A fundamental goal of the American Heart Association (AHA) is to prevent cardiovascular disease and, in particular, to reduce the incidence of coronary heart disease (CHD) and other atherosclerotic diseases in our society. A question of great concern to the AHA has been whether the American diet is a significant factor in the genesis of atherosclerosis. This question has been the subject of continuous dialogue within the Association for at least 25 years. Although many and frequently diverse opinions have been voiced within the several committees of the AHA, a concerted effort has been made to integrate the best available evidence on the subject. It is clear that the "diet-heart question" is complex, and new information is emerging continually. Still, a broad base of knowledge has been accrued, and because of the urgency engendered by the high incidence of CHD in the U.S., the AHA has felt a responsibility to provide the best possible guidance to the medical community and the American public on the diet question. For this reason the policy of the AHA has been to frequently update recommendations on the basis of the best currently available evidence. The rationale and documentation for particular recommendations of the AHA are discussed below.

I. History of AHA Diet Statements, 1957 to Present

In 1957 a group chaired by Dr. Irvine H. Page prepared a report to the AHA summarizing the evidence on the relationship between diet and atherosclerosis. A review of the available data led these investigators to the following conclusions: (1) diet may play an important role in the pathogenesis of atherosclerosis; (2) the fat content and total calories of the diet may be the dominant contributing factors; and (3) the type of fat, or the balance between saturated and certain unsaturated fats, also may be important. In 1961 an ad hoc committee authorized by the AHA prepared an updated report on the possible relation of dietary fat to heart attacks and strokes. The authors of this report reached the following conclusions: (1) overweight persons should decrease their caloric intake and attempt to achieve their desirable body weight; (2) weight reduction should be facilitated by regular, moderate exercise; (3) the composition of the diet should be altered by reducing intakes of total fats, saturated fats and cholesterol, and by increasing polyunsaturated fats; (4) particular attention should be given to dietary alteration by men at increased risk for CHD (e.g., those with a previous atherosclerotic event, a strong family history of CHD, elevated plasma cholesterol or hypertension); and (5) for those at high risk, dietary changes should be carried out under medical supervision.

Since the time of these recommendations, the AHA has continuously reviewed new data pertaining to the possible relationship between diet and CHD. The responsibility for updating recommendations about diet has rested primarily with the Nutrition Committee. This committee consists of a group of scientists representing several disciplines (especially epidemiology, pathology, nutrition and metabolic diseases). It meets two to three times per year to evaluate new information, to reexamine its position in the light of this information and, when appropriate, to develop new recommendations. Tenure of membership on the committee is 3 years, and care is taken to recruit new members who are experts in their fields. The Nutrition Committee works closely with the various councils of the AHA (for example, Arteriosclerosis, Hypertension, Epidemiology, Cardiovascular Disease of the Young, and Clinical Cardiology) to obtain their views. The product of these deliberations is subjected to systematic review by the Council Affairs Committee and Steering Committee before it is finally approved. The development of a new statement on diet and CHD usually takes 3-5 years.

The AHA issued another statement on diet in 1965. It recommended caloric restriction to achieve desirable weight, substitution of polyunsaturated fats for saturated fats where possible, reduction in cholesterol intake and inclusion of the whole family in dietary changes. The 1965 report did not specify precise quantities or set limits for the different dietary constituents to achieve these goals. In 1968 a new statement followed the same general recommendations, but it attempted to define more precisely desirable intakes of different nutrients. Weight reduction was again recommended. Intake of fat was set at 30-35% of total calories with a distribution of one third saturates, one...
third monounsaturates and one third polyunsaturates. A reduction of cholesterol intake to less than 300 mg/day also was recommended.

Since 1968 two statements have been issued, one in 1973 and the most recent in 1978. The AHA has continued to recommend that the general public consume a diet containing no more than 30-35% of calories as fat; further, the decrement in saturated fats should be replaced by complex carbohydrates and polyunsaturated fats, but intake of polyunsaturates should not exceed 10% of total calories. Cholesterol intake again was limited to 300 mg/day. Finally, relatively low intakes of salt were suggested because evidence exists that current levels of sodium intake of polyunsaturates should not exceed 10% of total calories. Cholesterol intake again was limited to 300 mg/day.

Table 1. Ten-Year CHD Deaths per 10,000 Males by Level of Total Serum Cholesterol (Standardized at Age 50)*

<table>
<thead>
<tr>
<th>Population (male)</th>
<th>At risk</th>
<th>Events</th>
<th>Total serum cholesterol (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>U.S. railroad</td>
<td>2246</td>
<td>93</td>
<td>247</td>
</tr>
<tr>
<td>Finland</td>
<td>1450</td>
<td>64</td>
<td>270</td>
</tr>
<tr>
<td>Mediterranean†</td>
<td>3321</td>
<td>41</td>
<td>103</td>
</tr>
</tbody>
</table>

*Data adapted from Keys. 1980.
†Greece, Yugoslavia and Italy.

**Reduction In Saturated Fatty Acids**

Saturated fats repeatedly have been shown to raise plasma cholesterol and LDL levels. The current American diet contains about 40% of total calories as fat, of which about 15 - 17% are derived from saturated fats. The major sources of saturated fats are meat, animal fats, some vegetable oils (palm oil, coconut oil, cocoa butter, and heavily hydrogenated margarines and shortenings), dairy products (whole milk, cream, butter, ice cream and cheese) and bakery goods. The AHA recommends a reduction of saturated fat intake to less than 10% of total calories. Equations developed in carefully controlled clinical studies have shown that a reduction in saturated fats from 17% to 10% of total calories will decrease plasma cholesterol about 20 mg/dl.

A reduction in saturated fats requires consideration of what should be substituted in their place. For an overweight person, removal of saturated fat can decrease total calories and promote weight reduction. For people of normal weight, saturated fats can be replaced by unsaturated fats and complex carbohydrates.

**Substitution of Unsaturated Fats**

The fatty acids of these fats can be either monounsaturated or polyunsaturated. Oleic acid is the major representative of the former; linoleic acid is the dominant polyunsaturated fatty acid. Both types of fatty acids lower plasma cholesterol when substituted for saturated fats. Linoleic acid is somewhat more effective in cholesterol reduction than oleic acid and, consequently, there has been a widely held belief that saturated fats should be replaced as much as possible by polyunsaturated fats; this belief may be one explanation for a gradual increase in polyunsaturates in the U.S. diet. Still, at present, they only provide 5-6% of total calories. The AHA has been cautious about recommending marked increases in polyunsaturates; the consequences of prolonged ingestion of large quantities of these fats are not known. To be on the safe side, the AHA has not recommended very high intakes of polyunsaturated fats for the general population. While some replacement of saturated fats by polyunsaturates seems safe, it may be prudent not to exceed 10% of total calories.

