REVIEWS

Resolving the Coronary Artery Disease Epidemic Through Plant-Based Nutrition

Caldwell B. Esselstyn, Jr., MD

The world's advanced countries have easy access to plentiful high-fat food; ironically, it is this rich diet that produces atherosclerosis. In the world's poorer nations, many people subsist on a primarily plant-based diet, which is far healthier, especially in terms of heart disease. To treat coronary heart disease, a century of scientific investigation has produced a device-driven, risk factor-oriented strategy. Nevertheless, many patients treated with this approach experience progressive disability and death. This strategy is a rear-guard defensive one. In contrast, compelling data from nutritional studies, population surveys, and interventional studies support the effectiveness of a plant-based diet and aggressive lipid lowering to arrest, prevent, and selectively reverse heart disease. In essence, this is an offensive strategy. The single biggest step toward adopting this strategy would be to have United States dietary guidelines support a plant-based diet. An expert committee purged of industrial and political influence is required to assure that science is the basis for dietary recommendations. (Prev Cardiol. 2001;4:171–177)

I have drawn two compelling observations from my service as the program director of two national cholesterol conferences and my participation in three others over the past decade. First, a great deal is known about what factors are responsible for causing coronary artery disease (CAD) and what populations are vulnerable. Second, the present emphasis on identifying risk factors and those who are particularly vulnerable to atherosclerotic disease will not resolve the cardiovascular epidemic, which currently threatens one of every two Americans and is predicted to become the number one global disease burden by the year 2020.[1]

From the Cleveland Clinic Foundation, Cleveland, OH
Address for correspondence/reprint requests: Caldwell B. Esselstyn, Jr., MD, The Cleveland Clinic Foundation, 9500 Euclid Avenue, Desk A80, Cleveland, OH 44195
Manuscript received August 22, 2000; accepted September 28, 2001

Autopsy data from the conflicts in Korea[2] and Vietnam,[3] the Bogalusa study,[4] and the Pathological Determinants of Atherosclerosis in Youth (PDAY)[5] study all testify to the ubiquitous nature of the disease in young Americans. Recently, intra-arterial ultrasonography confirmed that "normal" segments in patients with CAD also have diffuse symmetric atherosclerosis, which is not yet disfiguring the intraluminal diameter and thus is invisible on angiography.[6] This work is further confirmation of the Roberts autopsy data,[7] which demonstrate that essentially all patients with ischemic heart disease have triple-vessel involvement.

However, CAD is virtually absent in cultures that eat plant-based diets, such as the Tarahumara Indians of northern Mexico,[8] the Papua highlanders of New Guinea,[9] and the inhabitants of rural China[10] and central Africa.[11] Hundreds of thousands of rural Chinese live for years without a single documented myocardial infarction.[12]

Modern North Americans and Europeans pride themselves on having the world's most advanced medical care. What are these health care systems doing about CAD?

PRESENT HEART DISEASE MANAGEMENT STRATEGIES

The present strategy focuses on interventional procedures and risk factor modification. This approach is strictly defensive. It is pressing the limit of what society can afford. Our present cardiology budget exceeds one quarter trillion dollars per year.[1] Millions of symptomatic patients—generally, those with arterial stenosis of more than 70%—have had interventions such as bypass, angioplasty, stenting, or atherectomy.[13] Unfortunately, these interventions are accompanied by significant morbidity, mortality, and expense, provide only temporary benefit, and do nothing for patients at greatest risk for myocardial infarction: those with juvenile plaques of 30%–50% stenosis, which are the ones most prone to rupture.[14] As Forrester states, "Angiography does not identify and interventional strategies don't treat those lesions most likely to cause a heart attack."[15]

Therapies involving diet and lipid-lowering medication are not ignored by our health care leaders, but sadly, their recommendations are clearly inadequate. The American Heart Association and the National Cholesterol Education Program (NCEP) recommend consumption of not more than 30% dietary fat and cholesterol levels below 200 mg/dL; numerous studies confirm that people who adhere to these recommendations experience not arrest and reversal of their heart disease, but rather continued disease progression.[16] A question arises whether these recommendations expose millions to disease development and progression. However, because of the general respect commanded by these organizations, many doctors and patients believe that following their recommendations will protect against heart disease.

