Family-Directed Preventive Cardiology

Dennis M. Davidson, MD, and Edward J. Doyle, Jr, MD Irvine and Long Beach, California

Cardiovascular events continue as the leading cause of death in the United States, despite a decline in recent years. Epidemiological evidence has confirmed that in adults smoking, high blood pressure, hypercholesterolemia, a family history of atherosclerotic disease at an early age, diabetes mellitus, and certain behavioral patterns are associated with a higher risk for myocardial infarction and other manifestations of coronary artery occlusion. In addition, a lack of regular physical activity, being overweight, and excess ethanol consumption are associated with increased cardiac disease rates. Since these indicators of risk may appear in childhood, and the earliest manifestations of atherosclerosis or hypertension can become manifest soon thereafter, the prevention of cardiovascular events must begin early.

This paper reviews the evidence for the origins of cardiovascular risk in childhood, and offers recommendations to family physicians to help them provide parents with the proper information, motivation, and skills to teach their children healthy nutritional, exercise, and behavioral patterns.

Investigators who suggest that the atherosclerotic process begins in childhood usually cite autopsy data from American servicemen killed in the Korean and Vietnam wars. In both studies the mean age of subjects was 22 years. Using gross examination of the coronary arteries, Enos et all found that 77.3 percent of Korean War casualties had some degree of atherosclerosis, while 15.3 percent had over 50 percent narrowing of luminal diameter of at least one coronary artery. McNamara

et al² studied the coronary arteries in 105 men killed during combat in Vietnam. Four men had severe disease in all three major coronary arteries. Twenty-seven men had some involvement in two vessels, with another 20 showing atherosclerotic change in only one vessel. Postmortem coronary angiography showed only one lesion that represented 50 percent or greater luminal occlusion. Differences in the two studies may be partially attributed to methods, but the presence of any atherosclerosis in these two young populations gives impetus to investigation into the earliest stages of this common disorder.

Studies in children and adolescents have found characteristics that appear related to subsequent development of coronary artery disease, including blood pressure, obesity, nutritional choices and in-

From the Preventive Cardiology Program, Department of Medicine, University of California, Irvine, and the Department of Family Medicine, Naval Regional Medical Center, Long Beach, California. Requests for reprints should be addressed to Edward J. Doyle Jr, LCDR, MC, USN, Department of Family and Preventive Medicine, University of Southern California, 2025 Zonal Ave, Los Angeles, CA 90033.

take, serum lipids, smoking, alcohol abuse, initiation of oral contraceptive use, and certain behavioral patterns. Many of these risk indicators undergo temporal changes from infancy through early adulthood, with different patterns noted in the female and male populations. Attention to these factors was drawn by the Surgeon General in his report "Healthy People." The importance of these factors, however, may not yet be fully appreciated by the medical community. In a recent sampling of primary care physicians in Massachusetts, only blood pressure and smoking were considered to be "very important" factors in health promotion.⁴

Fortunately, techniques do exist that permit the identification and modification of cardiovascular risk at early ages.⁵ These methods will be reviewed in this paper.

Risk Factors

Blood Pressure

As part of the Lipid Research Clinics (LRC) study, investigators in Cincinnati acquired crosssectional data in black and white children aged between 6 and 19 years. During the decade from late childhood (6 to 9 years) to late adolescence (15 to 19 years), mean systolic blood pressure rose from 94.8 mmHg to 101.7 mmHg in girls and from 92.2 mmHg to 111.7 mmHg in boys. Mean diastolic blood pressure increased from 54.1 mmHg to 68.3 mmHg in girls and from 54.0 mmHg to 69.1 mmHg in boys. The investigators found no significant differences between black and white children, but surveys of adults in the same community revealed the usual pattern of higher blood pressure in blacks.6 In a study of black children in Dayton, Ohio, inner-city children had considerably higher blood pressures than their counterparts living in the suburbs.7 The Bogalusa, La, longitudinal study demonstrated that blood pressure rankings within a group of children persist for several years, given reproducible measurements, including multiple blood pressure determinations at baseline and on subsequent visits.8

A link from late adolescence to adulthood was provided in a longitudinal study of men from college days to middle age by Paffenbarger et al.⁹ Approximately one half of the men who were diagnosed in adulthood as hypertensive had systolic

blood pressures of 130 mmHg or greater in college. In contrast, only one fourth of those without subsequent evidence of high blood pressure had such systolic readings while in college.

