Differences in the Nature of Stroke in a Multiethnic Urban South African Population

The Johannesburg Hospital Stroke Register

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Background and Purpose—The burden of stroke is increasing in Sub-Saharan Africa (SSA) as the population undergoes epidemiological and demographic transition. Little is known about the nature (risk factors, stroke type and subtype, and causes) of stroke in SSA and whether it differs from stroke in high-income populations. We aimed to compare the nature of stroke between black and white populations in South Africa.

Methods—We used overlapping sources to ascertain consecutive first-ever-in-a-lifetime stroke patients admitted to Johannesburg Hospital over 23 months. We assessed each patient's demographic details, risk factors, CT confirmed pathological stroke type, ischemic stroke subtype and stroke severity, and compared the nature of stroke between black and white stroke patients.

Results—524 patients with presumed stroke were referred. Of these, 432 were first-ever strokes; 308 patients were black and 76 white. Black patients were significantly younger (mean age 51) than white patients (61). Stroke severity was similar (median NIH stroke score 10; 95% CI 8 to 11). More black than white patients had cerebral hemorrhage (27% versus 15%), lacunar stroke (28% versus 22%) and total anterior circulation infarcts (28% versus 22%). Large vessel atherosclerosis (none detected) and ischemic heart disease were very uncommon (1%) as a cause of stroke in black patients. Hypertension (70% versus 68%) and diabetes (14 versus 15%) were as common in black and white stroke patients, but mean cholesterol levels were lower (4.6 mmol/L; 95% CI 4.3 to 4.9 versus 5.3 mmol/L; 4.8 to 5.7) and cigarette smoking less frequent in black patients (23 versus 54%).

Conclusions—Although this was a hospital-based study, the difference in the nature of stroke between black and white stroke patients likely reflects the profile of stroke risk factors. There is an opportunity to prevent an otherwise inevitable increase in atherosclerotic stroke (and IHD) by targeting dietary and smoking habits in the black South African population. **(*Stroke*. 2009;40:355-362.)**

Key Words: Africa south of the Sahara ■ South Africa ■ cerebral hemorrhage ■ cerebral infarction ■ subarachnoid hemorrhage ■ ethnic groups

The prevalence of disabling stroke in rural South Africa is already as high as in high-income countries.[1] Stroke is likely to increase in Sub-Saharan Africa (SSA) in the future as the population ages and undergoes epidemiological transition,[2–4] moving from a pattern of disease dominated by infection, perinatal illness, and other poverty related diseases to one dominated by noncommunicable diseases including vascular disease.[2,5,6] Early in this transition stroke is far more prominent than ischemic heart disease and peripheral vascular disease. Later, as the burden of atherosclerosis grows in the population, so ischemic heart disease and peripheral vascular disease increase. The nature of stroke also probably changes during this transition with the emergence of extracranial carotid and coronary atherosclerosis causing large artery and cardioembolic ischemic stroke, though the data to support this are limited.[3,7]

No previous studies have compared the nature of stroke in different ethnic groups living in SSA at the present time. However, in South Africa there is such an opportunity. The recent political history has resulted in wide discrepancies in socioeconomic status, predominantly between the black and white population groups. In the black population the main illnesses are those associated with low socioeconomic standing (early phases of the epidemilogic transition), whereas disease in the white population is similar to that seen in high-income white populations elsewhere (later stages of the epidemiological transition).[8] Knowing more of the nature of stroke in South Africa's black population will not just

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advance the development of regionally appropriate treatment and prevention strategies, but also allow comparison of the nature of stroke between the black and white populations which may provide some insight into the mechanisms of epidemiological transition.

Our aim was to compare the nature (pathological stroke type, subtypes, causes, and risk factors) of stroke in black and white urban stroke patients admitted to the Johannesburg Hospital. Although there are 4 main population groups in South Africa—blacks, whites, mixed race, and Asian/Indian—the latter two make up such a very small proportion of the population in Johannesburg that we did not anticipate that we would be able to study sufficient strokes in these populations to reliably comment on their nature.

Methods

Ethics Approval

Ethics approval for the study was granted by the University of the Witwatersrand Human Ethics Research Committee (M00/03/7).