The newer NCEP clinical guidelines, known as the Adult Treatment Panel III, suggest broadening the identification of those at risk. This will mandate that millions of Americans take cholesterol-reducing drugs as well as make some dietary and physical activity adjustments. This is a rear-guard, after-the-fact approach. It tacitly acknowledges that our food environment is so toxic that millions will become at risk and develop disease. As will be discussed, it is preferable to advise the public to avoid the categories of food that cause atherosclerotic disease.[17]

The National Research Council, in its 1989 report *Diet and Health*,[18] recommended an upper limit of total cholesterol of 200 mg/dL and 30% dietary fat, even though "…a number of the scientists felt that a greater reduction would confer additional health benefits."[18] However, the committee felt that setting the cut-off too low would merely frustrate the public. The council also surmised, incorrectly, that if the upper level were set at 200 mg/dL, most Americans would achieve a total cholesterol level of 150 mg/dL or less.[18] That has not happened. Most Americans and their physicians feel "safe" with a total cholesterol level of up to 200 mg/dL. They are not safe. In the Framingham study,[19] 35% of ischemic heart disease occurred in patients with total cholesterol levels between 150 and 200 mg/dL. In the Cholesterol and Recurrent Events (CARE) study,[20] the average total cholesterol level in patients with a history of heart attack was 209 mg/dL. In contrast, the American Cancer Society recommends no more than 20% dietary fat,[21] while the World Health Organization prefers no more than 15%.[22]

Dr. Scott Grundy, chairman of the NCEP, proclaimed approximately 14 years ago[23] that 90% of heart attacks could be prevented if the population's cholesterol were 150 mg/dL or less—a figure identical to that hoped for by the National Research Council in 1989. However, neither the National Research Council, the American Heart Association, nor the NCEP is on record to show precisely what diet will achieve the cholesterol goal of 150 mg/dL. The basic diet favored by these groups contains not only grains, legumes, vegetables, and fruit, but also oil, low-fat milk and milk products, butter, cheese, poultry, lean meat, and fish. I am unaware of any research proving that by eating such a diet one can achieve a cholesterol level of 150 mg/dL or avoid CAD.

The so-called Mediterranean diet and monounsaturated oils have become unjustifiably popular because of the Lyon Diet Heart Study.[24] This approach is difficult to accept. No studies of monounsaturated oils have shown them to arrest and reverse coronary disease. The Lyon study did show a slower rate of progression, but this is hardly an acceptable goal. In a study of patients with coronary disease, Blankenhorn et al.[25] actually showed the reverse: disease progressed as rapidly in patients on a monounsaturated diet as it did in those on a saturated-fat diet. Rudel and coworkers[26] demonstrated a similar result in African green monkeys over a 5-year period. Particularly compelling was his finding that disease in the two groups was equivalent, even though the monounsaturated group had higher levels of high-density lipoprotein (HDL), lower levels of low-density lipoprotein (LDL), and a more favorable LDL-to-HDL ratio. He recently replicated the results in rodents.[27]

The number of heart attacks continues to increase every year.[28] Although the age-adjusted death rate for heart disease has declined, the decline may be artifactual.[12] Stamler et al.[29] found that deaths from cardiovascular disease approached 40% of all deaths in a group of 80,000 young men, with follow-up ranging from 16–34 years. The data confirmed a continuous, graded relationship of serum cholesterol level to long-term risk of coronary heart disease, cardiovascular disease, and all-cause mortality. There was also substantial absolute risk and increased excess risk of coronary heart disease and cardiovascular disease death for younger men with elevated cholesterol levels and, conversely, a longer estimated life expectancy for younger men with favorable lipids. Our stop-gap, device-driven, risk factor-oriented approach is not working. Why? Because it fails to address our toxic food environment, which is responsible for the disease. It is focused only on those who are already ill or whose elevated lipids reflect an inability to detoxify their American diet. What are the other alternatives?

TAKING THE OFFENSIVE

As I have reported earlier,[30,31] a plant-based diet in conjunction with cholesterol-reducing medication eliminated progression of CAD over a 12-year period in patients with triple-vessel CAD. Most of the 18 patients had experienced failure of an earlier intervention of bypass surgery or angioplasty. All patients who maintained the diet achieved the cholesterol goal of less than 150 mg/dL and had no recurrent coronary events during the 12 years. At 5 years, angiography was repeated in most cases. By analysis of the stenosis percentage, none had progression of disease and 70% had selective re-

gression.[30] These data are compelling when one considers that the same group had experienced more than 49 coronary events during the 8 years before this study.[30]

