A subcommittee of the Inter-Society Commission for Heart Disease Resources recently recommended an immediate, nationwide change in dietary habits to reduce the risk of coronary heart disease in later life. Specifically, the Commission urges that people eat less than 300 mg of cholesterol each day, that the total calories from fat be less than 35% of the diet, and that the fat calories essentially be divided equally among saturated, monounsaturated, and polyunsaturated sources. (A commentary on these recommendations appears in this issue of Pediatrics.)

The Committee on Nutrition, realizing that pediatricians will increasingly be asked about diets for children to reduce the risk of heart disease in later life, has evaluated the Commission's report for its application to pediatric practice. The Committee stresses that such dietary intervention at present, experimental and recommends against dietary changes for all children. Dietary intervention may be warranted in special circumstances, but not before 1 year of age. Reasons for these recommendations will be given in this report.

The evidence relating dietary cholesterol to coronary heart disease is summarized as follows:

1. Some inborn or acquired diseases with hypercholesterolemia are associated with premature atherosclerosis.
2. Serum cholesterol levels are higher than usual in persons with coronary heart disease.
3. Persons with high cholesterol levels in prospective studies developed coronary heart disease more often than those with normal levels.
4. The mortality rate from coronary heart disease in different countries varies in relation to the average blood cholesterol values (or dietary fat intake).
5. Experimentally induced hypercholesterolemia in animals is associated with atherosclerotic deposits.
6. Atherosclerotic plaques contain lipids similar in composition to those in the blood.

The foregoing evidence would be more persuasive if restriction of dietary cholesterol in the population at large could be shown to reduce the frequency of coronary heart disease. To date, the results of studies on this subject are far from convincing. Since a large-scale study designed to resolve this issue for the general population in the United States would take at least 10 years to complete, the Commission has urged an immediate, nationwide campaign to reduce dietary cholesterol—with concurrent evaluation of the effects on the incidence of coronary heart disease. In addition, the Commission has stressed the need to control obesity, diabetes mellitus, and two other major risk factors—hypertension and cigarette smoking.

However, the foregoing are not the only determinants of coronary heart disease. Recent progress has been made in defining the role of genetics in heart disease. People with familial type II hyperlipoproteinemia are particularly prone to coronary heart disease. This trait, apparently transmitted as an autosomal dominant, has been provisionally estimated to be present in as many as 1 in 200 newborn infants. It has also been estimated that, among male heterozygotes with this trait, the chance of developing ischemic heart disease is 5% by age 30, 51% by age 50, and 85% by age 60. This is a tremendous morbidity rate from a single disease. The frequencies for women with this trait are less, although much greater than the normal rate. Disability among presumed homozygotes with type II defect is greater and occurs earlier than among heterozygotes. Homozygotes may have xanthomas and vascular disease before 10 years of age. The other forms of familial hyperlipoproteinemia (types I, III, IV, and V) are rare in children.

Unproven preventive measures should be tested first in a group at exceptionally high risk of the disease concerned. Thus, polio
virus vaccine was initially administered to children in the first three grades of primary school because they were known to have the highest attack rates. Similarly, dietary intervention should, before being recommended for everyone, be tested in persons with an exceptionally high risk of having coronary heart disease—namely, those with familial type II hyperlipoproteinemia.6 Identifying these individuals early in life is feasible, and motivation for dietary restrictions is likely to be greater in affected families than in the general population. The benefits, or possible adverse effects, of dietary changes can be evaluated from a study of this special group before changes are recommended for the population at large. A nationwide alteration in diet may well impair the sense of well-being of the general public, apart from any unforeseen, organic harm.

The modified diet for persons with familial type II defect may not provide a convincing answer to the benefits to be derived by the population at large; however, it will provide some experience as to safety, acceptability, and effectiveness of the general approach. Until the results of such trials are available, the Committee does not support the Commission’s recommendation for dietary intervention for all children. However, pediatricians should identify children with familial type II defect and recommend the appropriate diet for them.

A family history of coronary heart disease before 50 years of age in first cousins or closer relatives justifies obtaining a fasting serum specimen from the child for cholesterol, triglyceride, and lipoprotein analysis. Such determinations cannot as yet be recommended before 1 year of age because normal values during infancy are only now in the process of being established. Other family members should be studied when a child is found to have abnormal values.

When a child with type II hyperlipoproteinemia is found, at least three blood lipid determinations should be made 1 week apart to establish a baseline before beginning dietary management. The dietary cholesterol for children from 1 to 12 years of age with type II hyperlipoproteinemia should be kept as low as possible. A handbook concerning the disease and the diet can be obtained from the National Heart and Lung Institute or the American Heart Association. The diet for type II hyperlipoproteinemia is considered to be therapeutic and is not designed for the general population or for patients with other forms of hyperlipoproteinemia.

A sustained reduction of at least 10% in serum cholesterol or triglyceride levels signifies that a dietary effect has been achieved, and the diet should be continued. At present, there is no good reason for initiating the diet before 1 year of age, especially since the benefits from present infant feedings are too great to be jeopardized. When the diet is begun, care must be taken, of course, to insure that food constituents essential to normal growth are provided in adequate quantities. Drugs to control type II hyperlipoproteinemia in children should only be used if the condition is severe and does not respond to dietary therapy alone.

Families with no evidence of type II hyperlipoproteinemia who request guidance about diets to reduce coronary heart disease can be advised that no harm is known to occur from moderate dietary alteration and, conceivably, that some benefits may accrue. In particular, this may apply to children with diabetes mellitus or a strong family history of diabetes, as well as to children in families having a history of early coronary heart disease not related to type II defect; in these instances, no therapeutic regimen would be prescribed. For these children, the diet proposed by the Inter-Society Commission for Heart Disease Resources would be appropriate: adjust caloric intake to maintain optimal weight and reduce dietary cholesterol (for children) to 150 mg/day; less than 35% of the calories should come from fats, which essentially should be divided equally among saturated, monounsaturated, and polyunsaturated sources.
REFERENCES

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