Setting and Population

The Johannesburg Hospital is a 1088-bed academic referral hospital.[9] As well as accepting tertiary referrals, the hospital also provides immediate (primary and secondary) health care to much of the indigent population of Johannesburg.[9,10] In 2000 the population of Johannesburg included (proportion for the whole country in brackets): 72% (79%) blacks, 17% (9%) whites, 7% (9%) mixed race, and 4% (3%) Indian/Asian inhabitants.

Case Ascertainment and Assessment

The Johannesburg Hospital Stroke Register (JHSR) included all cases of stroke admitted to Johannesburg Hospital, or which occurred while in hospital, over 23 months during two periods: from July 1, 2000 to December 31, 2000 and from August 1, 2001 to December 31, 2002. The JHSR was widely advertised around the hospital and at academic meetings. We identified all patients likely to have had a stroke using multiple overlapping sources. Medical and nursing staff referred patients using a dedicated phone line with answering machine if the stroke assistant (a clerk who had coordinated the hospital neurovascular clinic for several years) was not available. Every weekday, the stroke assistant questioned medical staff in the emergency room and admission wards about admissions over the previous day, reviewed ward admission books, death certificates, and the files of patients who had died during the previous 24 hours. Over weekends, a JHSR clinician searched the emergency wards and admission records for acute strokes.

After finding likely stroke patients, the stroke assistant informed the patient or their carers about the stroke register and sought verbal consent for a doctor from the stroke team to assess them. She also provided them with an information sheet about the register in the language of their choice (English, IsiXhosa, IsiZulu, Shangaan, Sesotho, and Afrikaans were available).

Clinicians from the Division of Neurology then assessed all patients, their medical notes, and results of investigations in detail. We documented the patient's demographic details and assessed their risk factors for stroke including: hypertension, diabetes mellitus, elevated cholesterol, atrial fibrillation, past symptoms of myocardial infarction accompanied by any signs of ischemic heart disease on ECG, peripheral vascular disease (defined as intermittent calf claudication or absent leg pulses or femoral bruits or having had peripheral vascular disease surgery), cigarette smoking (current smoker, former smoker for more than one year, never smoked), alcohol consumption (never/hardly ever, exdrinker for more than one year, current alcohol use), history of transient ischemic attack, and a family history of stroke. We looked for symptoms on history and evidence from the patient's clinical record of previous stroke and documented their functional status prior to the stroke. We assigned population groups (ethnicity) according to a patient's self-defined ethnicity using the current Statistics South Africa groupings.[10]

We defined hypertension as current use of antihypertensive medication, a history of having been diagnosed as hypertensive by a doctor or nurse prior to the stroke, documented blood pressure of greater than or equal to 140 mm Hg systolic or 90 mm Hg diastolic before the stroke or more than 1 week after the stroke, or evidence of left ventricular hypertrophy on ECG or echocardiography. Diabetes mellitus was diagnosed when the patient was using antidiabetic drugs, if a doctor or nurse had diagnosed diabetes before their stroke, or if the patient had documented nonfasting blood glucose of greater than 11.1 mmol/L on admission or fasting blood glucose of greater than or equal to 7.0 mmol/L.

Our examination included a detailed neurological assessment, measurement of the patient's blood pressure, and examination for any evidence of vascular diseaseincluding hypertensive end-organ damage affecting the heart or fundal vessels and any potential sources of emboli. We assessed the person's modified Rankin score[11,12] on admission and before their stroke, and National Institutes of Health Stroke Scale (NIHSS) at the time of the examination.[13]

One of us (M.C.) reviewed the majority of patients and all questionnaires and scans personally and assigned a final diagnosis, pathological stroke type, and subtype using the Oxfordshire Community Stroke Project and TOAST classifications.[14–16] We included patients if they had had a stroke within the previous 3 months irrespective of their reason for admission or if they had had a stroke within the last 6 months if the stroke was the main reason for seeking assistance. We excluded patients who had had their stroke more than 6 months previously. We defined stroke according to the World Health Organisation (WHO) criteria as "rapidly developing signs of focal (or global) disturbance of cerebral function, leading to death or lasting longer than 24 hours, with no apparent cause other than vascular." Only individuals with first-ever-in-a-lifetime strokes were included.