A recent case is particularly telling. During September and October of 1996, a 44-year-old physician experienced occasional chest discomfort, yet neither electrocardiography, stress echocardiography, nor thallium scanning found evidence of disease. While eating the typical American diet, he had a total cholesterol of 156 mg/dL and an LDL of 97 mg/dL. He was lean, nondiabetic, and normotensive; he did not smoke and had no family history of coronary disease. His lipoprotein(a) and homocysteine levels were normal. On November 18, 1996, after his surgical duties, he became acutely ill with pain in the left arm, jaw, and chest. Immediate coronary catheterization found all vessels to be normal except for the left anterior descending artery, the distal third of which was diseased. Enzyme tests confirmed a myocardial infarction. However, no intervention was deemed appropriate.

This patient was aware of my ongoing study and was curious for more information. He and his wife consulted me for an in-depth review of the plant-based diet and techniques of this coronary disease arrest and reversal study. He became the personification of commitment to the plant-based diet. Over the next 32 months, without cholesterol-lowering drugs, he maintained a mean total cholesterol of 89 mg/dL and an LDL of 38 mg/dL. The repeat angiogram 32 months after his infarction showed that the disease was completely reversed (Figure 1).

Even though many people might find a plant-based diet initially difficult to follow, every patient with the diagnosis of CAD should at least be offered the option of this potentially curative arrest and reversal approach. As this young physician's case illustrates, our plant-based diet approach can achieve total disease arrest and selective regression even in advanced cases. This approach is particularly compelling because patients can take control over the disease that was destroying them. If traditional interventional cardiology is a rear-guard action, our arrest and reversal therapy can be likened to a military offensive against atherosclerosis.

Limitations of this study are its modest number of participants and lack of comparable controls. Nevertheless, its size permitted the caregiver an opportunity for frequent patient encounters. These interactions enabled 75% of participants to achieve profound lipid reduction, dietary goals, and relief of symptoms, which continued to improve throughout the study's 12-year duration. Patients essentially served as their own controls, often achieving dramatic angiographic reversal of disease, as reviewed in the angiographic core laboratory (Figures 1–4).

NEW RECOMMENDATIONS FOR A HEALTHY DIET

The expert faculty at the First National Conference on the Elimination and Prevention of Coronary Artery Disease has issued a new set of recommendations[32]:

1. Present nutritional guidelines of government and national health organizations do not provide a maximal opportunity either to arrest or to prevent coronary artery disease.
2. The optimal diet consists of grains, legumes, vegetables, and fruit, with <10%–15% of its calories coming from fat. This diet minimizes the likelihood of stroke, obesity, hypertension, type 2 diabetes, and cancers of the breast, prostate, colon, rectum, uterus, and ovary. There are no known adverse effects of such a diet when mineral and vitamin contents are adequate.
3. Children and adolescents require major attention to develop early habits of optimal nutrition. Schools should assume a significant leadership role in achieving this goal.
4. Speculation about the degree of public compliance with a low-fat diet must not alter the accuracy of the recommendations.

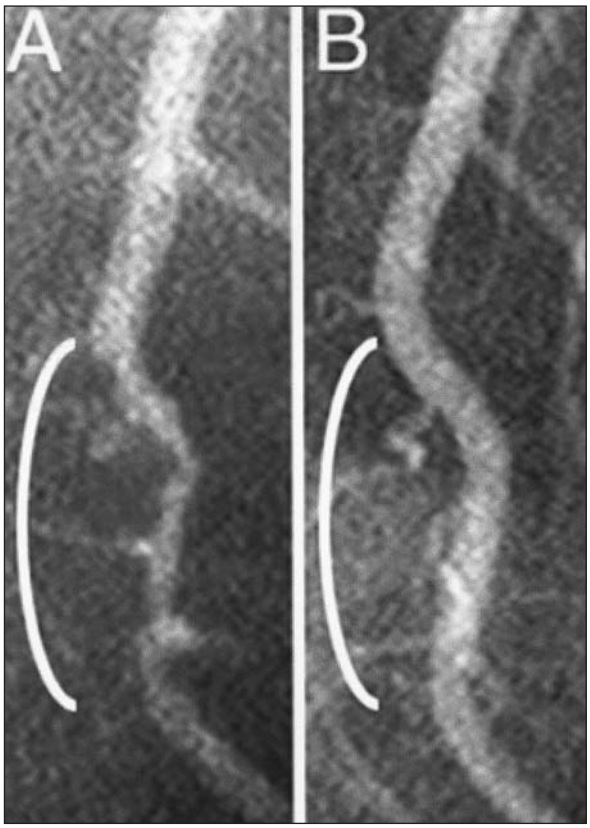

Figure 1. Coronary angiograms of the distal left anterior descending artery before (left) and after (right) 32 months of a plant-based diet without cholesterol-lowering medication, showing profound improvement