Body Weight Obesity

Although underwater weighing is the most accurate indicator of body density, 10 it is neither readily available for most clinical practices nor feasible for large population studies. "Ideal" weights derived from insurance company statistics are subject to both controversy and change. Therefore, mathematical expressions of height and weight have been widely used for clinical and epidemiologic studies. Of the possible combinations of height vs weight relationships, the ratio of weight to the square of height, named the Quetelet index, has been in frequent use in clinical and epidemiological studies.

From late childhood to the final stages of adolescence, the Quetelet index increases by 44 percent in girls and boys, both black and white. ¹¹ During this time of sexual maturation, dietary consumption of calories, cholesterol, total and saturated fat, protein, and sucrose is increased, with greater changes in boys. ¹² By early adulthood, black children are taller and heavier than whites, but have less subcutaneous fat.

Obesity predisposes to diabetes, high blood pressure, and atherosclerosis, but much remains to be learned about the family, social, and cultural antecedents to its development.

Nutrition and Lipids

Studies in the United States and other countries indicate that population blood lipid levels in children are directly related to morbidity and mortality from coronary artery disease in adults of the same geographic area. Further, when examining socioeconomic, cultural, ethnic, and gender strata within communities, the relationship persists. 16-21

How early in life do differences arise? Using umbilical cord blood, investigators found black and white children have similar blood cholesterol levels at birth. ¹⁶ Differences among certain ethnic and racial groups, however, appear as early as one year after birth. Some of this divergence may be nutritional, since infants given table foods early had higher levels of blood cholesterol. ¹⁷ Studies comparing five-year-old children in Wisconsin and Mexico showed that the American children had

significantly higher levels of total cholesterol.¹⁸ In one Native American culture (Pima Indian), children have substantially lower blood cholesterol levels than other American children.¹⁹ White American children have higher serum triglyceride values than black children from the same area,²⁰⁻²² and a positive relationship has been noted between serum glucose and serum triglyceride levels.¹¹

The differences noted in blood levels may be partly explained by body composition and by nutritional choices. As the Quetelet index increases through adolescence, it is positively associated in each age stratum with serum triglycerides, low-density lipoprotein cholesterol fraction, and both systolic and diastolic blood pressures, while it is inversely correlated with the high-density lipoprotein cholesterol fraction. ^{6,11,23}

Because total serum cholesterol can increase secondary to elevations of any of its fractions, and because the fractions may move in opposite directions with interventions, it is important to fractionate total blood cholesterol, particularly when investigating high levels of this parameter.²⁴

The Cincinnati group compared lipid values in children at the five levels of the Tanner scale, ^{25,26} finding that cholesterol levels tend to fall, while triglyceride levels rise, during the period of sexual maturation.²⁷ Both boys and girls maintain their lipid level rank among their peers during these years, leading to the hypothesis that those in the upper deciles of lipid values during adolescence will become the adults with the highest risk of coronary artery disease development.

Black children have higher high-density lipoprotein levels than white children with the same total cholesterol, 24,28,29 despite the absence of a difference in cord blood levels at birth. 30 The black-white high-density lipoprotein differences become more pronounced in adulthood, especially for black men. 31

At particular risk for later coronary artery disease are children of persons who have experienced myocardial infarction before the age of 50 years. Such children have total cholesterol values up to 20 mg/dL higher than children without such a family history.³² Another study examined first-degree relatives of children who were identified at screening as being in the upper 5 percent of cholesterol distribution. Coronary artery disease deaths occurred more frequently in relatives of children with high cholesterol levels than in families of

children with lower cholesterol levels. The same investigative group showed considerable familial clustering of high total cholesterol values.³³

Infants fed a low-cholesterol and low-saturatedfat diet have lower serum cholesterol levels at 6 months and 3 years of age.³⁴ Adolescents eating a diet low in cholesterol and saturated fats experience a 10 to 15 percent reduction in total cholesterol and low-density lipoprotein levels compared with teenagers with a more typical American diet.