We did not have much funding for this study and could not influence the investigation of stroke patients. We decided against using clinical stroke scores such as the Siriraj and Guy's Hospital stroke scores to diagnose pathological stroke type, as these were inaccurate in our experience.[17] Our findings therefore are based on information from the routine care available at the hospital during the study.

Data Analysis

We entered all questionnaires onto Microsoft Access 2002 SP3 and analyzed the data using STATA software.[18] In some instances we calculated confidence intervals using Confidence Interval Analysis software.[19] We assessed differences in patient characteristics, risk factors and investigations, between ethnic groups and by stroke type and subtype, using a χ^2 test (for categorical variables) or analysis of variance (for continuous variables). We compared median values using the Wilcoxon test, and we used logistic regression to assess the influence of age on the risk of cerebral hemorrhage. We considered a level of $P<0.05$ as statistically significant.

Results

The Figure shows the outline of cases referred to and included in the JHSR. Thirteen strokes occurred in hospital. Table 1 shows the characteristics of the stroke patients, categorized by population group and sex. The number of Mixed Race and Indian/Asian stroke patients was so small that we have excluded them from further analysis.

Almost double the proportion of black (n=55; 27%) compared to white (n=7; 15%) stroke patients who were scanned (n=254) had a cerebral hemorrhage, though this

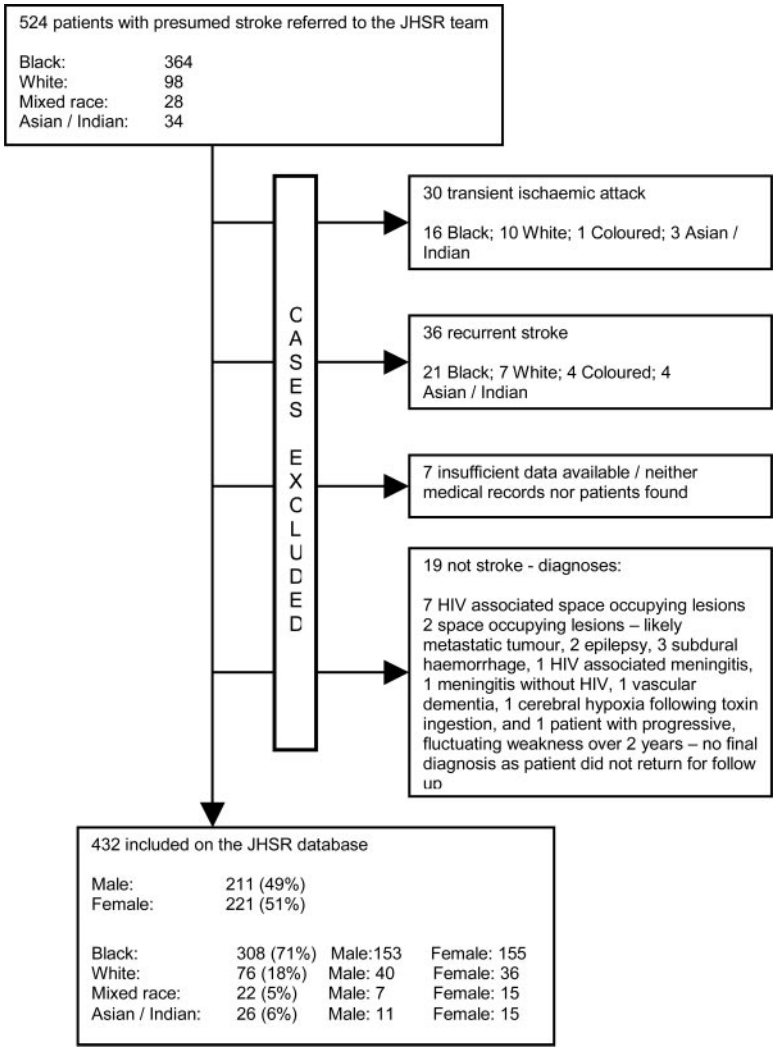

Figure. Study outline: cases included in the Johannesburg Hospital Stroke Register.