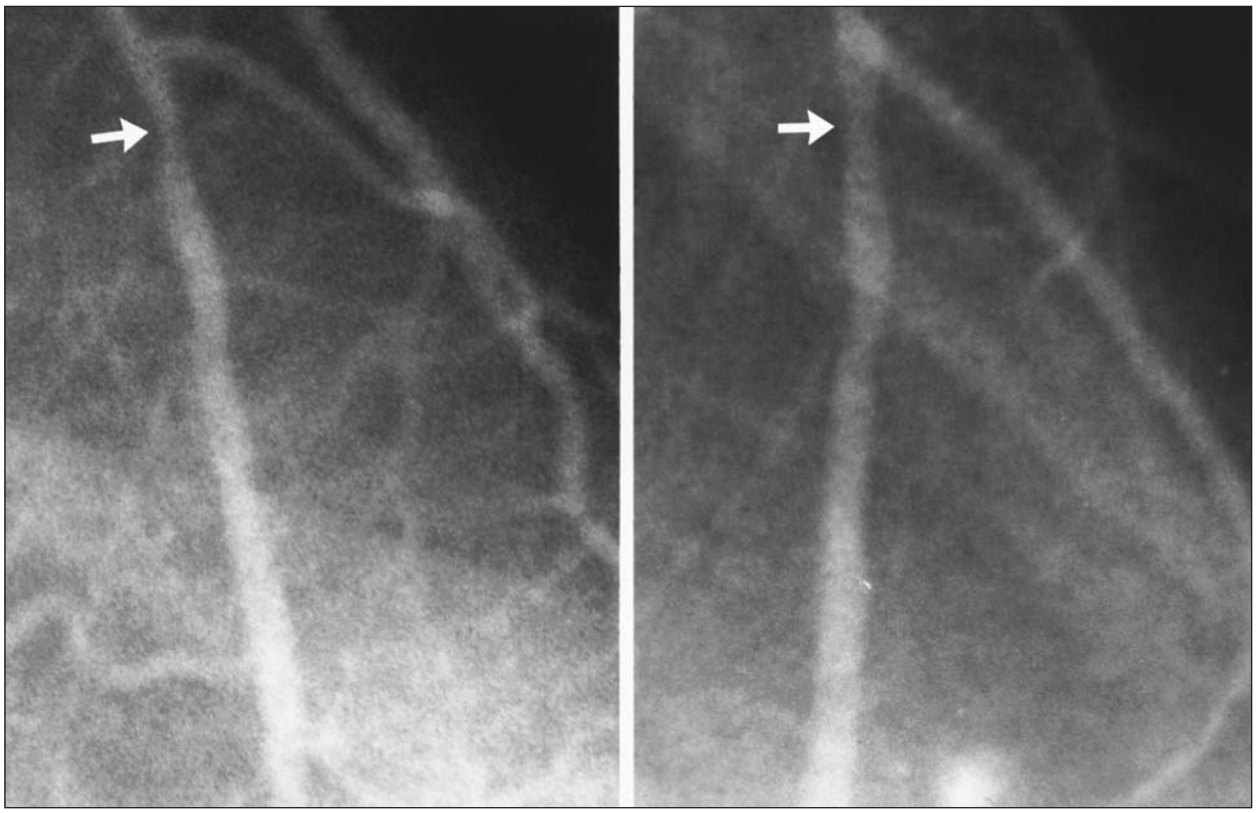

Figure 2. Coronary angiograms of the proximal left anterior descending artery before (left) and showing 10% improvement (right) following approximately 60 months of a plant-based diet with cholesterol-lowering medication

