very low calorie diets a few years ago, the Agency required a warning label on products which provide 400 or fewer kcal per day.

In addition to obesity, a related area, the prevalence of eating disorders, particularly anorexia and bulimia, which occur primarily among young women, is of concern. An FDA analysis now under way of data from the Health Promotion and Disease Prevention Supplement to the 1985 National Health Interview Survey suggests that substantial numbers of the thinnest young females in our population state that they are presently attempting to lose weight and are taking active measures to do so by reducing calories. This information certainly interjects caution into the general messages given to women about weight loss.

On the one hand, nutrition is a central component of any health program. On the other hand, there are substantial areas where knowledge is lacking to make definitive statements about the direction to take.

Women's Health: Nutrition

Nutrition and Cardiovascular Diseases of Women

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Synopsis

Atherosclerosis and hypertension are, by far, the most common cardiovascular diseases affecting women, and both are influenced by diet. Atherosclerosis occurs more commonly in men than women; generally women are 10 to 15 years older than men when symptoms develop. The prevalence of hypertension is about equal in the two sexes, particularly in middle aged and older persons. These cardiovascular diseases are major causes of death and disability in this country. Atherosclerosis results in myocardial infarction, thrombotic strokes, and claudication. Hypertension, when severe, damages small blood vessels, causing kidney failure, hemorrhage, strokes, and heart failure; when the condition is mild to moderate, it produces atherosclerosis.

Nutritional factors are of primary importance in both atherosclerosis and hypertension. Risk factors for atherosclerosis related to nutrition are hypercholesterolemia, hyperglycemia-diabetes, and for hypertension, obesity, high salt intake, and excessive use of alcohol. Of all these risk factors, obesity seems to be the most important because it is strongly linked to hypertension and diabetes. Dietary intake of saturated fat is a potent factor in determining the blood cholesterol level, and reducing intake often decreases the level, thus lessening the risk of atherosclerotic complications. Although high salt intake and excessive alcohol use produce hypertension in susceptible people, less is known about the frequency of this adverse effect than is known about obesity.

ATHEROSCLEROSIS AND HYPERTENSION are by far the most common cardiovascular diseases and each is influenced by dietary factors. Much of the information about nutrition and these cardiovascular diseases has come from the work of American epidemiologists as a reflection of the national preoccupation with food and health. Until recently, the emphasis has been on coronary heart disease in men and little attention has been paid to this common cardiovascular disease in women. This is understandable because, although women do suffer from atherosclerosis, they are usually 10 to 15 years older than men when symptoms develop. Now that people are living longer, interest in atherosclerosis and hypertension and the possibility of preventing them have increased greatly. Since dietary factors are known to be important and since they can be manipulated with reasonable ease (or so many people think), there is increasing emphasis on understanding their roles in these cardiovascular diseases and in developing means to incorporate this evidence into programs of prevention.

On the basis of current information, nutritional factors do not differ between men and women. A genetic predisposition for one of the dietary influences seems to have no sex bias. Therefore, this

discussion will not speak specifically to nutrition and these cardiovascular diseases of women, but generally to these relationships in both sexes.

Known risk factors for atherosclerosis are hypertension, hypercholesterolemia, cigarette smoking, hyperglycemia-diabetes, male sex, aging, and genetic traits. Atherosclerosis, with its serious complications of myocardial infarction, thrombotic stroke, and claudication, is for more frequent in persons with hypercholesterolemia and diabetes, both of which are influenced by diet—hypercholesterolemia by the intake of saturated fat and diabetes by obesity due to excess calories. For hypertension, many nutritional factors have been implicated: obesity, high salt intake, excess alcohol use, genetic traits, and decreased intakes of potassium, calcium, and polyunsaturated fatty acids. In fact, there are so many that if all of the suggestions were followed, one would wonder what would be the ideal diet. Fortunately, at the present time only three factors, excess calories leading to obesity, excess dietary salt intake, and excess alcohol consumption, have been clearly established as important and these are not operative in every individual because a predisposition is apparently necessary for any of them to cause hypertension.

Atherosclerosis is a major cardiovascular disease that develops slowly over decades, finally expressing itself as coronary heart disease (angina, myocardial infarction, or sudden death) or as intermittent claudication, thrombotic strokes, or hypertension due to atherosclerotic stenosis of the renal arteries. Although women do not suffer from atherosclerosis as frequently as men, it is a numerically significant health problem in older women.

