Editorial Comment

A Family History of Risk Factors and Cardiovascular Diseases

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For the most part the significance of coronary risk factors has been established in older adults. This has been accomplished by the longitudinal epidemiologic study of older adults to observe the relation of early measurements of cholesterol, blood pressure, smoking, diabetes, and obesity, as well as other factors, to later development of coronary heart disease, stroke, and renal disease.

In order to explore the importance of coronary risk factors in younger adults, the National Heart, Lung, and Blood Institute established a multicenter study (Coronary Artery Risk Development in Young Adults [CARDIA]) of black and white young adults aged 18–30 years. In this issue of Circulation, the CARDIA study presents the relation of risk factor levels in young adults to a parental history of cardiovascular diseases. In young adults, parental hypertension was associated with higher systolic and diastolic pressures; parental myocardial infarction with higher plasma cholesterol, higher blood pressure and lower high density lipoprotein (HDL) cholesterol levels; and parental diabetes with higher fasting blood sugar levels in both races and higher triglyceride and lower HDL cholesterol in only black participants. A parental history of obesity was related to less favorable lipoprotein levels in white young adults. A history of stroke in parents was associated with higher blood pressure in black but not in white young adults.

The strategy of relating coronary risk factor levels to parental disease has also been applied in children. For example, in two studies of cholesterol levels in school-age children, Schrott et al1 and Moll et al2 have shown a greater frequency of deaths from cardiovascular diseases of the adult relatives of children with cholesterol levels exceeding the 95th percentile than in subjects with lower levels. Burns et al3 have shown that persistently obese children with high blood pressure have significantly more relatives dying of cardiovascular causes than children who are lean or who are obese with lower blood pressure levels. This strategy of examining morbidity and mortality in the relatives of younger subjects adds a level of biologic significance to coronary risk factor measurement in the young. These observations suggest that there is a familial propensity to the development of coronary heart disease that begins at an early age. This familial propensity could be the result of genetic factors, nutritional practices, life style characteristics, or combinations thereof. Indeed, the mechanisms responsible may be various and family specific.

The utility of a family history is that it identifies young individuals who are at an increased risk both from abnormal coronary risk levels at a young age and from a familial propensity for the development of cardiovascular diseases later in life. This strategy allows us to target our clinical practice so that we may identify such individuals and provide them the appropriate examinations and preventive advice about nutrition, exercise, the need to stop smoking, and in selected cases with high blood pressure or cholesterol levels, drug therapy.

This approach of using a family history to add to the significance of low density lipoprotein (LDL) cholesterol levels in adults over the age of 20 years was suggested by the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults of the National Cholesterol Education Program (NCEP).4 They have suggested that adults without coronary heart disease and with LDL cholesterol levels between 130 and 159 mg/dl be given individual counsel if they have two additional risk factors, one of which is a family history of coronary heart disease under the age of 55 years, and the others include male gender, HDL cholesterol ≤35 mg/dl, diabetes, smoking, hypertension, obesity, and cerebral or peripheral vascular disease.

A number of authors have suggested universal screening of cholesterol levels in children to identify those with high levels so that individual medical care can be instituted. Some have indicated that screening cholesterol only in those with a family history of cardiovascular diseases will miss many with elevated cholesterol levels.5–7 In Muscatine, Iowa, where a cohort of children, aged 8–18 years, was followed until they were 20–30 years without individual intervention, 75% of girls and 56% of boys whose chole-
terol levels exceeded the 75th percentile on two successive readings as children would not qualify for individual intervention as adults according to NCEP criteria. Additionally, 57% of girls and 30% of boys whose cholesterol levels exceeded the 90th percentile on two examinations would not qualify as adults for intervention. Thus, universal screening of school-age subjects could result in many young people being given individual care that may not be necessary. However, a strategy of selective screening of children may be more rewarding. In a study of the progeny of young ischemic heart disease subjects with positive coronary angiograms, Lee et al found that 50% of the offspring ranging in age from 3–30 years had some form of dyslipidemia. It is estimated that about 20% of all hospital discharges for ischemic heart disease are under the age of 55 years. Therefore, large numbers of young people at risk will be identified if practitioners who care for young ischemic heart disease patients could take the time to examine the offspring of these patients.

This study from CARDIA indicates that a family history in young adults adds significance to coronary risk factor levels in young adults. Because of the various mechanisms that may operate in each family, it is now incumbent upon us to not only define their frequency and public health importance, but also to develop targeted family-specific interventions.

It is likely that many with higher risk factor levels as young adults will be missed if only a family history is used for their identification. However, the significance of these risk factors without a family history of disease is somewhat uncertain in those with mild abnormalities. Thus, we must await the data from CARDIA that will tell us how well coronary risk factor levels in the age group 18–30 years will predict levels at a later age when the predictive value of risk factor levels for cardiovascular diseases and the benefit of therapy have been clearly established. In the meantime, young subjects with a family history of cardiovascular diseases who also have elevated risk factor levels appear to be those for whom we can most clearly recommend individual physician care.

References

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