Another substitute for saturated fatty acids could be monounsaturated fatty acids. The latter do not reduce plasma cholesterol quite as much as polyunsaturates, but they have certain potential advantages. The human body synthesizes considerable quantities of monounsaturated fatty acids, and thus these fatty acids probably...
do not possess any inherent "toxicity." Large amounts of monounsaturates are ingested as olive oil in the Mediterranean region without evidence of adverse effects. Indeed, the prevalence of CHD in this region of the world is relatively low compared to countries where ingestion of saturated fats is high. At present, no inexpensive sources of natural monounsaturated fats similar to olive oil are readily available in the U.S., but there are several potential sources of monounsaturates which could be explored were they deemed a useful substitute for saturated fats. Actually, a major portion of monounsaturated fatty acids in the U.S. diet are consumed with animal fats, and a reduction of saturates to 10% of caloric intake would produce a similar decrease in monounsaturates. The AHA does not at present recommend increasing these monounsaturated fatty acids since total fat calories would then exceed 30%. Instead, the recommendation is to replace saturated fats principally with polyunsaturates and carbohydrates.

Increase in Carbohydrates

Reducing caloric intake from fats to a maximum of 30% would require an increase in carbohydrates from 45% to 55% of total calories. This increase probably should take the form of complex carbohydrates: the long-chain carbohydrates, as contained in vegetables, beans, cereals and some fruits, seem preferable to simple sugars because they impart less risk for dental caries, avoid surges of hyperglycemia and provide more roughage to the diet. When carbohydrates are substituted for saturated fatty acids, plasma cholesterol and LDL usually fall; this decline is in the range of that with monounsaturated fatty acids and somewhat less than with polyunsaturates. An increase in dietary carbohydrate causes a rise in the plasma triglyceride level of fasting patients, but in persons without abnormal lipid levels the rise is usually small and transient. Furthermore, a reduction in fat intake decreases the rise in plasma triglycerides that normally follows fat ingestion. Finally, in populations habitually consuming high-carbohydrate diets, plasma triglycerides are not increased noticeably. Thus, the low total cholesterol on a low-fat, high-carbohydrate diet would seem to outweigh any slight increase in plasma triglycerides.

Substantial Reduction in Dietary Cholesterol

Most carefully controlled metabolic studies have shown that dietary cholesterol increases total plasma cholesterol, but undoubtedly there is much individual variation. The AHA recommends a further reduction to less than 300 mg of cholesterol per day. For each 100 mg per day decrease in dietary cholesterol, the total plasma cholesterol falls an average of about 7 mg/dl.

Caloric Intake Adjusted To Achieve and Maintain Desirable Weight

When diet-related risk factors are present they usually are accentuated by obesity and its associated increased caloric intake. Excess body weight frequently is associated with high VLDL, moderately high LDL, low HDL, hypertension and diabetes mellitus. Furthermore, in young and early middle-aged men, obesity seems to enhance risk for CHD independent of its effect on these other risk factors. Several reports indicate that correction of obesity can reduce plasma lipid levels increase HDL, decrease blood pressure and improve glucose tolerance. Thus, avoidance of obesity beginning early in life, or a supervised weight reduction for those above their desirable weight, is strongly recommended. Moderate exercise is a useful adjunct to caloric restriction in a program for weight control.

### III. Rationale and Documentation of AHA Position on Diet

**Evidence for a Diet-CHD Relationship**

Early investigations demonstrated that dietary cholesterol can cause atherosclerosis in several animal species. This finding led to the concept that excess dietary cholesterol also may accelerate atherosclerosis in man. In animals the atherogenic influence of dietary cholesterol appears to be mediated to a large extent through the plasma cholesterol. When plasma cholesterol is raised by dietary cholesterol, atherogenesis is enhanced; in animals resistant to hypercholesterolemia, atherosclerosis usually is minimal. Therefore, in animals, cholesterol and perhaps other dietary components seem atherogenic in proportion to their hypercholesterolemic effect. The same could be true for man, but before the validity of this hypothesis can be considered, it seems a prerequisite to review evidence relating plasma cholesterol, or lipoproteins, to human atherosclerosis.

**Plasma Cholesterol, Lipoproteins and CHD**

**Epidemiologic Studies**

Several large epidemiologic surveys have shown a significant correlation between total plasma cholesterol and the incidence of CHD. These surveys have been carried out both in the U.S. and other countries. One of the more germane to the U.S. population is the Pooling Project, in which the data from several similar epidemiologic studies in the U.S. were pooled. The project compared CHD risk to serum cholesterol concentrations through five steps of increasing cholesterol levels. The populations under study were men aged 40-64 years. The five quintiles of serum cholesterol were I (< 194 mg/dl), II (195-218 mg/dl), III (219-240 mg/dl), IV (241-268 mg/dl) and V (> 268 mg/dl). There were no significant differences in CHD rates between quintiles I and II, but risk for CHD increased in the higher quintiles (risk ratios 1.5, 1.64 and 1.99, respectively, for quintiles III, IV and V as compared to I and II). These differences in risk were greater at younger ages. For instance, at ages 45 to 49 the risk ratio of IV/III+II was 2.9 and of V/III+II was 3.6. With advancing years the differential risk between quintiles declined; for example, at ages 60 to 64 the relative risk for quintiles V/III+II was only 1.5. This does not mean that elevated plasma cholesterol is less dangerous in older people; however, the absolute number of patients with higher cholesterol levels rises with age so that more people are in the higher quintiles and thus are at greater risk in the older age groups.

Generally, total cholesterol concentrations are a good reflection of LDL levels, and the high correlation between plasma cholesterol and CHD risk thus suggests that high concentrations of LDL is atherogenic. In support, recent epidemiologic studies indicate that elevated LDL is an in-
dependent factor. A direct relationship between LDL concentrations and extent of coronary atherosclerosis has been shown in several angiographic studies. Epidemiologic studies also show an inverse correlation between CHD rates and HDL levels in populations with relatively high levels of total plasma cholesterol. On the other hand, low HDL may not increase risk for CHD in populations in which LDL concentrations also are low. Thus, in the absence of relatively high LDL, mildly decreased HDL may not be particularly dangerous.

Finally, most epidemiologic studies have found a stronger correlation between plasma cholesterol and CHD than for plasma triglycerides; there are exceptions, however. Clinically, many patients with CHD have raised triglyceride levels, and hypertriglyceridemia may denote increased risk in some patients. Still, it may be questioned whether high triglycerides are an independent (or causative) risk factor; large amounts of triglycerides are not found in atherosclerotic plaques. A high plasma triglyceride may accompany some other abnormality in lipoprotein metabolism that is associated with greater risk. For example, high triglycerides can be associated with low HDL, higher LDL or β-VLDL, all of which seemingly have atherogenic potential. Elevated triglycerides, at least for some patients, may be a marker for increased CHD risk even if they are not a causative factor.

What is the Ideal Plasma Cholesterol?