The primary risk factors for atherosclerosis are hypertension, hypercholesterolemia, diabetes, and cigarette smoking (*1*). The importance of nutrition in atherosclerosis is obvious when one realizes that three of these four major risk factors are influenced by diet.

The association between hypercholesterolemia and atherosclerosis, particularly myocardial infarction, has long been recognized. Much information concerning this relationship has come from studies done in many different population groups so that it is now accepted that cholesterol levels of 280 mg per dl or greater are a potent risk factor for myocardial infarction in men (*2*). Most of the information we have concerns men, and we assume that it is reasonable to extrapolate it to women.

Lipids are transported in the blood attached to proteins as lipoproteins, including the triglyceride-rich, very low density lipoproteins (VLDL), cholesterol-rich, low density lipoproteins (LDL), and high density lipoproteins (HDL). Elevated LDL (that is hypercholesterolemia) is associated with myocardial infarction and peripheral vascular disease, while HDL affords protection against these disorders. In fact, a total cholesterol to HDL ratio of > 4.5 indicates a high probability of developing these atherosclerotic complications. Lipoprotein levels are greatly influenced by diet, including total calories, amount and type of fat (saturated or unsaturated), and carbohydrate and cholesterol intake.

Caloric intake in excess of energy needs results in obesity, which can lead to hypertriglyceridemia, hypercholesterolemia, and decreased HDL (*3*), especially when obesity is primarily of the abdominal type (*4*). Insulin resistance also develops which can result in compensatory hyperinsulinemia and/or frank diabetes. This hyperinsulinemia increases triglyceride synthesis and VLDL production. As a result, LDL levels often rise which, in association with the decreased HDL, increase the risk of atherosclerotic complications.

The relative proportion of dietary saturated fat to unsaturated fat influences all of the major classes of lipoproteins—VLDL, LDL, and HDL. Generally speaking, a high intake of saturated fat increases LDL and VLDL, while raising the proportion of dietary fat that is unsaturated decreases the levels of these lipoproteins. This is the reason for the widely disseminated recommendation that Americans decrease the saturated fat in their diets and increase the polyunsaturated fat (*5*). Part of this recommendation includes an increase in carbohydrate intake because the relative proportion of total fat to carbohydrate intake influences the plasma level of lipoproteins. Usually, if the amount of carbohydrate eaten is increased and the amount of fat decreased, total and LDL cholesterol decrease. With HDL cholesterol unchanged, or increased, the total cholesterol to HDL ratio decreases, which lessens the risk of atherosclerostic complications, The Lipid Research Clinics Coronary Primary Prevention Trial (*6*) showed that for each 1 percent decrease in cholesterol level mortality was reduced by 2 percent.

Diabetes is another potent factor in the development of atherosclerosis; in fact, coronary artery disease is responsible for more than 50 percent of deaths among diabetics (*3*), and peripheral vascular disease in such people is frequent. The nutritional factor in diabetes is excess calories, which causes most maturity-onset diabetes (*7,8*). Abnormalities of lipoproteins occur commonly; the most frequent finding is elevated VLDL, with a lesser frequency of

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elevated LDL and total cholesterol; HDL may decrease. Usually weight reduction and control of hyperglycemia will considerably reverse these abnormal findings.

Hypertension is another major cardiovascular disease with serious consequences. It affects men and women almost equally. Its complications are of two general types: when hypertension is severe, arterioles and small arteries are damaged producing heart failure, hemorrhagic stroke, and kidney failure (*9*); when it is of mild to moderate degree, premature atherosclerosis occurs with all the complications noted previously.