difference was not statistically significant (Table 2). Cerebral hemorrhage tends to be more common in younger rather than older stroke patients in community-based stroke studies[20] and we therefore repeated the comparison adjusting for age. This did not alter the significance ($P=0.62$). Although not statistically significant, a higher proportion of black than white stroke patients had cerebral hemorrhages in both younger (≤45 years of age) and older (>45 years of age) age groups. Young black stroke patients had twice as many cerebral hemorrhages as their white counterparts (21 of 96 [21%] young black strokes versus 1 of 11 [9%] young white strokes). Similarly, in older patients, blacks had double the proportion of cerebral hemorrhage that whites had: 34 (31%) of 111 older black stroke patients versus 6 (16%) of 36 older white stroke patients, though admittedly the numbers of strokes particularly in the young stroke age group were small.

We did not find a statistically significant difference in ischemic stroke subtype between the groups (Table 2) using the Oxfordshire Community Stroke Project (OCSP) classification, although blacks had a higher proportion of total anterior circulation and lacunar infarcts than whites, and whites had more partial anterior circulation infarcts than blacks.

However, when we classified ischemic stroke subtype using the TOAST classification, we found significant differences between black and white patients ($P<0.001$; Table 2). We did not find any atherothrombotic strokes in black patients and cardioembolic stroke was twice as common in whites as in blacks. None of the 6 black patients with ischemic stroke we investigated with carotid Doppler or of the 10 who had a cerebral angiogram had any evidence of atherosclerosis (one patient had both investigations). We diagnosed lacunar stroke if the patient had a traditional lacunar syndrome, appropriate CT brain findings, no cardiac source or coagulopathy, and no carotid artery bruits or clinical suspicion of large artery atherosclerosis (ischemic or peripheral heart disease). Similar proportions of black and white patients had "other" causes of stroke, though the nature of these causes differed. In black patients, stroke was thought to have been caused by: dissection in 4 patients, Takayashu's arteritis in 1, intraoperative boundary zone infarction in 2, and fibromuscular dysplasia in 1. The remaining 8 patients were all human immunodeficiency virus (HIV)-positive. Three of these 8 had cerebrospinal fluid features compatible with tuberculous meningitis, and 1 had cryptococcal meningitis. Other causes in white patients included: giant cell

Table 1. Characteristics of Black and White Stroke Patients in the Johannesburg Hospital Stroke Register, by Population Group

	Black		White		Total	
	M	F	M	F	M	F
Strokes, n (ratio M:F)	153 (1.0)	155	40 (1.1)	36	193 (1.0)	191
Age, mean (SD) (95% CI)	51 (15) (48 to 53)	50 (17) (49 to 54)	58 (14) (53 to 62)	66 (15) (61 to 71)	52 (15) (50 to 54)	54 (17) (52 to 57)
Age, mean (SD) (95% CI) both sexes	51 (16) (49 to 52)		61 (15) (58 to 65) $P<0.01$*		53 (16) (52 to 55)	
Age, median (95% CI) (range)	51 (49 to 55) (18 to 85)		63 (56 to 67) (28 to 92) $P<0.01$**		54 (51 to 56) (18 to 86)	
Independent before stroke†/ all with this feature assessed (% of all with available data for population group)	212 / 217 (98%)		57 / 58 (98%) $P=0.8$		269 / 275 (98)	
No. scanned (% of all strokes in population group)	207 (67%)		47 (62%) $P=0.4$		254 (66%)	
No. scanned within 24 hours of admission (% of those scanned)	121 (59%)		29 (62%) $P=0.5$		150 (59)	
No. scanned within 7 days of admission (% of those scanned)	193 (92%)		41 (87%) $P=0.5$		234 (92%)	
NIHSS, mean (SD)	12 (9)		12 (10) $P=0.7$*		12 (10)	
NIHSS, median (95% CI)	10 (8 to 12)		7 (5 to 12) $P=0.4$**		10 (8 to 12)	

M indicates male; F, female; NIHSS, National Institutes of Health Stroke Scale.

*Analysis of variance comparing mean age of population groups and mean time to presentation between population groups; ** Wilcoxon (Mann–Whitney *U*) test for comparison of medians; †Independent before stroke defined as a modified Rankin score of 0 to 2.

arteritis (1), arterial dissection (3), and intracranial venous thrombosis (1).