Few investigators doubt that high concentrations of plasma cholesterol (and LDL) contribute to coronary atherosclerosis. However, the relation between CHD and cholesterol levels has not been shown to be linear over a broad range of cholesterol concentrations. We might inquire as to the ideal plasma cholesterol level. In a recent workshop a group of epidemiologists, clinical investigators and experimental pathologists attempted to develop a consensus on the ideal cholesterol level from data of several kinds. They concluded that the ideal level for adults would be in the range of 130 — 190 mg/dl (mean 160 mg/dl).

A question of importance is whether the relationship between plasma cholesterol and CHD is linear over a broad range of cholesterol levels. The results of the Pooling Project might be taken as evidence against such a relationship; in this study the risk for CHD in men with cholesterol levels in the first quintile (<194 mg/dl) was not clearly reduced compared to those in the second quintile (195-218 mg/dl). Men with especially low plasma cholesterol may have had less CHD, but the evidence was weak. Evidence for a linear association, on the other hand, comes from multi-country epidemiological studies in which CHD risk is extremely low. In these populations cholesterol levels are also very low. In reality the relationship probably is curvilinear, so that above a certain "threshold" region, risk accelerates with rising cholesterol levels. If the results of the Pooling Project are taken as evidence, this threshold range for total cholesterol probably is in the range of 200-220 mg/dl, and desirable cholesterol concentrations would be below this level. This conclusion is consistent with the above-proposed ideal range of 130-190 mg/dl.

Although the concept of a desirable range for plasma cholesterol probably has validity, it may have to be modified under certain circumstances. For example, a reduction of plasma cholesterol to 200 mg/dl might not reduce CHD risk to an acceptable level if a person smokes or has either hypertension or diabetes mellitus. Furthermore, plasma total cholesterol is not the only lipid factor related to CHD. Concentrations of HDL and other lipoprotein subtypes, or their components, may affect CHD risk independent of total cholesterol. The same may be true of the lipid composition of the diet, as is discussed subsequently. Thus, the concept of a desirable range for total cholesterol probably pertains more to population than to individuals.

Genetic Hyperlipidemia

Although dietary factors undoubtedly raise plasma cholesterol, genetic influences on lipoprotein concentrations must not be overlooked. Genetic factors probably predominate in people whose plasma LDL concentrations are above the 90-95th percentile of the population. Some patients at these high levels have monogenic forms of hypercholesterolemia. For instance, at least one in 500 people has familial hypercholesterolemia, a disorder associated with severe elevations of plasma LDL, defective clearance of LDL and very premature CHD. Another monogenic disorder is "familial combined hyperlipidemia"; this disease is characterized by increases in LDL, VLDL or both, and it too is associated with premature CHD. Yet another monogenic disease is "familial dysbetalipoproteinemia" which is manifested by the abnormal lipoprotein β-VLDL and premature atherosclerotic disease. These monogenic hyperlipoproteinemias provide strong support for the concept that elevated plasma lipoproteins can accelerate atherosclerosis. They also show that not all hyperlipidemias are dietary in origin, and if not, dietary change alone may not normalize plasma lipids in these disorders; drug therapy may be required in addition to diet modification.

Most of the remainder of patients with cholesterol levels above the 90-95th percentile probably have polygenic hypercholesterolemia. Again, dietary factors may affect plasma cholesterol in these patients, but the dominant cause of their hypercholesterolemia seemingly is the interaction of multiple genes. Thus, drug therapy may be needed to reduce cholesterol levels in these patients to the desirable range.

Finally, consideration should be given to the role of heredity in raising plasma cholesterol into the 50-90th percentile of the U.S. population, which probably is above the desirable range. Polygenic factors likely are at play here as well, but diet often may be the major factor. In the mildly elevated range, diet probably acts on a genetic foundation, but despite hereditary influence, dietary change alone may be sufficient to reduce plasma cholesterol to desirable levels.

Diet and CHD

Evidence of several kinds strongly supports the concept that the composition and quantity of dietary constituents can influence rates of atherogenesis and thus affect risk for CHD. These data can be reviewed briefly.

Diet and Atherosclerosis in Experimental Animals

Many studies have shown that diet can induce atherosclerosis in animals. As a general rule, the athero-
genic effect of diet appears to be mediated mainly through the plasma lipoproteins. In animals which are resistant to diet, cholesterol levels usually are not increased. On the other hand, in responsive animal species, even mild rises in cholesterol concentrations, to the range found in the U.S. population, are associated with deposition of cholesterol in the arterial wall. While the animals studies in general support the notion that ingestion of saturated fats and cholesterol can accelerate atherosclerosis, it is important to note that hypercholesterolemia induced by diet in most species is associated with unusual types of lipoproteins. Most typically the increase occurs in cholesterol-rich particles, β-VLDL74,75 or large LDL76 which normally do not occur in significant quantities in man. These abnormal lipoproteins may be unusually atherogenic. Nevertheless, despite differences between experimental animals and man, the basic concept that dietary factors can induce abnormalities in cholesterol transport which in turn can accelerate atherosclerosis almost certainly is valid. Note-worthy are investigations in primates showing the atherogenic potential of dietary cholesterol and saturated fats in species not far removed from man.77-83

Epidemiologic Studies

Two kinds of investigations have been carried out to determine the influence of diet on CHD. These are epidemiologic surveys and intervention trials. Epidemiologic surveys and population comparisons may be considered “clinical trials of nature.” As such, they have definite advantages over man-made trials. First, they encompass large numbers of subjects, whereas intervention trials frequently have too few participants to draw definite conclusions; and second, they eliminate the problem of duration because the population under study usually has maintained a constant dietary pattern for many years. On the other hand, they are more subject to misinterpretation because of confounding variables. A short summary of the major epidemiologic studies thus would seem worthwhile.

Geographic Pathology of Atherosclerosis. In 1960 an extensive pathological study reported the extent of atherosclerosis in approximately 21,000 people in 15 countries throughout the world.84 An attempt was made to correlate the severity of aortic and coronary atherosclerosis with particular dietary habits of different populations under study. The data of this project revealed a high correlation between the estimated level of fat in the diet and the severity of atherosclerosis. A similar association was found between the percentage of fat in the diet and cholesterol concentrations in serum, and the latter in turn was highly correlated with the extent of atherosclerosis.

The Seven Countries Study.85 This cross-population study was designed to compare dietary composition and energy expenditure among men of different countries including the regions of northern and southern Europe, the United States and Japan. The data showed a high correlation between percent of calories as saturated fat and CHD deaths. A similar relation was noted between saturated fats and serum cholesterol and between the latter and CHD deaths. Thus, the results are strongly suggestive that amounts of dietary saturated fats affect the incidence of CHD, at least in part through their effect on plasma cholesterol. Since the intakes of dietary cholesterol were not measured in this study, its contribution is unknown.