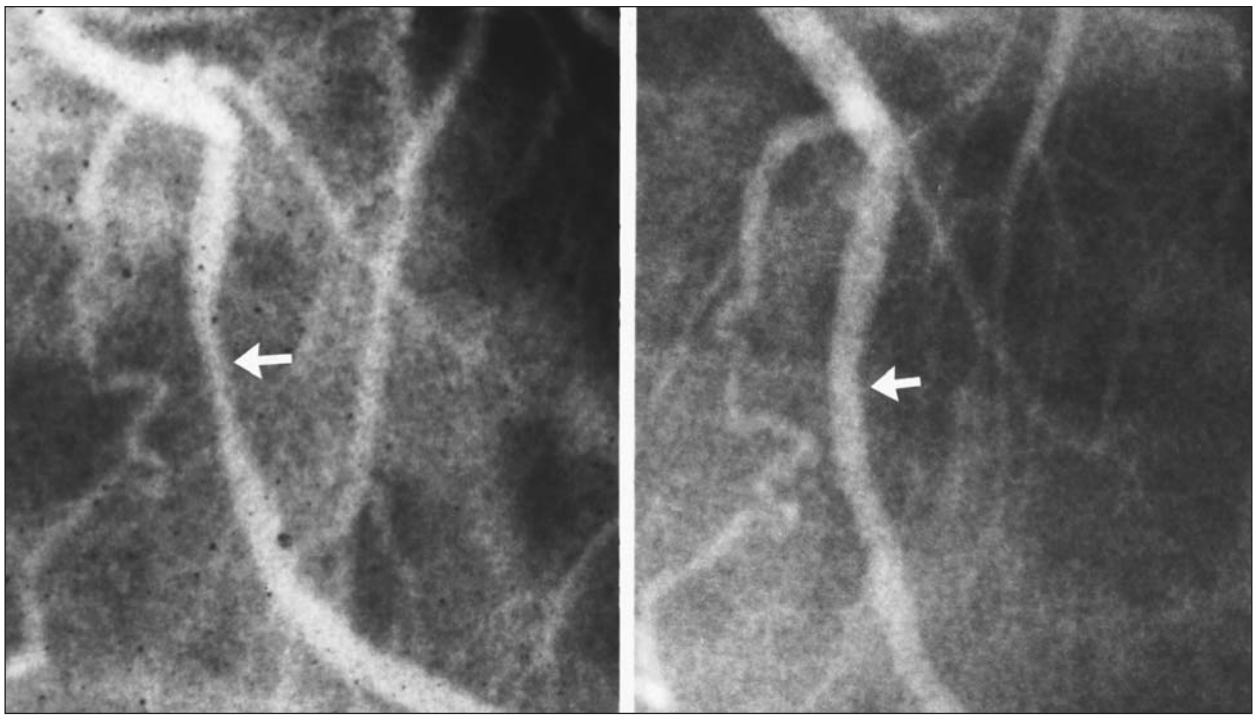

Figure 3. Coronary angiograms of the circumflex artery before (left) and showing 20% improvement (right) following approximately 60 months of a plant-based diet with cholesterol-lowering medication