Of the three well-established nutritional factors for hypertension, obesity seems to be the most potent. The association between overweight and hypertension is well-known with information from studies all over the world involving millions of people (*10*). Hypertension is a characteristic of industrialized populations and so is weight gain with age. In contrast, hypertension is rare in primitive populations that are not obese and do not gain weight as they age. In the United States, actuarial, epidemiologic, and longitudinal studies have shown a clear association between relative weight and/or weight gain and hypertension (see reference *11* for literature review). The 1959 Build and Blood Pressure Study of the American Society of Actuaries detailed the association in a large cohort of insured subjects. In the Framingham study, the prevalence of hypertension was found to be influenced by overweight as it was in the screening of a million Americans in the report of Stamler and co-workers. Of great significance for the possible prevention of hypertension is the role of weight gain in young people. The Framingham study showed that weight gain in young adults often presaged the subsequent development of hypertension. Similarly, in the 1,000-aviator study, the development of hypertension during a 24-year followup was found to be strongly influenced by weight gain, and the Evans County, GA, resurvey of young people 7 years after the first examination showed that the incidence and/or worsening of hypertension was related to weight gain in white men and women and black women. In fact, the Evans County data have been interpreted as indicating that if young people stayed lean or, if obese, reduced their body weight to normal, the development of hypertension would be prevented in 48 percent of whites and 28 percent of blacks (*12*). Although there is no clear understanding of the mechanism of obesity-related hypertension, one thing is clear, and that is the frequent blood pressure-lowering effect of weight reduction.

Excess salt produces hypertension in susceptible individuals, and some fraction of essential hypertensive patients achieve normotension with salt restriction. Further, hypertension is rare in primitive populations that have a salt intake of less than 50 meq per day. These facts and the ability to produce hypertension in animals by feeding large amounts of salt have led to the widely held belief that all essential hypertension is caused by the habitual salt intake of industrialized societies. The extension of this belief is that reducing salt intake would prevent the development of hypertension (see reference *13* for literature review).

Excess salt raises arterial pressure in susceptible individuals. However, as yet we have no information about the true frequency of that susceptibility. For instance, Kirkendall and associates found no increase in blood pressure in normotensive men fed 240 meq of sodium per day (14 g per day) for 1 month and Luft and co-workers found no change in blood pressure in normal subjects during 3-day sequential increments of sodium intake until a level of 400 meq per day (23 g of salt per day) was reached (*13*). In contrast, Falkner and co-workers (*14*) produced modest pressure increases with 10 g of salt per day added to the usual sodium intake of adolescents who had hypertensive parents, but not in those with normotensive parents.

Weinburger and co-workers (*15*) evaluated the sodium sensitivity of 375 normotensive subjects and 192 hypertensive patients from their arterial pressure responses to 2 l of normal saline given intravenously over 4 hours followed the next day by salt depletion achieved with a 10-meq sodium diet and three 40-mg doses of furosemide. They found that 26 percent of the normotensive subjects had a fall in mean arterial pressures (MAP) of $>$ 10 mm Hg but 51 percent of the hypertensive patients had MAP responses of this degree. The two groups did not differ in their MAP increases when saline was infused, but the average MAP increased only about 3 mm Hg. Thus, the arterial pressure of hypertensives is probably more salt sensitive than that of normotensives. It is

difficult to extrapolate these results to a free-living population which is advised to decrease salt intake to less than 5 g per day to reduce the pressure of those who are hypertensive and to prevent hypertension in those with normal pressure (*16,17*). However, at the least it would not be harmful, and at the most, it might help a few hypertensive patients because moderate salt restriction has been shown to reduce pressure in small groups of patients with mild hypertension.

Excess alcohol intake has been recognized only recently as a risk factor for hypertension. Results of several cross-sectional studies involving large numbers of subjects have clearly shown an association between increasing daily alcohol intake and rising blood pressure (*18,19*). The prevalence of hypertension has been found to be greater in drinkers than nondrinkers; it has been estimated to be 50 percent greater in people consuming three to four drinks per day and 100 percent greater with six to seven drinks per day, compared with abstainers. Further, it has been calculated that about 5 percent of hypertension can be accounted for by the intake of more than three drinks per day.

Abstinence has been reported to result in a considerable and rapid decrease in arterial pressure of hypertensive men who consumed six to eight drinks per day, and even in normotensive men 4 days of abstinence produced a significant fall in pressure. The Kaiser-Permanente study of more than 66,000 people found that the association between blood pressure and alcohol intake was stronger in men than women, in whites than blacks, and in persons over 55 years of age (*20*).

In conclusion, there seems little question that dietary factors play a major role in atherosclerosis and hypertension. Of these the most important appear to be excess calories and the resultant obesity. Obesity is a major associate of hypertension and is clearly related to maturity-onset diabetes; these two conditions are among the most important risks for atherosclerosis. There is every likelihood that prevention of obesity in industrialized nations would substantially reduce the occurrence of hypertension and atherosclerosis leading to a decreased mortality from these vascular diseases.

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