The Ni Hon-San Study.86-91 This project compared CHD deaths among Japanese living in Japan, Hawaii and San Francisco. Intakes of saturated fat calories for the three populations were estimated to be about 7%, 12% and 14%, respectively. With increasing intakes of saturated fats, dietary cholesterol and mean body weights also rose. Serum cholesterol was higher in proportion to the greater intakes of saturated fats and cholesterol; they were 12% greater in Hawaii and 21% higher in San Francisco than in Japan. Compared to Japan, death rates from CHD were 1.7 times higher in Hawaii and 2.8 times greater in San Francisco. Thus, in a population containing a high degree of genetic homogeneity, the composition of the diet correlated significantly with both serum cholesterol and CHD mortality.

Seventh-Day Adventist Study.92-96 This project compared CHD mortality rates in Seventh-Day Adventists who were predominately lacto-ovo-vegetarians with an age-matched group in the general population of California. Relatively low levels of serum cholesterol have been shown repeatedly in this population. Although Seventh-Day Adventists may be protected from CHD in part by an absence of other risk factors (e.g., lack of smoking), their low cholesterol levels, apparently due to dietary habits, probably contribute to their low CHD death rates relative to the control population.

The Western Electric Study.97 Recently, the results of another survey were reported in which diet, serum cholesterol and other variables were examined in 1900 middle-aged men. These men were examined at entry into the study, one year later and, finally, twenty years later. Scores summarizing each participant’s dietary intake of cholesterol, saturated fatty acids and polyunsaturated fatty acids were calculated according to standard equations relating diet to plasma cholesterol.13,14 There was a positive association between diet score and serum cholesterol concentration at the initial examination, a positive correlation between change in diet score and change in serum cholesterol concentration from the initial to the second examination, and a positive association prospectively between mean base-line diet score and the 19-year risk of death from CHD. These correlations were maintained after adjustment for potentially confounding factors.

These epidemiologic studies are consistent with the theory that intakes of saturated fats and cholesterol affect incidence of CHD. So are a host of other studies not discussed here.98-113 It must be pointed out that several epidemiologic studies done within the United States have failed to observe significant correlations among dietary fat, serum cholesterol concentrations and CHD rates114,115; however, because of considerable genetic heterogeneity and dietary homogeneity in the U.S. population, it is not surprising that correlations were not found, and because of limitations of design these negative findings do not necessarily mean that correlations do not exist. It is important to note that the Western Electric Study97 was able to detect a correlation between diet and CHD rates within a large group of U.S. men.

Despite the many observations of a relationship between dietary factors and CHD, as described above, the various studies have been criticized because all the factors that might affect CHD risk (other than diet) were not held constant. Therefore, we might consider the results of dietary intervention trials to test the hypothesis that dietary change per se may not affect atherogenesis.

Dietary Intervention Trials

Several clinical trials to test whether dietary alteration...
would change CHD risk were started in the 1950s and 1960s. Two such studies were carried out in Chicago and New York using free-living men following a prudent diet.116-118 These "first generation" trials were inadequate because of their small sample size and lack of randomized control groups. Several other large-scale clinical trials have been attempted and while they have limitations, the findings are nevertheless of interest. Generally, these trials have tested diets higher in polyunsaturated fat content than that recommended by the AHA for the general public. Nonetheless, they addressed the question of whether lowering of plasma cholesterol by dietary means can reduce risk of CHD.

One trial involved institutionalized men and women in two mental hospitals in Helsinki, Finland.119 The study employed a crossover design with two periods of about 6 years each. A diet low in saturated fats and cholesterol and high in polyunsaturated fats produced a decrease in serum cholesterol and an apparent decrease in CHD mortality rates. A similar trial was carried out in a Minnesota mental hospital; it also compared a diet low in saturated fats and cholesterol and high in polyunsaturated fats with a control group on a diet high in saturated fats. A preliminary report of this trial120 indicated that rates of coronary events were less in men under 40 years on the cholesterol-lowering diet, but they were not different in control and diet-treatment groups for older men and women.

Another dietary trial was conducted at the Veterans Administration domiciliary facility in Los Angeles121; a double-blind experimental design with a randomized control group was used for the study. The results suggested that a diet low in saturated fats and cholesterol and high in polyunsaturated fats reduced coronary events; in spite of the improved experimental design, the study has been criticized because it was a mixed trial (i.e., partly primary and partly secondary prevention), had too few subjects and the subjects were too old at entry (average age 65.5 years). Additionally, total death rates in the two experimental groups were the same.

Finally, two intervention trials have been carried out in Oslo, Norway. The first, reported in 1966,122 was a secondary prevention trial using a low-cholesterol, high-polyunsaturated fat diet. The results suggested that use of this diet reduced recurrent rates of myocardial infarction. The findings of the second trial have been reported recently.123 This primary prevention trial examined whether lowering of serum lipids by a polyunsaturated fat diet and reduction of smoking would decrease the incidence of new CHD in 1,232 high-risk patients studied for 5 years. At the completion of the trial, myocardial infarction and sudden death were 47% lower in the treated group than in the controls. Statistical analysis revealed that reduction in CHD in the intervention group was due mainly to a decrease in plasma cholesterol by diet and, to a lesser extent, to a reduction of smoking.

Although all the above studies were flawed in one or another aspect of experimental design, they were uniform in reporting a favorable trend toward decreased CHD risk with cholesterol-lowering diets. Still, a major problem was that too few patients were studied for too short a time to be certain about this conclusion. In the view of some investigators, a definitive answer to the diet-heart question cannot be obtained until an adequate primary-prevention trial has been carried out. Indeed, the lack of such a trial is the major criticism against giving recommendations about diet to the U.S. public. For this reason, the National Institutes of Health undertook a thorough review of the requirements for an adequate trial124,125 but concluded that a major, large-scale diet trial should not be undertaken. The following were some of the reasons for rejecting the trial:

First, the most direct approach to the diet-heart issue would be an induction trial, preferably beginning early in life, to test whether a diet high in saturated fats and cholesterol does indeed raise a higher rate of CHD, or one low in saturated fats and cholesterol, all else controlled. Since such a trial obviously is out of the question, the next best approach would be a preventive trial. To insure a definite answer, any primary-prevention trial would be extremely costly. A sample size of at least 60,000 participants, and perhaps more, would be required. At least a decade would be needed to complete the study. Furthermore, a diet-heart trial of adequate size would be impossible in a closed population — the number of people required are not available — so that the use of an open population would be necessary. The latter immediately raises many difficult questions: (1) How would a double-blind trial using different diets in a large number of people, who are rightly concerned about their risk for CHD, be possible? (2) How could other risk factors — blood pressure, smoking, and obesity — be regulated and controlled? (3) How could motivation and adherence be insured for such a long study? (4) How would it be possible to maintain a constant population under investigation in a society of great mobility such as that of the U.S.? Even if these problems could be overcome, the cost in dollars would be prohibitive under the present system for funding clinical research. In a word, a major diet-heart prevention trial probably cannot and likely never will be carried out in the U.S., and a decision about dietary recommendations will have to be made from currently available data. Perhaps the forthcoming data of the Multiple Risk Factor Intervention Trial (MRFIT)126,127 and the Lipid Research Clinic Coronary Prevention Trial128,129 will provide additional support for the diet-heart theory, but these trials cannot substitute for a major diet-heart study.