Other desirable features in a heart-healthy diet include meals high in complex carbohydrates (whole grains, legumes, etc), high in fiber, and low in salt. Using such a diet for 24 days, persons of all ages have displayed a 20 percent reduction in total and low-density lipoprotein cholesterol as well as triglycerides.³⁵ However, if simple sugars rather than complex carbohydrates are used to increase the carbohydrate percentage in the diet, serum triglycerides may rise dramatically.³⁶

Smoking

The incidence of coronary artery disease is related directly to the number of cigarettes smoked daily. Fortunately, cessation of smoking dramatically lowers coronary disease risk.³⁷ In examining this relationship of smoking and heart disease, cigarette smokers have been noted to have lower high-density lipoprotein levels than nonsmokers, the magnitude of which is similar to those seen between adults with and without coronary artery disease.³⁸ The high-density lipoprotein differences appear early; in the Cincinnati study of children aged 12 to 19 years, marked differences between smokers and nonsmokers were present in both girls and boys.³⁹

Additional contributions to cardiovascular risk may be attributed to the effects of nicotine on heart rate, blood pressure, free fatty acids, platelet aggregation, and vasoconstriction. Use of filter cigarettes does not reduce the relationship of these smoking effects on cardiovascular disease incidence rates.⁴⁰

Living in a family where one or more members smoke also places the child at risk to undertake this habit. The risks are more than doubled again if the child's closest friend also smokes. Thus, not only must peer pressure to smoke be lessened, but the family must be committed to cessation efforts if they expect their children to avoid smoking.⁴¹

Teenage women have been the last to display a

downward trend in smoking.⁴¹ Because such women in lower socioeconomic groups are also at highest risk for pregnancy, their smoking habits take on additional importance. While a majority of young women smokers believe that maternal smoking may harm an unborn child, only approximately one in three stops completely during pregnancy. Further, most of these resume their normal pattern once the child is born. This presents a challenge to the primary practitioner who provides prenatal care as well as follow-up after parturition.

Oral Contraceptives

Concurrent use of oral contraceptives and cigarettes raises more than 20-fold the risk of myocardial infarction. ⁴² In addition to the adverse effects of smoking on cardiovascular risk indicators, use of estrogen-progestogen combination oral contraceptives increases total and high-density lipoprotein cholesterol levels. High-density lipoprotein levels are correlated directly with estrogen dose and inversely with progestogen doses. Triglycerides are higher in oral contraceptive users of all ages. The Cincinnati study compared lipid levels in 31 adolescent users of oral contraceptives and 31 matched control women, and found higher low-density lipoproteins, high-density lipoproteins, and triglyceride levels in users. ⁴³

Behavioral Patterns

Coronary-Prone Behavior

Certain behavior patterns in both men and women are associated with a higher risk for developing coronary artery disease and subsequent mortality.⁴⁴ This cluster of behaviors includes hostility, impatience, responding to a chronic sense of time urgency, aggressiveness, and extreme competitiveness, including achievement orientation in leisure activities.⁴⁵ This behavior pattern (type A) can be assessed by interview or questionnaire. Fortunately, this type of behavior can be modified, as recently demonstrated by the San Francisco group who originally identified its atherogenic effects.⁴⁶

It appears that this behavior pattern does not have a significant genetic component, but the behavior of parents can influence development of this behavior pattern in their children, particularly those of the same sex, when there are constant

escalations of performance standards and demands by parents.47 Waldron and her colleagues48 found that students in rural, working-class schools feel less time urgency, but are not less competitive or achievement oriented than suburban students. The authors speculated that the working-class community students may strive for their achievement in better defined, more readily attainable roles than do suburban students. The same team surveyed undergraduate students, finding that those with the type A pattern studied longer, attended classes more frequently, and achieved better grades than others, but were not more successful socially. Both male and female students displaying this pattern reported that their parent of the same sex often punished them physically, which, the authors speculate, may have led to the anger and aggression displayed by these students. As academic pressure increased throughout the semester, manifestations of the type A behavior increased.

Reactive Stress

In addition to those persons displaying coronary-prone behavior patterns, others are susceptible to the cardiovascular effects of reactivity to psychosocial stressors in daily life. These reactions include secretion of aldosterone, antidiuretic hormone, catecholamines, cortisol, growth hormone, and thyroid-stimulating and thyroid hormones as well as increases in systolic and diastolic blood pressures, heart rate, serum cholesterol, free fatty acids, plasma glucose, platelet adhesiveness, peripheral vasoconstriction, and blood viscosity. All of these factors have been associated with increased rates of development of atherosclerosis and hypertension.⁴⁹

Buell and Eliot⁴⁹ have recently reviewed the psychophysiological links between emotional stress and hypertension. The studies reviewed animal experiments that result in fixed hypertension even after conflict stimuli have been removed, epidemiologic studies of increased hypertension prevalence with the advent of urbanization, and stress-induced endocrine responses in persons with fixed hypertension.