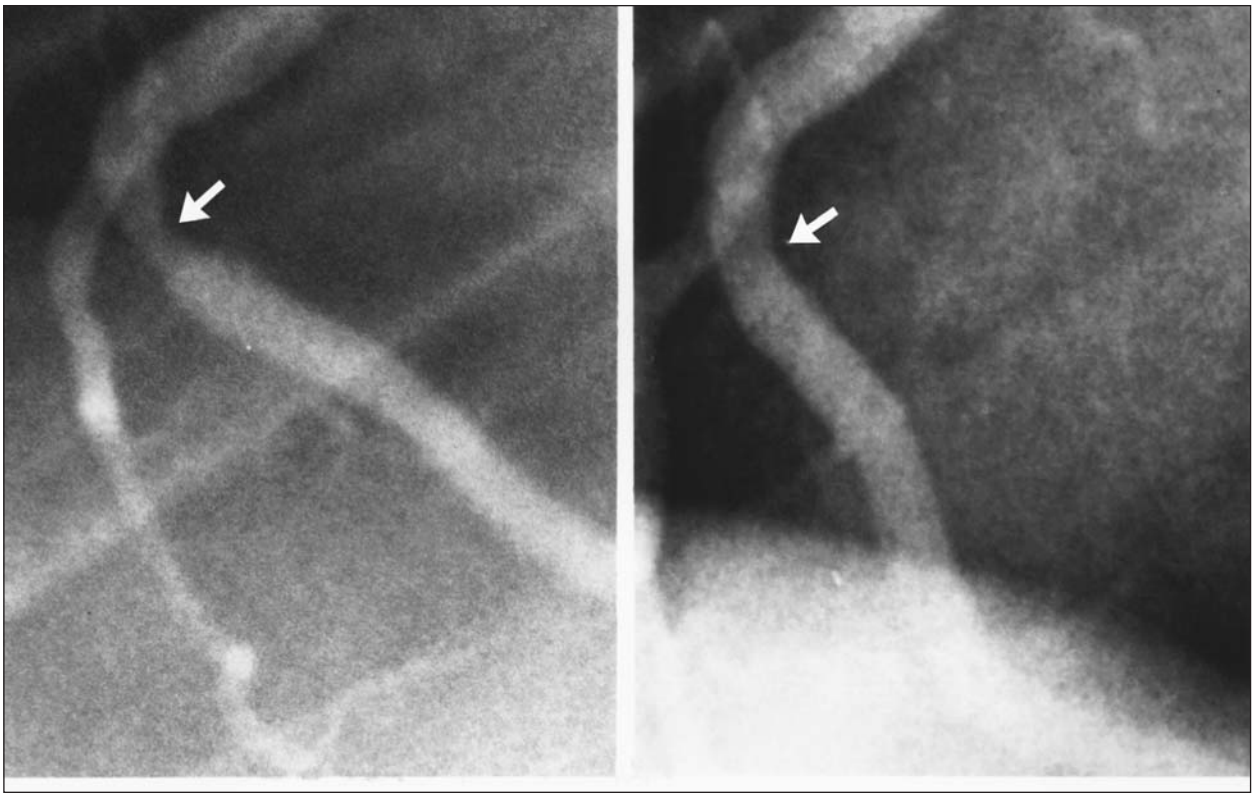

Figure 4. Coronary angiograms of right coronary artery before (left) and showing 30% improvement (right) following approximately 60 months of a plant-based diet and cholesterol-lowering medication.
Reproduced with permission from J Fam Pract. *1995;41(6):560–568.*[30]

At the 1999 National Cholesterol Summit Meeting, Dr. William Castelli was asked what he would do to reverse the CAD epidemic if he were omnipotent. His answer: "Have the public eat the diet of the rural Chinese as described by Dr. T. Colin Campbell," an author of the Cornell China study[33] (William Castelli, MD, personal communication, September 2–3, 1999). A recent prospective study of diet quality and mortality in more than 40,000 women confirms the benefits of consuming a diet high in fruits, vegetables, and grains.[34] Women consuming the greatest level of recommended foods had a 30% lower risk of mortality than those at the lowest level during 5.6 years of observation.

At the year 2000 national meeting on hypertension, the original DASH diet study[35] was updated.[36] It was found that a diet emphasizing grains, vegetables, and fruit (and including low-fat dairy products and lean meat), with particular attention to reducing sodium intake, resulted in blood pressure reductions equivalent to those produced by antihypertensive drugs.[36]

In addition, Dr. Dean Ornish and colleagues[16] have reported both 1- and 5-year data that support a plant-based approach to controlling CAD.

REPLACE THE "FOOD PYRAMID"

An integral part of this offensive must be to eliminate the toxic food environment. Consider the so-called Food Guide Pyramid, the familiar geometric symbol used to promote the recommendations of the US Department of Agriculture (USDA) and the Department of Health and Human Services. It is laden with dairy products, animal products, and oils, which are the essential building blocks of CAD. In addition, from a design standpoint, the choice of a pyramid is potentially confusing and misleading. Some viewers may be led to believe that the foods at the top (meats, sweets, and fatty foods) are the most helpful, when in fact they are the most harmful. To avoid such sources of confusion, we should eliminate geometric figures and promote three simple food categories: safe, condiments, and unsafe.

- ***Safe:*** *grains, legumes, lentils, vegetables, and fruits*
- ***Condiments:*** *nuts and seeds*
- ***Unsafe:*** *oils, dairy foods, meat, poultry, and fish (not regulated by inspection, and frequently contain unacceptable levels of PCBs, dioxin, and mercury)*

In addition, we should recommend dietary supplementation with a daily multivitamin and, for those over 50 years old, an additional 1000–1200 mg of calcium and 600–800 IU of vitamin D. These recommendations are in concert with those of the

expert faculty from the First National Conference on the Elimination of Coronary Artery Disease.[32]

WHY ARE THE CURRENT RECOMMENDATIONS SO WEAK?