Effects of Diet on Plasma Lipoproteins in Man

Results from experimental animals, epidemiologic surveys and clinical trials all provide highly suggestive evidence that diet can affect risk for CHD. Beyond these studies, however, are two well-established facts: dietary saturated fats and cholesterol directly raise the plasma total- and LDL-cholesterol, and high total- and LDL-cholesterol contribute directly to atherosclerosis and CHD. While these two facts may not prove a direct link between diet and CHD, the existence of such a link is almost inescapable. Thus, the combined data implicating the U.S. diet to the high incidence of CHD in our society present a strong challenge to the dietary habits of most Americans. Because of this challenge, the AHA has deemed it necessary to make a judgment on the probable best diet for the U.S. public. It recognizes that failure to make a recommendation is to condone the current American diet, i.e., a high intake of total calories, saturated fats and cholesterol.

Documentation of AHA Diet Statements

The above comments underlie the rationale for making a diet recommendation for prevention of CHD. Specific recommendations also were delineated. These recommendations have emerged through years of deliberations...
Nutritional Adequacy

In some countries of the world where a low-fat, high-carbohydrate diet is consumed, malnutrition is common. This has led some to ask whether the modified-fat diet recommended by the AHA can be made nutritionally adequate. Extensive analysis of possible menus has shown that this diet can easily be made adequate in essential nutrients — protein, vitamins and minerals — and need not be deficient in any required nutrient. Clearly, a diet with 25-30% of calories as fat need not predispose to infection. This level of fat intake is higher than in the Japanese diet which has only 10-15% of calories as fat; of interest, life expectancy in Japan exceeds that of the U.S. at every age.16,17 A somewhat better life expectancy also is found in Italians who eat less saturated fats and cholesterol than Americans.16,17 In a word, a diet low in total fat, saturated fats and cholesterol need not be nutritionally inadequate. Current cost estimates suggest that most people can meet the AHA diet recommendations, which are nutritionally adequate, with no more expense than the current U.S. diet which contains sizable quantities of relatively expensive animal products.

The question of nutritional adequacy is particularly important when diets are recommended for children. To date, the AHA has not singled out children or adolescents for specific recommendations, but the current diet statement stresses the need for diet modification for the whole family. There is no evidence that the AHA diet would be deleterious to children or adolescents. A radical change in basic nutrients is not proposed. Adequate amounts of high-quality protein can be obtained from skimmed milk, fish, chicken and a variety of plant foods. These foods can provide sufficient amounts of calcium, vitamins and other nutrients needed for growth. Again, the example of the Japanese diet demonstrates the possibility of nutritional adequacy of a low-fat diet.

Effectiveness of Diet in Plasma Lipid Lowering and Potential Impact on CHD

Since dietary change cannot drastically reduce plasma cholesterol levels, the potential impact of the AHA diet on CHD rates becomes an important issue. If the standard equations derived from metabolic ward investigations are employed, the proposed AHA diet should decrease plasma cholesterol by 30-40 mg/dl as compared to the current U.S. diet.12-15 A somewhat greater reduction might be achieved in obese subjects by weight reduction, and at the same time their HDL levels would be increased.34 The effect of such a change in cholesterol levels on CHD undoubtedly would depend on a variety of factors including the duration of the change, the age at which it occurred and the initial level of plasma cholesterol. Although a change in plasma cholesterol of 30 to 40 mg/dl may have a relatively small impact on risk for CHD when initial levels of cholesterol are near 200 mg/dl or below, some reduction in risk might still be possible. For people whose cholesterol is above this desirable range, however, a substantial dietary change should reduce plasma cholesterol sufficiently to decrease risk of CHD significantly.

Furthermore, the full impact of dietary change on CHD may not be predicted from equations derived from short-term diet studies conducted on the metabolic ward. Life-long adherence to a diet low in total calories, saturated fats and cholesterol actually may produce a greater lowering of cholesterol levels than observed in short-term dietary studies. Evidence for a long-term effect of diet is found in special groups of people whose mean cholesterol levels are lower than would be predicted from equations developed on the metabolic ward.13-15 One example is vegetarians of a Boston commune who eat principally whole grains, beans and fresh vegetables.139 When plasma cholesterol levels in the Boston group were compared to age-matched controls in Framingham, Massachusetts, their levels of total cholesterol and LDL-cholesterol were 31% and 38% lower, respectively, than the controls. Another report140 found plasma total cholesterol and LDL-cholesterol to be 33% and 45% lower in men and women vegetarians (ages 30-40) living in a Tennessee community compared to age-and sex-matched non-vegetarians in St. Louis. The findings in these two groups of American vegetarians suggest that the much lower cholesterol concentrations in people on very low intakes of saturated fats and cholesterol (such as the Japanese), as compared to most Americans, may be related more to diet than to racial or genetic factors. This lack of genetic influence is also supported by the results of the Ni-Hon-San study.85-91 Of interest in the dietary trial at the Los Angeles Veterans Administration domiciliary,121 prolonged ingestion of a diet rich in polyunsaturated fats resulted in gradual but continuous decline in total plasma cholesterol over several years. Finally, another phenomenon possibly reflecting the long-term effect of diet is the accentuated rise in plasma cholesterol which occurs with aging in the U.S. population.92 Although this rise may be universal, it is less marked in other populations,141 and the pronounced rise in Americans could be related to weight gain with aging or to prolonged intakes of saturated fats and cholesterol.