We could not classify over a third of ischemic stroke patients using the TOAST classification, and the proportion of "undetermined" strokes was much higher in black (43%) than white (17%) stroke patients (Table 2). Apart from 1 young white patient who was extensively investigated and yet no cause found, the remainder of all patients with undetermined causes were classified as such because of incomplete investigation. Although black stroke patients were as likely as white stroke patients to have an ECG on admission (n=227 versus n=60, $P=0.3$) and an echocardiogram (n=42 versus n=15, $P=0.5$), they were less likely to have had a carotid Doppler (n=6 versus n=13, $P<0.001$) than white stroke patients.

We compared the potential cardioembolic sources of ischemic stroke in black and white stroke patients (Table 2). We included patients with strokes classified as cardioembolic using the TOAST classification and patients who did not fulfill the TOAST classification of cardioembolic stroke, but in whom we identified a cardioembolic source together with a large vessel ischemic stroke on CT brain scan and no other cause for their stroke. The potential causes of cardioembolic stroke differed in black and white patients. Atrial fibrillation was much more common in white (28%) than black (1%) patients. This may reflect a true difference, but the younger age of black stroke patients likely accounts for most of the discrepancy. A larger proportion of black patients had dilated cardiomyopathy and valvular (particularly rheumatic) heart disease than white patients (Table 2). Though we found no statistical difference in the proportion of black and white patients with ischemic heart disease (IHD) likely to have caused cerebral embolism, the numbers were very small in both groups.

We attempted to assign the cause of cerebral hemorrhage based on CT appearance, clinical features, and, when available, cerebral angiography. Hypertension caused 45 (82%) of 55 cerebral hemorrhages in black patients, and 6 (86%) of 7 cerebral hemorrhages in white patients. The cerebral hemorrhage in the remaining white patient was associated with anticoagulant use. The most likely causes in the remaining black patients with cerebral hemorrhage were thrombocytopenia (2), anticoagulant use (1), aneurysm (2), and unknown (6). The causes were not significantly different between the 2 population groups ($P=0.5$).

In Table 3 we compare the risk factors for all strokes by population group in the JHSR. Previous transient ischemic attack was uncommon in both groups, possibly the effect of under-reporting. Although ischemic heart disease was less common in black than white patients, we found peripheral vascular disease in similar proportions of patients.

Discussion

The Johannesburg Hospital Stroke Register was limited by scarce resources, a relatively low CT scan rate, and a high proportion of patients whom we were unable to investigate

Table 2. Comparison of the Pathological Stroke Type and Ischemic Stroke Subtypes (Using Both the Oxfordshire Community Stroke Project and Trial of Org 10172 Classifications) in Black and White Stroke Patients in the Johannesburg Hospital Stroke Register (Percentages in Brackets)

	Black	White	Total	
Total with CT brain scan	207	47	254	
Pathological stroke type				
Cerebral haemorrhage	55 (27)	7 (15)	62 (24)	$P=0.2$
Ischemic stroke	141 (68)	36 (77)	177 (70)	
Subarachnoid haemorrhage	11 (5)	4 (9)	15 (6)	
Oxfordshire Community Stroke Project (OCSP) ischaemic stroke subtype				
Total anterior circulation infarction	39 (28)	7 (19)	46 (26)	$P=0.4$
Partial anterior circulation infarction	45 (32)	17 (47)	62 (35)	
Lacunar infarction	40 (28)	8 (22)	48 (27)	
Posterior circulation infarction	17 (12)	4 (11)	21 (12)	
Trial of Org 10172 (TOAST) classification				
Atherothrombotic	0	5 (14)	5 (3)	$P<0.001$
Cardioembolic	21 (15)	11 (31)	32 (18)	
Lacunar	25 (18)	6 (17)	31 (18)	
Other cause	24 (17)	5 (14)	29 (16)	
Undetermined	61 (43)	6 (17)	67 (38)	
Multiple possible cause	10 (7)	3 (8)	13 (7)	
Sources of cardioembolic stroke* (percentage of ischaemic stroke)	25 (18)	14 (39)	39 (22)	
Atrial fibrillation	8 (1)	10 (28)	18 (11)	$P=0.02$
Unknown cause	6	8	14	
With ischemic heart disease	1	1	2	
With dilated cardiomyopathy	0	1	1	
With hypertensive heart disease	1	0	1	
Ischemic heart disease	2 (1)	2 (6)	4 (2)	$P=0.5$
Dilated cardiomyopathy	7 (5)	0	7 (4)	$P=0.03$
Valvular heart disease	8 (6)	1 (3)	9 (5)	$P=0.08$
Rheumatic valvular disease	6	0	6	$P=0.046$
Infective endocarditis	0	1	1	
Other	0	1†	0	