Physical Fitness

Several studies relating levels of physical activity at work and leisure have found associations with subsequent development of atherosclerotic

disease. Physical activity levels in most of these investigations, however, have been measured by self-report or by inference from job descriptors. Recently investigators analyzed the relationship of physical work capacity (as measured by bicycle ergometry) to subsequent symptomatic myocardial infarction in 2,779 healthy men aged under 55 years who were evaluated at the time of employment for fire and police departments. During a mean follow-up period of five years, the relative risk of myocardial infarction for those with physical work capacity below the median was 2.2 times that of persons scoring above the median. When further analyzed, the excess risk was concentrated in those men who either smoked or had abovemedian values of serum cholesterol or systolic blood pressure. In men with two or three of these "risk factors," relative risk was 6.6.50

Alcohol Consumption

When daily absolute alcohol consumption exceeds 50 g (eg, 150 mL of 80 proof spirits, 1.5 L of beer, or 450 mL of wine), cardiac dysrhythmias, cardiomyopathies, stroke, and hypertension may occur.⁵¹ Such changes can be detected even in young persons.⁵² Studies firmly linking more moderate alcohol use as an independent risk indicator are handicapped by several factors, including difficulties in accurate quantification of consumption if only self-report is used. A second confounding factor is the putative effect of mild to moderate alcohol consumption on high-density lipoprotein concentrations, although the epidemiologic importance of this association has been recently questioned.⁵³

While the overall incidence of alcoholism is approximately 5 percent, there is significant familial clustering of alcohol abuse. For example, having one first-degree relative who is alcoholic increases one's risk for alcoholism to 20 percent. This risk can exceed 50 percent in the case of identical twins.⁵⁴

Pregnancy

In recent years, the public has become more aware of potentially toxic effects on the fetus of nutrition, smoking, alcohol, and drug ingestion. ^{55,56} Because of the close patient-physician relationship, pregnancy presents a special opportunity to modify health habits not only in the woman, but in other family members as well.

Recommendations

The preceding review of cardiovascular risk indicators suggests that the family physician is in a unique position, as physician to both parents and children, to facilitate acquisition at an early age and maintenance of health behaviors that can reduce risk for cardiovascular disease. The following recommendations are offered to help all family members adopt nutritional, exercise, and behavioral patterns that can become lifelong habits.

General

The use of quantitative health risk appraisal has increased in recent years, particularly in industrial and office settings.⁵⁷ The Centers for Disease Control have developed a computer program that requires only blood pressure and serum cholesterol input from the physician, in addition to information obtained from questionnaire responses of the patient, to compute risks of various chronic diseases.⁵⁸ In addition to computation of present risk, this program indicates to the patient estimates of risk change given alterations in health habits, such as smoking cessation, lowered cholesterol intake, weight reduction, and increased physical activity.

More important, these risk indicators, once identified, can be altered. In North Karelia, Finland, a school-based and community-based intervention program successfully reduced serum total cholesterol and delayed smoking onset in 13-to 15-year-old children.⁵⁹

Nutrition

Because nutritional intake has such a marked effect on other risk indicators (blood pressure, serum lipids and lipoproteins, and obesity), a nutritional plan compatible with goals established by the Senate Committee on Nutrition and Human Needs⁶⁰ and the American Heart Association⁶¹ is recommended for all families. This plan has the following calorie composition: 50 to 60 percent complex, nonsucrose carbohydrates (such as vegetables, whole grains, and fruits), a maximum of 10 percent saturated fat, 10 to 15 percent monounsaturated and polyunsaturated fats, and the balance of calories from protein. Cholesterol should be limited to 300 mg or less daily. Sodium intake should not exceed 4 to 5 g daily. Foods high in fiber can be encouraged. Sucrose and other refined sugars, because of their high caloric content,

absence of other nutritional value, and relative expense, should account for no more than 10 percent of total caloric intake. Fructose, in its natural form, is acceptable.