Finally, serious consideration must be given to the possibility that the influence of diet on CHD may not be linked only with plasma cholesterol and LDL levels. Other poorly understood mechanisms may be involved. The findings in the Seven Countries Study are in accord with an independent effect of diet. In table 1142 the 10-year probability of CHD death by serum cholesterol level in CHD-free men at age 50 are compared for U.S. railroad workers, Finnish men, and men from the Mediterranean region (Greece, Yugoslavia and Italy). At similar cholesterol levels, risk was about the same in U.S. and Finnish men, but rates were much lower in Mediterranean men. In the former two groups, intakes of fats averaged 40% of total calories. Saturated fats and cholesterol constituted a relatively large proportion of the calories from fat. In Mediterranean men, intakes of total fats and cholesterol were lower, and monounsaturates were the major type of fat ingested. A discrepancy between plasma cholesterol and CHD risk might have several explanations. Obviously, factors unrelated to lipid and lipoprotein metabolism could affect the relative risk for CHD among the different countries. On the other hand, a single measurement of plasma cholesterol may not adequately reflect CHD risk, or saturated fats and cholesterol might affect lipoprotein-arterial wall interactions in a way not revealed by estimation of total plasma cholesterol alone.143,144
Treatment of High Plasma Cholesterol and Other Risk Factors

Since atherosclerosis is a multifactorial disease, it can be asked whether the most effective approach to prevention of CHD might be to concentrate on other risk factors (e.g., smoking and hypertension). This is a reasonable question, but evidence of several types supports the concept that a relatively high level of LDL is a crucial factor in atherogenesis; furthermore, epidemiological studies suggest that other risk factors may not contribute greatly to CHD until the concentration of LDL has reached a certain level.17,145 Therefore, if LDL concentrations can be reduced through dietary means, the risk from other factors might be lessened significantly. This is not to say that modification of other risk factors is not important and, from a practical point of view, their elimination could have a profound influence on risk in the U.S. population.

Practicality for U.S. Population

One criticism of the AHA diet has been that it would be poorly accepted by the U.S. public. This criticism may not be valid because the American diet is changing.16 Intakes of cholesterol and possibly saturated fats have decreased; in addition, ingestion of polyunsaturated fats has increased, although not to levels above the limits proposed by the AHA. Many individuals have consciously chosen to alter their diets to one low in saturated fats and cholesterol, and these changes may be partly responsible for the apparent reduction in plasma cholesterol of the American population over the past few years.

The AHA contends that, at the very least, the public should be given the option of choosing foods consonant with its recommended diet. These foods should be readily available in both groceries and restaurants. Labeling of foods is strongly advocated so that the public can more easily choose the desired composition. Many people seemingly desire to alter their eating habits but have insufficient knowledge of dietary principles to accomplish the change. Thus, before dietary recommendations can have a great impact on eating patterns, they must be associated with a program of public education.

Weight Reduction vs Change in Diet Composition

Although the debate continues whether American people should change the composition of their diets, the prudence of maintaining body weight in the desirable range has received almost universal acceptance. Indeed, a reduction in body weight may decrease risk for CHD through (1) reduction of total plasma cholesterol (especially VLDL-cholesterol), (2) an increase in HDL-cholesterol and (3) a reduction in blood pressure. If caloric restriction is carried out by decreasing the intake of saturated fats and cholesterol, LDL-cholesterol also should fall in most people. This reduction of LDL can be achieved only if saturated fats and cholesterol are decreased and will not occur with some reducing diets that stress significant quantities of animal fats at the expense of other constituents; indeed, the latter actually may raise LDL.

Clearly, the AHA recommends weight reduction in obese subjects and the maintenance of desirable weight in general. However, many people whose weights are in the desirable range still have a relatively high LDL, and in these only a change in diet composition (e.g., a decrease specifically in saturated fats and cholesterol) can reduce LDL concentrations toward an acceptable range. For this reason the AHA does not accept the premise that weight control should be the only dietary recommendation for the general public.

Dietary Change in Low-risk Groups

A common criticism of the AHA position is that universal dietary change would lead to "treatment" of many people who are already at low risk for CHD. Several points can be made in response to this criticism. First, the possibility of a curvilinear relation between plasma cholesterol and CHD risk is still hypothetical, and a reduction in plasma cholesterol through dietary modification may further decrease the risk for CHD even in those who already have cholesterol levels in the so-called "ideal range."19 For example, CHD rates apparently are lower in countries with severely restricted intakes of saturated fats and cholesterol than in people of the U.S. population whose plasma cholesterol is in the first quintile (i.e., less than 194 mg/dl). This difference could be due to lower mean levels of cholesterol in these other populations as compared to the first quintile,7 but as indicated above an excess intake of cholesterol and saturated fats may contribute to atherosclerosis through mechanisms not revealed by plasma cholesterol level alone in the fasting patient.

Second, unless eating habits of Americans are widely changed, people in the higher-risk categories probably will not alter their eating patterns. The important point should be made that there is nothing sacred about the current American diet high in fat and total calories. The AHA diet, which emphasizes variety (especially fruits, vegetables, whole grains, legumes and animal protein sources such as chicken, fish, skim milk and egg whites), can certainly be as palatable as one containing large quantities of fat-rich meats, egg yolks and dairy fats. As the U.S. public is becoming more diet-conscious, there is an increasing interest both in health value and eating pleasure that can be derived from diets patterned along Mediterranean and Far Eastern lines. In addition to the valuable AHA cookbook, several others have recently been published which offer many tasty recipes that are in line with the AHA recommendations.

Third, in the absence of universal screening for CHD risk, it is impossible to identify specific individuals who might benefit most from the AHA diet. Therefore, since CHD risk may be reduced to some degree even in those in low-risk categories, the diet can be recommended to all who have not had careful screening for plasma lipids and other risk factors. Finally, cholesterol levels usually rise with age, and even if screening is carried out at an appropriately early age, future cholesterol concentrations cannot be predicted with accuracy. Indeed, adherence to a diet low in cholesterol and saturated fats may actually diminish the increase in plasma cholesterol that occurs commonly with aging.141 There is no evidence that the recommended changes could result in an elevation in plasma cholesterol.

AHA Diet and Individual Variability of Response

Another common criticism of the recommendation for universal dietary alteration is that changes in plasma lipids vary from person to person. In other words, some people appear more diet-responsive than others. Those who do not respond significantly may not derive any
direct benefit from dietary alteration. This is a logical criticism, but the extent to which it holds in man has not been determined. Variability in responsiveness can be marked in some animals fed cholesterol. Some degree of variability almost certainly exists for man as well. However, carefully controlled studies on metabolic wards suggest that variability is less than might be anticipated from animal studies. For instance, responses to dietary cholesterol over a considerable range of cholesterol intakes in normal men are reasonably constant. Further, exchange of polyunsaturated fats for saturated fats produces a fairly uniform alteration in cholesterol concentrations in the metabolic ward. These findings suggest that some of the reported variability in outpatient studies may be due to differences in adherence to diet. Obviously, more investigation is needed to define the extent to which individual responses to diet differ. But even if the degree of change among individuals varies considerably, this does not rule out the validity of recommending generalized change in diet to bring about a net lowering of plasma cholesterol in the whole population.