*Included patients with strokes classified as cardioembolic using the TOAST classification and patients that did not fulfill the TOAST classification of cardioembolic stroke, but in whom we identified a cardioembolic source together with a large vessel ischaemic stroke on CT brain scan and no other cause for their stroke.

†This cardioembolic event occurred intraoperatively during aortic valve replacement/aortic aneurysm repair.

fully. Nonetheless, we feel that our case ascertainment was thorough and that we have provided, to the best of our knowledge, the only data comparing black and white Sub-Saharan African stroke patients, albeit in a hospital rather than in a population based sample. Important differences between the 2 populations emerged:

1. Black stroke patients in the JHSR were ≈10 years younger than white stroke patients. This is typical of other studies of stroke in Sub-Saharan Africa.[5,21–23] Although stroke occurs at an earlier age in populations undergoing epidemiological transition,[2] we cannot ignore the possibility of hospital-admission bias influencing the age of black patients in our and other hospital-based studies in Sub-Saharan Africa.
2. Black stroke patients had almost double the proportion of cerebral hemorrhages we found in white patients (Table 2). This too is in keeping with the findings of other studies from Sub-Saharan Africa that used brain imaging.[22–25] The proportion of cerebral hemorrhages in the JHSR white patients was similar to that found in predominantly white high-income populations elsewhere,[26,27] and also similar to that found in African-American stroke patients.[28] A high proportion of cerebral hemorrhage is a feature of populations in early transition,[2] but is also a feature of stroke in younger populations.[20] The younger age of our black stroke patients did not appear to account for the increased proportion of cerebral hemorrhage, however. Although hospital-

Table 3. Risk Factors in the Johannesburg Hospital Stroke Register by Population Group

	Black	White	Total	
Total No. of strokes, n	308	76	384	
Hypertension, n=380	214 (70)	50 (68)	296 (70)	$P=0.3$
Mean total cholesterol level at time of presentation (95% CI), n=154	4.6 (4.3 to 4.9)	5.3 (4.8 to 5.7)	4.8 (4.5 to 5.0)	$P=0.02$
Proportion with total cholesterol≥6 mmol/L, n=154	14 (12)	9 (22)	23 (15)	$P=0.1$
Cigarette smoking, n=308				
Current	57 (23)	34 (54)	91 (30)	$P<0.001$
Ex	28 (11)	15 (24)	43 (14)	
Never	160 (65)	14 (22)	174 (57)	
Alcohol use, n=297				
Current	63 (27)	14 (23)	77 (26)	$P=0.4$
Ex	16 (7)	7 (12)	23 (8)	
Never	158 (67)	39 (65)	197 (66)	
Diabetes mellitus, n=376	42 (14)	11 (15)	53 (14)	$P=0.4$
Previous transient ischemic attack, n=377	4 (1)	4 (5)	8 (2)	$P=0.09$
Ischemic heart disease, n=236	8 (4)	12 (25)	20 (9)	$P<0.001$
Known previous atrial fibrillation, n=377	10 (3)	7 (10)	17 (5)	$P=0.02$
Peripheral vascular disease, n=377	9 (3)	4 (5)	13 (4)	$P=0.5$
Family history of stroke, n=376	27 (9)	11 (15)	38 (10)	$P=0.3$

Percentage of all patients assessed in each population group shown in brackets, unless otherwise stated. n indicates No. assessed. *P: significance of difference.

based stroke studies may bias the inclusion of cerebral hemorrhages which often have a more dramatic presentation,[29,30] this is unlikely to account for the higher proportion of cerebral hemorrhages in our black and white hospital patients because they had similar stroke severity (Table 1).