When dietary recommendations are issued with the stamp of approval of the US government, the public should be able to trust that these recommendations accurately guide them to foods that are unlikely to cause disease and away from those that are known to cause harm. Thus, any group promoting dietary guidelines for the public should base its decisions on science. However, the USDA has been subjected to intensive industry lobbying, which compromises its capacity to be fair and objective.[37] At the least, neither the experts who testify before the committee nor the committee members themselves should have relationships, financial or otherwise, to the food industry. These same rules regarding conflict of interest should apply to scientists who lead or are members of the NCEP and the Food and Nutrition Section of the American Heart Association.

As recently as October, 2000, the Physicians Committee for Responsible Medicine successfully litigated the USDA to ascertain the compensation sources of the US Dietary Guidelines Committee. Six of the 11 committee members, including the chairman, had relationships with the meat, dairy, or egg industry (Neal Barnard, MD, Chairman of the Physcian's Commitee for Responsible Medicine, personal communication, December 21, 2000). Such conflict insures a perception that the American public and school children will not receive an unbiased recommendation of what constitutes the healthiest food choices. The USDA, by definition a protector of the agriculture industry, should disqualify itself from this responsibility, which more correctly may belong to the Centers for Disease Control and Prevention.

SUMMARY

The present device-driven, risk factor-identification, rear-guard strategy diagnoses disease after the fact and offers no promise of preventing disease or controlling its progression. We are fortunate to possess the knowledge of how to prevent, arrest, and selectively reverse this disease. However, we are not fortunate in the capacity of our institutions to share this information with the public. The collective conscience and will of our profession is being tested as never before. Ties to industry and politics result in conflict within our private and governmental health institutions, compromising the accuracy of their public message. This is in total violation of the moral imperative of our profession. Now is the time for us to have the courage for legendary work. Science—not the messenger—must dictate the recommendations.

REFERENCES

1 Braunwald E. Shattuck Lecture: cardiovascular medicine at the turn of the millennium: triumphs, concerns and opportunities. *N Engl J Med.* 1997;337:1360–1369.
2 Enos WF, Holmes RH, Beyer J. Coronary disease among United States soldiers killed in action in Korea. *JAMA.* 1953;152:1090–1093.
3 McNamara JJ, Molot MA, Stremple JF, et al. Coronary artery disease in combat casualties in Vietnam. *JAMA.* 1971;216:1185–1187.
4 Berenson G, Srinivasan S, Bau W, et al. Association between multiple cardiovascular risk factors and atherosclerosis in children and young adults. *N Engl J Med.* 1998;338:1650–1656.
5 Strong JP, Malcolm GT, McMahan C, et al. Prevalence and extent of atherosclerosis in adolescents and young adults. *JAMA.* 1999;281:727–735.
6 Ziada KM, Kapadia SR, Tuzcu EM, et al. The current status of intravascular ultrasound imaging. *Curr Probl Cardiol.* 1999;24(9):541–566.
7 Roberts WC. Coronary atherosclerosis: is the process focal or diffuse among patients with symptomatic or fatal myocardial ischemia. *Am J Cardiol.* 1998;82(10B):41T–44T.
8 Connor WE, Cerqueira MT, Connor RW, et al. The plasma lipids, lipoproteins, and diet of the Tarahumara Indians of Mexico. *Am J Clin Nutr.* 1978;31:1131–1142.
9 Sinnett PF, Whyte HM. Epidemiological studies in a total highland population, Tukisenta, New Guinea. Cardiovascular disease and relevant clinical, electrocardiographic, radiological and biochemical findings. *J Chron Dis.* 1973;26:265.
10 Campbell TC, Parpia B, Chen J. Diet, lifestyle, and the etiology of coronary artery disease: the Cornell China Study. *Am J Cardiol.* 1998;82(10B):18T–21T.
11 Miller K. Lipid values in Kalahari Bushman. *Arch Intern Med.* 1968;121:414.
12 Breslow JL. Cardiovascular disease myths and facts. *Cleve Clin J Med.* 1998;65(6):286–287.
13 The Bypass Angioplasty Revascularization Investigation (BARI) Investigators. Comparison of coronary bypass surgery with angioplasty in patients with multivessel disease. *N Engl J Med.* 1996;335:217–225.
14 Ambrose JA, Fuster V. Can we predict future coronary events in patients with stable coronary artery disease? *JAMA.* 1997;277:343–344.
15 Forrester JS, Shah PK. Lipid lowering versus revascularization—an idea whose time for testing has come. *Circulation.* 1997;96:1360–1362.
16 Ornish D, Scherwitz LW, Billings JH, et al. Intensive lifestyle changes for reversal of coronary heart disease. *JAMA.* 1998;280:2001–2007.
17 Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). Executive Summary of The Third Report of The National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, And Treatment of High Blood Cholesterol In Adults (Adult Treatment Panel III). *JAMA.* 2001;285:2486–2497.
18 National Research Council (US) Committee on Diet & Health. *Diet and health: implications for reducing chronic disease risk.* Committee on Diet and Health, Food and Nutrition Board, Commission on Life Sciences; National Research Council. Washington, DC: National Academy Press; 1989.
19 Castelli W. Take this letter to your doctor. *Prevention.* 1996;48:61–64.
20 Sacks FM, Pfeffer MA, Moye LA, et al. The effect of pravastatin on coronary events after MI in patients with average cholesterol levels. *N Engl J Med.* 1996;335:1001–1009.
21 The American Cancer Society 1996 Advisory Committee on Diet, Nutrition, and Cancer Prevention. Guidelines on diet, nutrition, and cancer prevention: reducing the risk of cancer with healthy food choices and physical activity. *CA Cancer J Clin.* 1996;46:325–341.