AHA Diet and Genetic Hyperlipidemias

The question has been raised whether the public health approach of the AHA may not overlook or inadequately treat the large number of people in the U.S. population who have genetic hyperlipidemias. These people are at high risk for CHD and dietary change alone may be inadequate to bring their plasma lipids to desirable levels. This is an important point, and it brings up the issue of how much emphasis should be put on the search for patients with significant risk factors. The AHA has never advocated screening the total population on a universal and recurring basis because of impracticality. On the other hand, the attempt has been made to educate the medical community on the importance of checking plasma lipids in individuals suspected of being at greater risk (i.e., patients who have existing CHD, hypertension, diabetes mellitus, obesity, who are smokers or have a family history of CHD or hyperlipidemia). The question of who should be screened for hyperlipidemia and what conditions is still a subject of debate within the Association, but the need to seek out high-risk patients is clearly recognized. The advantage of identifying patients with hyperlipidemia should be twofold. First, if a patient is found with a distinct elevation in plasma cholesterol, he can be informed of his increased risk and be urged to correct the abnormality. (It is also important to find other family members who may have hypercholesterolemia.) Second, if a person clearly has a low cholesterol he can be reassured not to be inappropriately concerned. The logical screening approach to lipid and lipoprotein levels has the advantage of allowing for individualized modification of any abnormality. However, from a practical standpoint, detection or screening would not be sufficient as the only means of controlling the high prevalence of relatively elevated cholesterol levels in our society. First, the medical community is not organized to carry out universal screening for hypercholesterolemia. Second, and of greater importance, a single determination of plasma cholesterol is not sufficient to define an individual’s status with respect to plasma lipids and lipoproteins; instead, multiple determinations are needed at several different ages to obtain an accurate assessment of risk. Because of biological and methodological variability, many patients would be mislabeled by a single determination into believing that their plasma cholesterol was in a “safe” range when, in fact, a cholesterol level integrated over a long period of time might not be. And third, even if patients with “dangerous” and “safe” levels of cholesterol could be differentiated, no means are currently available to adequately counsel large numbers of patients on proper diet and to provide long-term nutritional guidance. Therefore, the public health approach of diet modification is needed along with case detection. Through the combination of both approaches, maximal modification of abnormal plasma lipids should be achieved.

Polyunsaturated Fats and AHA Diet

A common misconception is that the AHA currently recommends the intake of large quantities of polyunsaturated fats. It is true that the recommendation is to increase the mean intake for the U.S. public from its present 5-6% to an upper limit of 10%. The major reasons for the recommended increase are twofold: (1) polyunsaturated fats have an inherent cholesterol-lowering property and (2) they provide a preferable source of fat calories to cholesterol-containing saturated fats. On the other hand, the AHA has been reluctant to advocate a marked increase in polyunsaturated fats (i.e., to 15-20% of total calories) for several reasons. First, no large population has consumed such large quantities of polyunsaturated fats for many years with demonstrated safety. Second, high-fat diets promote weight gain. Third, polyunsaturated fats may increase the risk for gallstones in some people. Fourth, these fats have been shown to have co-carcinogenic potential in experimental animals. And fifth, they can alter the composition of cell membranes. Thus, it may be prudent for the U.S. public to avoid very large amounts of polyunsaturated fats for the present time.

AHA Diet vs Very Low Fat Diets

Criticism of the AHA diet has also come from those who believe Americans should eat a very low fat diet (i.e., a fat intake of 10-20% of total calories). They cite the low incidence of CHD in populations in whom total fat intake is very low (e.g., Japanese in Japan, rural Guatemalans, Tarahumara Indians, etc.), and they conclude that the AHA diet is too liberal in total fat. However, a careful review of epidemiological studies, particularly the Seven Countries Study, suggests that the crucial atherogenic factors in the diet are saturated fats and cholesterol and not percent total fat. For example, CHD deaths are very low in Greece and adjacent areas despite an intake of fat of 30-35% of calories; in this region, however, monounsaturated fatty acids and not saturated fats make up the bulk of the fat intake. In Crete, for instance, fat intake is 40% of total calories, but saturated fats make up only about 8%, and CHD mortality rates are extremely low. Therefore, the existing data suggest that the AHA diet with recommendations to severely curtail saturated fats and cholesterol should achieve essentially the same results as a very low intake of total fat. It is unlikely that the American public would accept such a marked departure from its usual fat intake, and it probably is not necessary.

AHA Diet, Low Plasma Cholesterol and Cancer

During the past few years several epidemiological...
studies have suggested an inverse relationship between naturally occurring serum cholesterol and the incidence of malignant neoplasms and other causes of death. The first report of this type indicated a significantly lower average level of blood cholesterol among men in England who died of cancer of the colon. Following this report, a significant inverse relationship between serum cholesterol and overall mortality was reported for New Zealand Maoris, a group of Polynesians who have adopted a Western life style. A subsequent study from England of 18,000 men, aged 40-64 years, again noted an increased death rate from cancer and other causes in men with lowest plasma cholesterol; however, this inverse association was limited to the first two years of follow-up; afterward, total mortality and cholesterol levels were correlated evenly and positively. A similar phenomenon of early death in low-cholesterol subjects not confirmed in long-term follow-up has been reported in the Paris Prospective Study of Coronary Heart Disease.

In a study of 3,102 people in Evans County, Georgia, followed for 14 years, new cancer cases had significantly lower mean levels of serum cholesterol at the time of initial sampling than did the noncancer population; this relationship was stronger for men than women and was observed for various sites of cancer and cell types. In a report from Framingham, Massachusetts, 691 cases of cancer were documented in 5,000 subjects followed for 24 years. During this period, 691 cases of cancer were observed and significant associations of different types of cancer were observed with cigarette smoking, alcohol use, education, height-weight and parity. Furthermore, serum cholesterol levels were found to be inversely correlated with the incidence of colon cancer and possibly other sites in men (but not women). In another report of this type from Malmö, Sweden, a follow-up of 10,000 men revealed that deaths occurred in two peaks, one at the higher end of the serum cholesterol distribution but the other at the lower end. A significant portion of deaths in the low-cholesterol range appeared to be related to alcohol (acute intoxication, accidents and alcohol-induced diseases — cirrhosis of the liver, esophageal varices and pancreatitis).

Despite these reports, not all epidemiologic studies have reported a significant inverse correlation between plasma cholesterol levels and cancer. Of particular interest for the U.S. population, the large Pooling Project did not show the low cholesterol-cancer association. Therefore, the available epidemiologic data are tentative at best, and more prospective studies will be required before a site-specific relationship between low cholesterol levels and cancer can be proven.

Furthermore, several investigators have held for some time that high-fat, high-cholesterol, low-fiber diets also may play a role in the etiology of colon cancer and other malignancies. In most industrialized countries in which diets contain large amounts of fat and cholesterol, the incidence of colon cancer is greater than in countries where most people ingest a diet low in cholesterol and fat and high in fiber. Indeed, international comparisons suggest a significant relationship between mortality rates of CHD and colon cancer. Thus, when all the epidemiologic studies are taken into consideration, high intakes of fat and cholesterol do not seem to be protective against cancer; if anything, they may be related to the risk for malignancy. This has led to the suggestion that high intakes of fat and cholesterol may be associated with an increased influx of cholesterol and its conversion products (bile acids) into the large intestine and these steroids, or their secondary bacterial conversion products, may be carcinogenic.