When family history or other findings suggest that some members might be at high risk for subsequent development of cardiovascular disease, screening can be undertaken. The Nutrition Committee of the Canadian Pediatric Society recommends that total serum cholesterol determinations be made in children who (1) have a family member with myocardial infarction before the age of 50 years, (2) have parents with elevated serum lipid concentrations, or (3) are hypertensive or obese. They recommend high-density lipoprotein determinations be determined in those with elevated total cholesterol readings on two separate serum samples. 62

For children found to be at high risk, special diets are in order. For those not so identified, it is suggested that only breast or formula milk be used for the first four to six months. During the next 18 months, sugar or salt should not be added to solid foods. After two years, whole grain products can be initiated, intake of fruits and vegetables can be increased, and salt and refined sugar consumption may be allowed. Dietary fat should be limited. As with all nutritional plans, physical activity and nutrient energy intake should be adjusted to achieve and maintain ideal body weight.

Food should not be used as an emotional reward, a practice endemic in Western culture. Instead, children can be praised for leaving food on their plate when they feel satiated.

Obese children without obese parents are rare. 63 When found, such children will usually respond to a program of nutritional education, discussion of methods to increase energy expenditure, and maintenance of a food diary. Children should be accompanied by at least one parent at the initial evaluation and during return visits, which are scheduled at two- to four-week intervals. Parental participation provides essential daily supervision and allows positive reinforcement of the child's successful food selections and diary maintenance.

When one or both parents are obese, the likelihood of the child's maintaining a weight reduction is slight, unless the parents actively try to control their own obesity. When the family is receptive to treatment, a behavioral modification program can

be used that focuses on family dynamics as well as attitudes toward food and eating.¹⁵

Exercise

Parental modeling of the pleasurable benefits of regular aerobic exercise will greatly enhance the possibility that children will adopt similar habits. Coupled with a sound nutritional plan, regular exercise will consistently reduce blood pressure, serum lipids, and weight in persons of all ages, and may promote emotional health as well.

Exercise is usually prescribed according to frequency, intensity, and duration. For cardiovascular conditioning, one should exercise at least three times weekly, at heart rates from 60 to 80 percent of maximal heart rate, for a period of 30 minutes or more on each occasion. Activities should be chosen that are sufficiently pleasurable to sustain long-term interest and adherence. Several activities meeting these criteria can be alternated to allow development of different muscle groups.⁶⁴

Parents should be careful not to inject excess competition into family athletic endeavors, but simply to recognize and accept existing skills and potential limitations in their children.

Smoking

Efforts to prevent or postpone the onset of smoking in children⁶⁵ appear more promising than smoking cessation programs. Education and fear arousal regarding long-term consequences of smoking do not deter adolescent cigarette use. More effective are youth programs that use peer counselors or children one or two years older who are well respected by the target population, particularly when the focus is placed on the immediate sequelae of smoking. Advertising by government and voluntary health agencies that employ teenage role models may further contribute to a decrease in the alarming rates of smoking children. Finally, role modeling by significant adults can diminish cigarette use during adolescence.

Young women who persist in smoking should be offered alternatives to oral contraception.

Blood Pressure

Although marked elevation of blood pressure in children may require pharmacologic therapy, it is suggested that a nutritional and exercise program aimed at weight reduction is the first line of therapy for children in the upper ranges of blood pres-

sure distribution. Weight loss will effectively reduce blood pressure,66 although children are as susceptible as adults to recidivism in weight maintenance. Since clustering of blood pressure is often noted within families,67 adoption of nutritional and exercise habits by the entire family will facilitate treatment of those members with elevated blood pressure and may reduce the risk of other persons in the family from acquiring this disorder.

Alcohol Consumption

In families with one or more persons dependent on alcohol, the physician must provide education and counseling for children and adolescents regarding their risk for alcoholism. As with other cardiovascular risk indicators, the chances of prevention increase with more complete family involvement in treatment.68

Pregnancy

Prenatal care should include counseling regarding the effects of proper nutrition, weight maintenance, smoking, blood pressure, alcohol, and drug use on fetal health. Maternal concern regarding these subjects often stimulates improvement of health habits for the entire family.⁶⁹

Family Dynamics

The emotional health of family members can be enhanced through open, honest communication and regard for each other as unique individuals worthy of respect and unconditional love. Such attitudes should help parents maintain realistic expectations for achievement, not only for children but also for themselves. Children can acquire selfrespect and carry ongoing social support into their adult years. Given such a beginning, it is to be hoped they will avoid the perception that they can gain self-esteem only through incessant striving against the world.

