In the early years of epidemiologic investigation of the clinical disease syndromes related to atherosclerosis, the focus was on coronary heart disease as the complication most frequently responsible for precocious disability and mortality. Out of these investigations, serum cholesterol concentration, blood pressure, and cigarette smoking emerged as the strongest and most consistent predictors of coronary heart disease. These predictors comprised a mixture of environmental agents (smoking) and intervening variables that represented genetic-environmental interactions (serum cholesterol and blood pressure) and were found to be associated with the other clinical manifestations of atherosclerosis, namely, stroke and peripheral vascular disease.

By conventional gross, microscopic, and chemical analyses, the atherosclerotic arterial lesions underlying stroke and peripheral vascular disease are indistinguishable from those occurring in the coronary arteries. However, cigarette smoking and diabetes were recognized as the dominant predictors for peripheral vascular disease while serum cholesterol concentration was a relatively weak predictor. Indeed, peripheral vascular disease (including aneurysm of the abdominal aorta) almost never occurs in the absence of smoking or diabetes. We suspect, but are less than certain, that smoking and diabetes would not be associated with peripheral vascular disease if the overall average serum cholesterol concentration were low in the population examined. Somehow, elevated serum cholesterol appears to be a necessary, but not sufficient, condition for the development of peripheral vascular disease.

A different pattern emerged from the epidemiology of stroke, for which blood pressure was recognized as the dominant predictor; serum cholesterol, a significant but less strong predictor; and cigarette smoking, a weak predictor. Similar associations were found for blood pressure and serum cholesterol with atherosclerosis in the cerebral arteries.

The article in this issue of Stroke by Reed and associates from the Honolulu Heart Program confirms these previous observations with regard to the large cerebral arteries. Their article also extends the observations to small cerebral vessels, and they found that atherosclerotic lesions in these vessels are associated only with blood pressure. Lesions in both large and small arteries are closely associated with cerebral infarction, as anticipated. Cigarette smoking is a strong predictor of clinical stroke but not of cerebral atherosclerosis, a relation that parallels that seen with smoking, coronary heart disease, and coronary atherosclerosis.

Not anticipated is the inverse association of a western-type diet, compared with an Oriental diet, and cerebral atherosclerosis in the large arteries. This finding is contrary to the large body of knowledge linking dietary saturated fat and cholesterol with elevated serum cholesterol levels and atherosclerosis. However, it is important to remember that blood pressure is a much more important determinant of cerebral atherosclerosis than is serum cholesterol concentration, and the effect of diet on blood pressure may be a more important intervening variable for cerebral atherosclerosis than the effect of diet on serum cholesterol. Whatever the explanation, this isolated finding is not likely to counteract the large body of evidence regarding dietary fats and coronary heart disease.

Differences in the effects of blood pressure, serum cholesterol, and cigarette smoking on various arterial segments focuses attention on the much-discussed but still poorly understood “tissue factor” in atherogenesis. There are many differences in the anatomy and physiology of the coronary and cerebral arteries, but we do not know which of these differences are responsible for the great variation in their responses to atherogenic intervening variables. The article by Reed et al cautions us that we still have much to learn about what goes on in the arterial wall.

Reference


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Cerebral artery atherosclerosis and diet.
H C McGill, Jr

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