Regardless of the explanation for the apparent association between naturally occurring low plasma cholesterol and cancer, there are no data arguing that changing the diet toward that recommended by the AHA would heighten the risk for cancer. It might be noted that the authors of the clinical trial carried out in the Veterans Administration Domiciliary in Los Angeles reported that the therapeutic diet, which was rich in polyunsaturated fats, was associated with a greater number of cancer deaths than the control diet. The results have been widely quoted, but the statistical limitations have been stressed by many. In a review of the cancer experience from four other clinical trials using similar cholesterol-lowering diets, the conclusion was reached that diets rich in high-polyunsaturated fats did not influence cancer risk. Finally, it should be emphasized again that the AHA does not recommend high-polyunsaturated fat diets similar to those used in the above clinical trials for the general public.

AHA Diet and HDL

Recent reports suggest that cholesterol-lowering diets can cause a decrease in plasma HDL as compared to diets containing large amounts of saturated fats and cholesterol. To date, it has not been tested whether the AHA-recommended diet will alter HDL. However, the major causes of low HDL in our society seem to be obesity and sedentary life-styles. If desirable weight can be achieved along with an appropriate exercise program, these should cause a rise in the HDL concentration. This rise in HDL should offset any tendency for a lowering of HDL by reducing saturated fats and cholesterol or increasing polyunsaturates.

The role of diet on the metabolism of HDL is poorly understood, but it is a subject of much current interest. Several subspecies of HDL have been identified, and the impact and clinical significance of diet on each remains to be determined. The preliminary observations that feeding of saturated fats and cholesterol may raise HDL, which appears from epidemiologic studies to be associated with a lesser risk of CHD, is a paradox that needs to be resolved. Nevertheless, it seems likely that any lowering of HDL that might result from cholesterol-lowering diets would be offset by a similar reduction of LDL concentrations; this is in accord with the observations discussed above that populations with low LDL concentrations and decreased rates of CHD frequently have low levels of HDL as well.

AHA Diet for Children

The current AHA statement is consistent with the concept that dietary modifications should begin in childhood, but it does not elaborate. For this reason, a more specific statement is under consideration by the Nutrition Committee. More attention is now being focused on identifying risk factors for coronary heart disease in children so that preventive measures can be introduced at a stage when they are most likely to influence the underlying pathologic process. Although it is not known whether dietary modification in children will alter the incidence of CHD in later life, evidence supporting the benefit of lowering plasma cholesterol levels is strong. A major associated benefit is the possibility that dietary habits
learned in childhood may persist into adult life. The re-
duction of total dietary energy in the obese childhood
population is also an advantage. The recommended AHA
diet does not pose any danger of poor nutrition for chil-
dren. It is not a radical diet and is consistent with a good
nutritional status.

AHA Diet and Sodium

The current diet statement also contains the recom-
mandation that dietary sodium should be reduced. The
reason for decreasing sodium intake obviously relates to
the problem of hypertension, a known risk factor for
CHD. A reduction in dietary sodium might accomplish
either or both of two aims: (1) prevention of hypertension
in susceptible individuals or (2) reduction in the average
level of blood pressure in the general population.

Obviously, the relatively high consumption of salt by the
U.S. public does not cause hypertension in the majority
of people, and it can be asked whether a universal and
marked change in sodium intake is needed for the pre-
vention of overt hypertension in the 10-15% of the
population who are salt-sensitive. On the other hand, the
relation between hypertension and CHD risk seems to be
continuous over a wide spread of blood pressures, even
down into the "normal" ranges. If the average level of
blood pressure in the general population could be re-
duced by a universal decrease in salt intake, the overall
risk for CHD in Americans should be reduced. How much
decrease in dietary sodium is needed to produce a sig-
ificant lowering of mean blood pressure, unfortunately, is
not known; thus, a precise limit of sodium intake has not
been recommended. The Nutrition Committee recently
has been considering a specific proposal about sodium,
but it recognizes that the question of whether an effort
should be made to reduce the intake of sodium for the
general public is an unsettled matter.

IV. Strategy of AHA Position on
Plasma Lipid Modification

Based on the above considerations, a reasonable
approach to modification of high plasma lipids would be
two-fold: to recommend a generalized diet change for
healthy Americans and to seek out and treat individual
patients with unusually high levels of lipids or other risk
factors. For the first time, the AHA has taken the position
that a diet recommendation for the healthy U.S. popula-
tion is warranted. It should be emphasized that the rec-
ommended diet is not radical, and there is little likelihood
it would be nutritionally inadequate or harmful. There is
almost uniform agreement that caloric restriction to
achieve desirable weight is beneficial. There are no good
reasons related to health why Americans should not con-
sume a diet containing less total fat, saturated fats and
cholesterol. Since no harm can be visualized, and be-
cause it may lessen the risk for CHD, a change to such a
diet would seem prudent.

The second strategy is to find and treat patients with
known risk factors and to recommend modification of
these factors. This strategy has followed two lines: (1) to
educate the public about risk factors and to urge them to
be checked by their physicians and (2) to educate physi-
cians on the importance of screening for risk factors. The
checking for risk factors has two advantages. First, it en-
ables the physician to discover patients at high risk who
may be treated. For patients with genetic hyperlipidemia,
the AHA diet for healthy Americans may not be sufficient
to bring plasma lipids into the desirable range. Such pa-
tients may have to be treated with drugs or by more strin-
gent dietary therapy. The AHA will soon publish a revised
program for dietary therapy of hyperlipidemia. This pro-
gram will make use of the diet recommended for the
general public as the first phase of this therapy, but in
patients with significant hyperlipidemia, second and third
phases will progress to more and more restrictions of fat
and cholesterol intakes.

A second reason for measuring plasma lipids is the
possible identification of individuals with low lipid levels. If
these persons are of normal weight and have no other
risk factors, they can be reassured that a significant
change in lifestyle probably is not necessary. Neverthe-
less, before giving this reassurance, the physician should
cautions the patient that cholesterol levels tend to rise with
age, and future checks are advisable. There is also the
possibility that the link between diet and CHD may not be
reflected entirely by plasma lipid concentrations, particu-
larly at a single point in time. The data shown in table 1
suggest that total emphasis should not be put on mea-
sured plasma cholesterol.

To date, the AHA has not recommended or supported
widespread screening of the general population for
hyperlipidemia. This would be both costly and inefficient,
particularly because of a lack of a uniform mechanism for
treating the multitude of patients who would be detected.
Perhaps at a future time when the relationships between
plasma lipoproteins and atherosclerosis are better under-
stood, consideration may be given to how best to seek
individual cases for specific therapy. In the meantime,
however, the AHA has taken the position that education
of the general public about hygienic measures to reduce
CHD risk, and of physicians about detection and mod-
ification of risk factors, is the most appropriate approach
to the prevention of CHD.

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