Summary

Adoption of healthy habits by children will arise from increased knowledge, attitude change, and skill acquisition, but must include much practice with positive reinforcement. Working with parents, family physicians can make an unparalleled contribution to the future well-being of their young charges by helping the family achieve these goals.

References

1. Enos WF, Beyer JC, Holmes RH: Pathogenesis of coronary disease in American soldiers killed in Korea. JAMA 148:912, 1955

2. McNamara JJ, Molot MA, Stremple JF, et al: Coronary artery disease in combat casualties in Vietnam, JAMA

216:1185, 1971

3. Healthy People. The Surgeon General's Report on Health Promotion and Disease Prevention. Institute of Medicine (Rockville, Md). DHEW publication No. (PHS) 79-55071. Government Printing Office, 1979

4. Wechsler H, Levine S, Idelson R: The physician's role in health promotion—A survey of primary-care practitioners. N Engl J Med 308:97, 1983
5. Stamler J: Improved life style: The potential for the

primary prevention of atherosclerosis and hypertension in childhood. In Lauer RM, Shekelle RB (eds): Childhood Prevention of Atherosclerosis and Hypertension. New York, Raven Press, 1980, pp 3-36

6. Morrison JA, Khoury P, Kelly K, et al: Studies of blood pressure in schoolchildren (ages 6-19) and their parents in an integrated suburban school district. Am J Epidemiol 111:156, 1980

7. Burns MD, Morrison JA, Khoury PR, et al: Blood pressure studies in black and white inner-city and suburban

adolescents. Prev Med 9:41, 1980

8. Voors AW, Radhakrishnamurthy B, Srinivasan SR, et al: Plasma glucose level related to blood pressure in 272 children, ages 7-15 years, sampled from a total biracial population. Am J Epidemiol 113:347, 1981

9. Paffenbarger RS Jr, Thorne MC, Wing AL: Chronic disease in former college students: VII. Characteristics in youth predisposing to hypertension in later years. Am J

Epidemiol 88:25, 1968

10. Katch I, Michael ED, Horvath SM: Estimation of body volume by underwater weighing: Description of a simple method. J Appl Physiol 23:811, 1967

11. Laskarzewski P, Morrison JA, Mellies MJ, et al: Re-

lationships of measurements of body mass to plasma lipoproteins in schoolchildren and adults. Am J Epidemiol 111:

395, 1980

12. Morrison JA, Larsen R, Glatfelter L, et al: Nutrient intake: Relationships with lipids and lipoproteins in 6–19-year-old children—The Princeton School District Study. Metabolism 29:133, 1980

Stamler R, Stamler J, Riedlinger WF, et al: Weight and blood pressure. Findings in hypertension screening of 1 million Americans. JAMA 240:1607, 1978

14. Hubert HB, Feinleib M, McNamara PM, et al: Obesity as an independent risk factor for cardiovascular disease: A 26-year followup of participants in the Framingham Heart Study. Circulation 67:968, 1983

15. Epstein LH, Wing RR, Koeske R, et al: Child and parent weight loss in family-based behavior modification pro-

grams. J Consult Clin Psychol 49:674, 1981

16. Klimov AN, Glueck CJ, Gartside PS, et al: Cord blood high density lipoproteins: Leningrad and Cincinnati. Pediatr Res 13:208, 1979

17. Andersen GE, Lifschitz C, Friis-Hansen B: Dietary habits and serum lipids during the first 4 years of life. Acta

Paediatr Scand 68:165, 1979 18. Golubjatnikov RT, Paskey T, Inhorn SL: Serum cholesterol levels of Mexican and Wisconsin school children. Am J Epidemiol 96:36, 1972

19. Savage PJ, Hamman RF, Bartha G, et al: Serum cho-lesterol levels in American (Pima) Indian children and ado-

lescents. Pediatrics 58:274, 1976

20. Morrison JA, DeGroot I, Edwards BK, et al: Plasma cholesterol and triglyceride levels in 6775 school children, ages 6-17. Metabolism 26:1199, 1977 21. DeGroot I, Morrison JA, Kelly KA, et al: Lipids in

schoolchildren ages 6 to 17 of age: Upper normal limits.