22 WHO study group in diet, nutrition, and prevention of non-communicable diseases. *Diet, nutrition and the prevention of chronic diseases: report of a WHO study group*. WHO Technical Report Series. Geneva, Switzerland; 1990:797.

23 Elias M. Doctors get guidelines for healthy heart. *USA Today*. June 9, 1987;Final Edition-News:01A.

24 de Lorgeril M, Salen P, Martin JL, et al. Mediterranean diet, traditional risk factors, and the rate of cardiovascular complications after myocardial infarction. *Circulation*. 1999;99:779–785.

25 Blankenhorn DH, Johnson RL, Mack WJ, et al. The influence of diet on the appearance of new lesions in human coronary arteries. *JAMA*. 1990;263(12):1646–1652.

26 Rudel LL, Parks JS, Sawyer JK. Compared with dietary monounsaturated and saturated fat, polyunsaturated fat protects African Green Monkeys from coronary artery atherosclerosis. *Arterioscler Thromb Vasc Biol*. 1995;15:2101–2110.

27 Rudel LL, Kelley K, Sawyer JK, et al. Dietary monounsaturated fatty acids promote aortic atherosclerosis in LDL receptor-Null, human ApoB100-overexpressing transgenic mice. *Arterioscler Thromb Vasc Biol*. 1998;18(11):1818–1827.

28 Rosamond WD, Chambless LE, Folsom AR, et al. Trends in the incidence of myocardial infarction and in mortality due to coronary heart disease, 1987 to 1994. *N Engl J Med*. 1998; 339(13):861–867.

29 Stamler J, Daviglus ML, Garside DB, et al. Relationship of baseline serum cholesterol levels in 3 large cohorts of younger men to long-term coronary, cardiovascular, and all-cause mortality and to longevity. *JAMA*. 2000;284(3):311–318.

30 Esselstyn CB Jr, Ellis SG, Medendorp SV, et al. A strategy to arrest and reverse coronary artery disease: a 5-year longitudinal study of a single physician's practice. *J Fam Pract*. 1995;41:560–568.

31 Esselstyn CB Jr. Updating a 12-year experience with arrest and reversal therapy for coronary heart disease (an overdue requiem for palliative cardiology). *Am J Cardiol*. 1999;84:339–341.

32 Esselstyn CB Jr. Foreword: changing the treatment paradigm for coronary artery disease. *Am J Cardiol*. 1998; 82(10B):2T–4T.

33 Chen J, Campbell TC, Li J, et al. *Diet, Life-style and Mortality in China. A Study of the Characteristics of 65 Chinese Counties*. Oxford, UK: Oxford University Press; Ithaca, NY: Cornell University Press; Beijing, PRC: People's Medical Publishing House; 1991.

34 Kant AK, Schatzkin A, Graubard BI, et al. A prospective study of diet quality and mortality in women. *JAMA*. 2000;283(16):2109–2115.

35 Appel LJ, Moore TJ, Orbarzanek E, et al. A clinical trial of the effects of dietary patterns on blood pressure. *N Engl J Med*. 1997;336:1117–1124.

36 Sacks FM, and the DASH-Sodium Collaborative Research Group. Effects on blood pressure of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) diet. *N Engl J Med*. 2001;344(1):3–10.

37 Radetsky P. The live longer diet. Why doesn't the government want you to eat it? *Longevity Magazine*. 1994; May:40.