3. Athough we did not find a significant difference in clinical ischemic stroke subtypes between the 2 population groups, we did find differences in the underlying causes of the ischemic strokes (Table 2). In keeping with other hospital-based studies of South African black stroke patients, extracranial atherosclerosis was uncommon.[22,23,25] We did not find any extracranial carotid artery atherosclerotic disease in our black patients, but very few had carotid Doppler or cerebral angiography. Similarly, far fewer black than white stroke patients had ischemic heart disease, a condition which is for now not at all common in the South African black population (Table 3).[31,32] We diagnosed very few patients in either population group with peripheral vascular disease, possibly because our diagnosis was based on clinical rather than Doppler assessment. Extracranial atherosclerosis is rare in populations in early phases of epidemiological transition,[2,32] but intracranial atherosclerosis has been described in Sub-Saharan Africans[33] and is common in African-Americans.[34] Unfortunately so few of our patients had conventional or MR angiography that we cannot comment on the infrequency of intracranial atherosclerosis.
4. The underlying cardiac disease in ischemic stroke patients with cardioembolic stroke also differed between black and white stroke patients in the JHSR (Table 2). Black patients were more likely to have dilated cardiomyopathies and rheumatic heart disease than white patients, but less likely to have atrial fibrillation. Rheumatic heart disease and dilated cardiomyopathy are both still relatively common diseases in Sub-Saharan Africa,[35,36] and the difference in cause of cardioembolic stroke between black and white patients may reflect the different impact of the epidemiological transition on our 2 population groups. However, the younger age of our black stroke patients may also have added to the low frequency of atrial fibrillation.
5. Although hypertension and diabetes mellitus were equally common in both population groups, in Blacks cholesterol levels were lower (Table 3). As is typical of populations undergoing epidemiological transition, black South Africans without stroke have lower cholesterol than white South Africans.[37] Thus, our findings probably simply reflect the risk profile of the general population. Similarly, cigarette smoking was lower in black than white stroke patients as has been found in the healthy South African population.[38] It is likely that the risk profile seen in black South Africans[39,40] determined the causes of stroke we found, specifically the low frequency of extracranial large vessel atherosclerotic disease.

Hospital-based stroke studies such as this are unlikely to reflect stroke in the community, as not all stroke patients are admitted to hospital. There are several reasons why stroke patients may not be admitted including: early death before hospital, limited resources influencing admission, eg, those with mild stroke being referred to outpatient clinics, lack of

perceived advantage to hospital admission, eg, the elderly and disabled who are already in care, and someone with very mild stroke or their health practitioners may not feel they need to seek medical help.[41] As a result, hospital-based studies are more likely to include severe strokes and cerebral hemorrhages, which have a more dramatic clinical presentation, than mild ischemic strokes.[30] Hospital admission bias may also have resulted in the younger mean age of our stroke patients and high proportion of patients independent before stroke compared to that typically found community-based studies in high-income populations.[26] However, it is less likely to have influenced the differences we found between black and white patients.

The classification of race and ethnicity in medicine often leads to lively debate,[34] and is a particularly sensitive topic in South Africa given the history of apartheid. Whatever one's views, the socio-political impact of the past has created different patterns of vascular disease among South Africa's population based on ethnicity.[8] In the JHSR the nature of stroke in black South Africans was typical of that seen in populations in early epidemiological transition, whereas the nature of stroke in white South Africans was similar to that seen in high-income regions in a later phase of transition.[2,3]

In summary, we found differences in the proportion of risk factors and causes of stroke in urban hospitalized black and white South African stroke patients, although both groups had strokes of similar severity. Black stroke patients were younger, had more cerebral hemorrhages (though this was not statistically significant), had less evidence of large artery extracranial carotid and coronary atherosclerosis, smoked less, and had lower mean cholesterol levels than their white counterparts. Although the clinical OCSP ischemic stroke subtypes did not differ markedly between the 2 groups, the causes of ischemic stroke did differ. This profile of risk factors and relative absence of large artery atherosclerotic disease in black stroke patients may indicate an earlier stage of epidemiological transition and highlights an opportunity to reduce the population risk, in particular to reduce any increase in cigarette smoking and cholesterol levels, to prevent an otherwise inevitable increase in atherosclerotic stroke as the prevalence of stroke risk factors increases in the black South Africa population.

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Disclosures

None.

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