th} Q)$		Least depriv	red $(1^{\text{st}} \mathbf{Q})$	P value
	N=940	5	N=24	44	
	n	%	n	%	
No with ischaemic stroke	818		192		
Thrombolysis	98/783	12.5	23/189	12.2	0.897
Aspirin started at any time in acute	635/746	85.1	151/172	87.8	0.368
Aspirin started by 48 hours after stroke	472/523	90.3	112/124	90.3	0.980
No with failed	288		69		
swallow screen† Fed by enteral route †	149/280	53.2	36/68	52.9	0.968
Intravenous fluids	521/954	54.5	125/223	56.1	0.674
No requiring physiotherapy or occupational therapy *	796		183		
Physiotherapy or occupational therapy received, n(%)	640/764	83.8	154/176	87.5	0.218
No requiring speech and language therapy, *n(%)	666		163		
Speech and language therapy received	423/625	67.7	108/156	69.2	0.710

Values are number receiving intervention/number eligible for intervention (%) unless stated otherwise

^{*}data available from 2005.[§]All data analysis excluding patients with SAH. †within first seven days of stroke. ‡ Nasogastric or percutaneous endoscopic gastrostomy. *Analysis limited to patients admitted to hospital.