PROSPECTIVE EPIDEMIOLOGIC STUDY HAS IDENTIFIED HYPERTENSION as the major risk factor for stroke with incidence of stroke rising in direct relation to the level of blood pressure. At any level of blood pressure, however, persons with cardiac disease or impairments, (including Coronary Heart Disease, Congestive Heart Failure, Left Ventricular Hypertrophy by ECG and Atrial Fibrillation even in the absence of valvular heart disease), are at increased risk of stroke including ischemic stroke. The presence of diabetes also increases brain infarction risk. Several potential risk factors for atherosclerotic vascular disease of coronary and femoral arteries seem to exert a less direct impact on the development of brain infarction. These include cigarette smoking which increases brain infarction risk only in men in the younger age group, (mainly below the age of 65), and total serum cholesterol which seems to be inversely related to intracerebral hemorrhage in Hawaiian men of Japanese ancestry and has a quadratic relationship to brain infarction risk only in men in the younger age group, (mainly below the age of 65), and total serum cholesterol which seems to be inversely related to intracerebral hemorrhage in Hawaiian men of Japanese ancestry and has a quadratic relationship to brain infarction in the caucasian Framingham cohort.

The striking decline in stroke mortality confirms the presence of modifiable environmental factors in stroke occurrence. The decline was initially noted in intracerebral hemorrhage by clinicians who identified a decreased frequency of hemorrhage and a 10 year "delay" in onset of this disease, both of which they attributed to more effective treatment of severe hypertension. The decline in incidence of hemorrhage was confirmed in Rochester, Minnesota where a decline in ischemic stroke was also found.

Key to the decline in stroke mortality has been the identification of hypertension as the major risk factor for stroke, whether the stroke mechanism is hemorrhage or infarction, and the demonstration that treatment will reduce stroke and stroke death. In controlled clinical trials treatment of severe and moderately severe diastolic hypertension conducted in the late 1960s, demonstrated the efficacy of blood pressure lowering in stroke prevention. More recently, treatment of mild hypertension (diastolic pressure 90-109 mm Hg) in a placebo-treatment trial, as well as in a treatment versus usual care comparison, the Hypertension Detection and Follow-up Program, has demonstrated the efficacy of treatment of this most prevalent level of elevated blood pressure in stroke prevention. These studies have rather uniformly demonstrated blood pressure control to be followed by a reduction in stroke occurrence and death, as well as a reduction in hypertensive disease notably Congestive Heart Failure. This disease reduction effect is seen in a surprisingly short period of time following blood pressure reduction.

The decline of approximately 5% per year in stroke mortality in the United States over the past decade has not occurred in other nations with substantially equivalent levels of medical care implying that prevention, not more effective treatment of the stroke patient is responsible for the decrease in death from stroke. Data are sparse but at present there is little evidence for reduction in case fatality rates with improvements occurring in medical care as there may well be for prompt and innovative treatment of acute coronary attacks.

The reduction of stroke mortality is real and represents nearly a 50% decline in stroke death in little over a decade! It has occurred in men and women, in blacks and whites and in all areas of the United States. Stroke deaths have declined in the face of declines in total mortality rates and have been a major contributor to the most prominent decline, that of Cardiovascular Diseases; a demonstration that the stroke decline is real and not an artifact of death certification or coding practices.

It has been suggested, on the basis of Rochester, Minnesota data that spontaneous Subarachnoid Hemorrhage, which is almost entirely secondary to bleeding from ruptured berry aneurysm, is the only stroke type that has not declined. However, recent data have shown a decline in death rates for SAH in New Zealand. It is unlikely that this decline is related to improved survival following subarachnoid hemorrhage, which has remained quite constant over the past 30 years, and is probably due to declining incidence of this disease. Studies of women on Oral Contraceptives have shown an increase in fatal subarachnoid hemorrhage from aneurysm in women above age 35.
who smoked in this group, again strongly suggesting environmental factors related to subarachnoid hemorrhage.\(^\text{13}\)

In addition, prospective study of subarachnoid hemorrhage, (with lamentably small numbers of cases), have identified antecedent blood pressure elevation and cigarette smoking as significant risk factors.\(^\text{14}\) That subarachnoid hemorrhage is not an inevitable occurrence might have been suggested by the virtual absence of berry aneurysm at birth and the nearly 5% occurrence at autopsy in late life. In this issue of the Journal data from the prospective Honolulu Heart Study of more than 7000 Japanese men living on Hawaii provide additional evidence to support a relationship between hypertension and cigarette smoking and subarachnoid hemorrhage.\(^\text{15}\) The data linking host and environmental factors to Subarachnoid Hemorrhage are examined in depth by Longstreth et al in a comprehensive review. They conclude that SAH due to aneurysm shares with other stroke types the risk factors of Age and Hypertension but is more common in women and is probably related to oral contraceptive use, cigarette smoking and consumption of alcoholic beverages.\(^\text{16}\)

Examination of the role of other environmental factors may throw additional light on the reasons for the difference in site of vascular pathology in different racial and ethnic groups with Japanese, (and perhaps Blacks), having more intracranial vascular disease and caucasians having more extracranial atherosclerotic disease. The dietary factors examined in the Japanese men living in Hawaii in the Honolulu Heart Study may help to clarify this issue.

Better appreciation of cardiac risk factors in embolic stroke such as nonvalvular or lone Atrial Fibrillation, of other sources of emboli such as mitral valve prolapse or mural thrombi following myocardial infarction should yield benefits in prevention of stroke or stroke recurrence. Use of aspirin in persons with minor or threatened stroke has also been shown to be effective. A host of new agents are available to decrease infarction in an ischemic area. More important these drugs are being studied in a systematic fashion that should permit identification of efficacious agents and determine the dosage that is safe and effective.

This is clearly an exciting period in the epidemiologic study of stroke and atherosclerotic vascular disease. By gaining a better understanding of host and environmental factors contributing to stroke still more effective preventive measures can be promulgated. There is reason to be optimistic that continued attention to and modification of stroke and cardiovascular disease risk factors will continue to yield major dividends in disease prevention.

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