Pediatrics 60:437, 1977 22. Morrison JA, DeGroot I, Edwards BK, et al: Lipids and lipoproteins in 927 school children, ages 6-17 years.

Pediatrics 62:990, 1978

23. Voors AW, Webber LS, Frerichs RR, et al: Body height and body mass as determinants of basal blood pressure in children: The Bogalusa Heart Study. Am J Epidemiol 106:101, 1977

24. Morrison JA, DeGroot I, Kelly KA, et al: High and low density lipoprotein cholesterol levels in hypercholes-

terolemic school children. Lipids 14:99, 1979
25. Marshall WA, Tanner JM: Variations in the pattern of pubertal changes in boys. Arch Dis Child 45:13, 1970
26. Marshall WA, Tanner JM: Variations in the pattern of pubertal changes in sirls. Arch Dis Child 44:204, 1000

of pubertal changes in girls. Arch Dis Child 44:291, 1969

27. Morrison JA, Laskarzewski PM, Rauh JL, et al: Lipids, lipoproteins, and sexual maturation during adoles-The Princeton Maturation Study. Metabolism 28: 641, 1979

28. Morrison JA, DeGroot I, Kelly K, et al: Black-white differences in plasma lipoproteins in Cincinnati schoolchildren (one-to-one pair matched by total plasma cholesterol,

sex and age). Metabolism 28:241, 1979

29. Berenson GS, Srinivasan SR, Frerichs RR, et al: Serum high density lipoprotein and its relationship to cardiovascular disease risk factor variables in children—The Bogalusa Heart Study. Lipids 14:91, 1979

30. Glueck CJ, Gartside PS, Tsang RC, et al: Black-white similarities in cord blood lipids and lipoproteins. Metabo-

lism 26:347, 1977

31. Tyroler HA, Hames CG, Krishan I, et al: Black-white differences in serum lipids in Evans County. Prev Med

4:451, 1975 32. Hennekens CH, Jesse MJ, Klein BE, et al: Cholesterol among children of men with myocardial infarction. Pediatrics 58:211, 1976

33. Morrison JA, Kelly KA, Mellies MJ, et al: Parentchild associations at upper and lower ranges of plasma cholesterol and triglyceride levels. Pediatrics 62:468, 1978

34. Friedman G, Goldberg SJ: An evaluation of the safety of a low-saturated-fat, low cholesterol diet, beginning in infancy. Pediatrics 58:655, 1975

- 35. Davidson DM, Kleeman CR, Quiroga J, et al: Cardiovascular disease risk reduction in a 24-day interdisciplinary university program. Clin Res 30:6A, 1982
- 36. Coulston A, Liu G, Chen Y-D, et al: Effects of low fat-high carbohydrate diets in hypertriglyceridemic subjects. Clin Res 31:64A, 1983

37. Gordon T, Kannel WB, McGee D: Death and coronary attacks in men after giving up cigarette smoking. Lancet 2:1345, 1974

38. Castelli WP, Doyle JT, Gordon T, et al: HDL choles-

- terol and other lipids in coronary heart disease. The Cooperative Lipoprotein Phenotyping Study. Circulation 55:767, 1977
- 39. Morrison JA, Kelly KA, Mellies M, et al: Cigarette smoking, alcohol intake, and oral contraceptives: Relationships to lipids and lipoproteins in adolescent school children. Metabolism 28:1166, 1979

40. Castelli WP, Garrison RJ, Dawber TR, et al: The filter cigarette and coronary heart disease. The Framingham

Study. Lancet 2:109, 1981

41. Teenage smoking. National Patterns of Cigarette Smoking, Ages 12 Through 18, in 1972 and 1974. Public Health Service, National Institutes of Health. DHEW publication No. (NIH) 76-931. Government Printing Office, 1976
42. Stadel BV: Oral contraceptives and cardiovascular disease. N Engl J Med 305:612, 1981

43. Wallace RB, Tamir I, Heiss G, et al: Plasma lipids, lipoproteins, and blood pressure in female adolescents using oral contraceptives. J Pediatr 95:1055, 1979

