Preventing Stroke
Does Race/Ethnicity Matter?

Philip A. Wolf, MD; William B. Kannel, MD

Stroke, the third leading cause of death in the United States, is also the chief cause of neurological disability in the elderly. Lifetime risk of stroke at age 65 years is estimated to be 1 in 5 in women and 1 in 6 in men in the Framingham Study population. Despite improvements in management of the acute stroke and in rehabilitation poststroke, it is clear that prevention holds the key to decreasing the toll of cerebrovascular disease. In recent years, it has become increasingly clear that clinically inapparent strokes detected on magnetic resonance scan of the brain, so-called “silent” strokes, exert a discernible impact on brain function. Measurable reduction in cognitive performance that leads to vascular cognitive impairment, depressed mood, and impaired gait are consequences of these “silent” strokes. In addition, persons harboring these infarcts and a large burden of white-matter hyperintensities are predisposed to develop clinically apparent cerebral infarctions. Further, the presence of elevated levels of stroke risk factors exerts a measurable effect on brain structure and function, which results in reduced total cerebral volume and an increased presence and volume of white-matter hyperintensities on magnetic resonance scan of the brain, as well as reduced cognitive performance, particularly on tests of executive function.

In this issue of Circulation, Markus et al report on the results of the difficult task of discerning variations in frequency and risk factors that predispose to the different stroke subtypes in black and white patients in South London. Analysis of possible reasons for variation of stroke incidence in different ethnic or racial subgroups of the population requires taking into account place of birth, current geographic location, age, race/ethnicity, family history, personal habits such as cigarette smoking, physiological characteristics and disorders such as blood pressure, cardiac disease, diabetes, and many other risk factors. The finding of more frequent subcortical infarction and intracranial atherothrombosis as the pathology for ischemic stroke in the black patients versus extracranial atherothrombotic occlusion and cardiogenic embolism in whites is in keeping with the current understanding of stroke and vascular topography. A possible explanation for the increased occurrence of lacunar and subcortical infarction in blacks is the greater impact of hypertension, whereas cases of the extracranial atherothrombosis and cardiogenic embolism in whites reflects more atherothrombotic disease. This would not explain the propensity for intracranial atherothrombosis in blacks. It appears that influences associated with race/ethnicity above and beyond the current conventional risk factors for stroke are operating, and it is likely these are mediated through genetic mechanisms.

This multiplicity of influences on stroke incidence makes it extremely difficult to assess the reason for variations in stroke occurrence by race/ethnicity or even whether differences truly exist. Furthermore, there are little data on influence of many of the aforementioned factors on stroke subtype.

It is well known that death rates from stroke are at least twice as great in US blacks as in US whites. Among younger individuals ages 45 to 64 years, the stroke mortality rate for blacks is 3 to 4 times higher than for whites with a decreasing black-to-white mortality ratio with increasing age. However, data on stroke rates in different race/ethnic groups are generally based on crude measures such as death certification. The limited population-based incidence data available confirm stroke incidence rates in blacks are more than double that of whites living in the same geographic region, such as northern Manhattan in New York City or Greater Cincinnati in Ohio/Northern Kentucky.

Stroke is a heterogeneous condition resulting from hemorrhage (intracerebral and subarachnoid) and infarction. Infarction may be secondary to atherosclerothrombosis of the large arteries extra- or intracranial, embolism from the heart or aorta, lipohyalinosis or microatheroma with occlusion of small penetrating arteries resulting in lacunar and subcortical infarction, and a sizable group of ischemic events called “infarcts of undetermined cause.” Survival and functional outcomes vary according to ischemic stroke subtype, with lacunar infarction carrying the lowest case fatality rates and yielding the best functional outcomes, and cardioembolic stroke (largely in the presence of atrial fibrillation) carrying the highest mortality and poorest outcomes.

It has been shown that risk factors differ between hemorrhagic and ischemic stroke and that the relative impact of risk factors operative in all ischemic stroke subtypes vary in their impact among its subtypes. Risk factors for increased stroke mortality in blacks heretofore examined, primarily a higher prevalence of hypertension and diabetes and a lower socioeconomic status, explain only a fraction of this excess. It is possible that elevated blood pressure and insulin resistance/diabetes impact blacks more strongly, which perhaps suggests...
a genetic influence or susceptibility. It is also possible that blacks do not comply with treatment of hypertension and diabetes as well as more affluent segments of society. A more complete understanding of the mechanisms underlying the race/ethnic differences in stroke subtype will likely depend on advances in genomics. These discoveries may point the way to more focused and, therefore, more effective treatment programs for persons with specific subtypes in specific race/ethnic groups.

Primary prevention of stroke is the most effective goal. Considerable progress has been made in identifying persons at increased risk and in applying measures shown to be effective in clinical trials (Table).12 These include blood pressure reduction, particularly in hypertensive diabetics and statins. Other effective measures available include smoking cessation, weight loss, increased physical activity, and warfarin anticoagulation in higher-risk persons with atrial fibrillation.

Greater emphasis needs to be placed on the identification of persons at high stroke risk, which requires identification of those with elevated risk factor levels, particularly in vulnerable populations. This more cost-effectively targets candidates for preventive measures. The goals for risk factor modification and the aggressiveness of therapy should be linked to the level of multivariable risk. Obviously, attention to black populations will yield persons at heightened likelihood of obesity, hypertension, and diabetes, and a group with what appears to be an innate susceptibility to stroke, both hemorrhagic and ischemic. Once identified, preventive efforts (eg, weight loss, cessation of cigarette smoking, control of diabetes, and antihypertensive medication) should be prescribed. Identification, treatment, and control of blood pressure is key and requires medical attention to ensure patient compliance with medication. Statins have been recommended for high-risk persons as well.12

Stroke prevention is best accomplished by control of stroke risk factors, particularly hypertension. This message holds for individuals regardless of race/ethnicity and for prevention of all ischemic stroke subtypes.

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Disclosures

None.

References


Table. Stroke Risk Factors

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<tbody>
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<td>Age</td>
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<td>Gender</td>
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<tr>
<td>Medical Conditions</td>
<td>Behaviors</td>
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<td>Atrial fibrillation</td>
<td>Physical inactivity</td>
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<td>Diabetes-metabolic syndrome</td>
<td>Hormone replacement therapy</td>
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<td>Alchohol abuse</td>
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<td>Carotid stenosis</td>
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<td>Prior TIA or stroke</td>
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<td>† Plasma Hcy</td>
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<td>† CRP</td>
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TIA indicates transient ischemic attack; Hcy, homocysteine; and CRP, C-reactive protein.
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