44. Review Panel on Coronary-Prone Behavior and

Coronary Heart Disease: Coronary-prone behavior and coronary heart disease: A review. Circulation 63:1199, 1981

45. Waldron I, Hickey A, McPherson C, et al: Type A behavior pattern: Relationship to variation in blood pressure, parental characteristics, and academic and social activities of students. J Hum Stress 6:16, 1980

46. Friedman M, Thoresen CE, Gill JJ, et al: Feasibility of altering type A behavior pattern after myocardial infarc-

tion. Circulation 66:83, 1982

47. Mathews KA: Assessment and developmental antecedents of the coronary-prone behavior pattern in children. In Dembroski TM, Weiss SM, Shields JL, et al (eds): Coronary-Prone Behavior. New York, Springer-Verlag, 1978, p 207

48. Waldron I: The coronary-prone behavior pattern, blood pressure, employment and socio-economic status in

women. J Psychosom Res 22:79, 1978

49. Buell JC, Eliot RS: The role of emotional stress in the development of heart disease. JAMA 242:365, 1979 50. Peters RK, Cady LD Jr, Bischoff DP, et al: Physical

fitness and subsequent myocardial infarction in healthy workers. JAMA 249:3052, 1983

51. Larbi EB, Cooper RS, Stamler J: Alcohol and hyper-

tension. Arch Intern Med 143:28, 1983

52. Gordon T, Kannel WB: Drinking habits and cardiovascular disease: The Framingham Study. Am Heart J 105: 667, 1983

53. Keys A: Lipoprotein profile—Its value in prediction.

Prev Med 12:25, 1983

54. Goodwin DW: Is alcoholism hereditary? A review

and critique. Arch Gen Psychiatry 25:545, 1971

55. The Health Consequences of Smoking for Women, a Report of the Surgeon General. Office on Smoking and Health, Office of Assistant Secretary for Health, Public Health Service (Rockville, Md). Government Printing Office,

56. Sandor GGS, Smith DF, MacLeod PM: Cardiac malformations in the fetal alcohol syndrome. J Pediatr 98:771,

57. Rodnick JE: Health behavior changes associated with health hazard appraisal counseling in an occupational setting. Prev Med 11:583, 1982

58. Hall JH, Zwemer JD: Prospective Medicine Health Hazard Appraisal 3. Indianapolis, Methodist Hospital, 1980

59. Puska P, Vartiainen E, Pallonen U, et al: The North Karelia Youth Project: Estimation of two years of intervention on health behavior and CVD risk factors among 13- to 15-year old children. Prev Med 11:550, 1982

60. Dietary Goals for the United States. US Senate Select Committee on Nutrition and Human Needs. Gov-

ernment Printing Office, 1977
61. Weidman W, Kwiterovich P Jr, Jesse MJ, Nugent E: Diet in the healthy child. Task Force Committee of the Nutrition Committee and the Cardiovascular Disease in the Young Council of the American Heart Association. Circulation 67:1411A, 1983

62. Nutrition Committee of the Canadian Paediatric Society: Children's diets and atherosclerosis. Can Med

Assoc J 124:1545, 1981
63. Garn SM, Bailey SM, Higgins ITT: Effects of socioeconomic status, family line, and living together on fatness and obesity. In Lauer RM, Shekelle RB (eds): Childhood Prevention of Atherosclerosis and Hypertension. New York, Raven Press, 1980, pp 187-204 64. Blackburn H: Physical activity and coronary heart

disease. J Cardiac Rehabil 3:171, 1983
65. Perry C, Killen J, Telch M, et al: Modifying smoking behavior of teenagers: A school-based intervention. Am J Public Health 70:722, 1980
66. Rames LK, Clarke MD, Control MD

66. Rames LK, Clarke WR, Connor WE, et al: Normal blood pressure and the evaluation of sustained blood pressure elevation in childhood: The Muscatine study. Pediatrics 61:245, 1978

67. Epstein LH, Wing RR, Kuller L, et al: Parent-child obesity and cardiovascular risk factors. Prev Med 12:437, 1983

68. Berger A: Family involvement and alcoholics' completion of a multiphase treatment program. J Stud Alcohol 42:517, 1981

69. Medalie JH: The family life cycle and its implica-

tions for family practice. J Fam Pract 9:47, 1979