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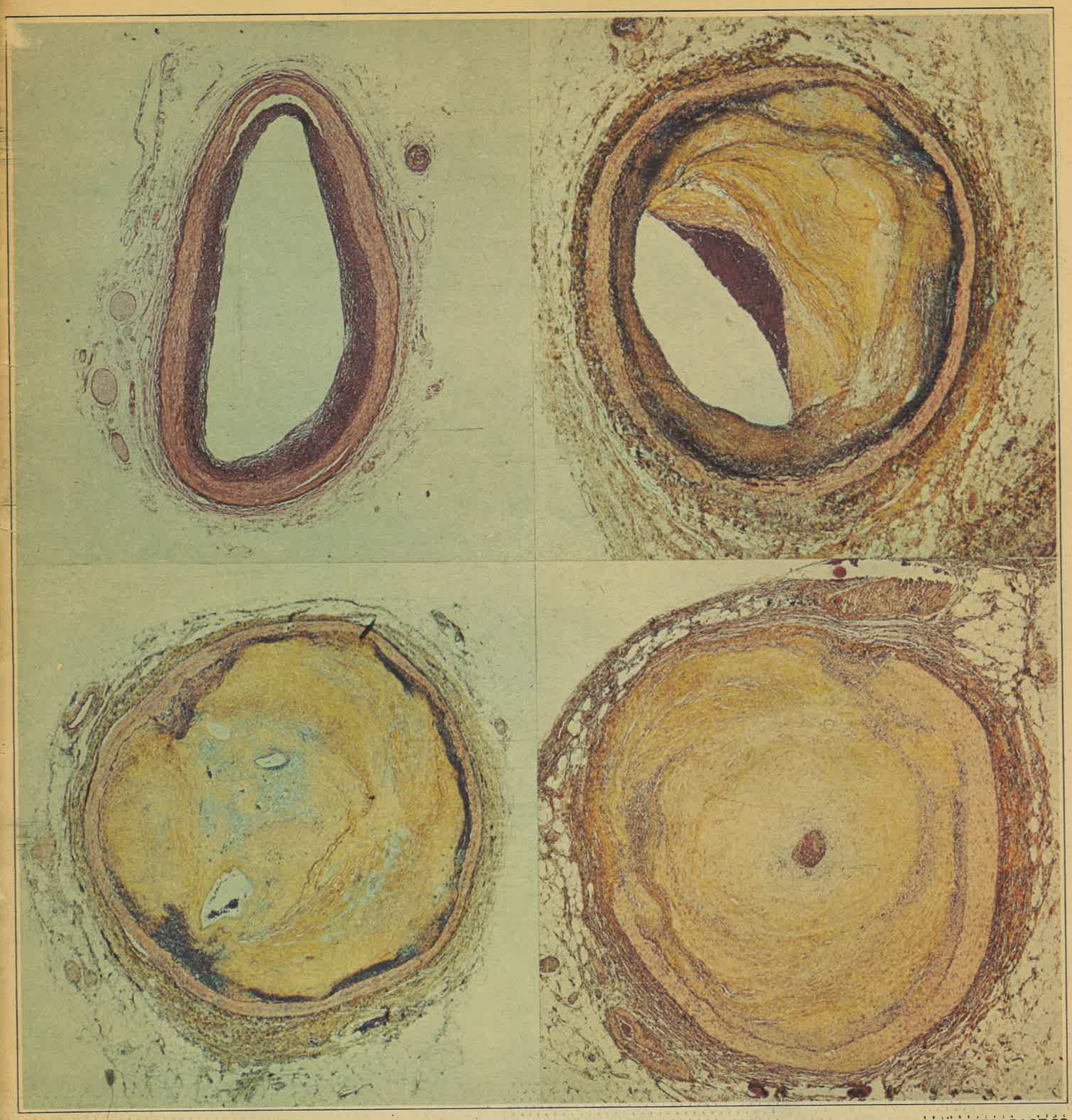

Coronary artery: Closing in on life

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The real villain in heart disease

By C. P. Gilmore

The first thing you notice in Dr. Campbell Moses's office is that picture on the wall. It shows photographs of eight tubes, like sections of white rubber hose, cut open and stretched out so you can see the inside. The one on the left has a clear, smooth inner surface. The second contains a few barely discernible streaks. In the third, you see a sprinkling of tiny, inconspicuous white lumps, looking like so many seed pearls set into the lining. Each successive tube has more and larger growths; by number five they begin to take on a pinkish tinge and the terrain is growing progressively rougher. Number eight looks like a crater-strewn landscape, with angry red protrusions covering much of the surface.

C. P. Gilmore, executive editor of Popular Science, has received several awards for articles on heart research published in The Times Magazine.

The tubes are sections of human aorta — the large artery that curves out of the top of the heart and plunges down through the chest and abdomen, supplying blood to most of the body. The specimens, which were removed in autopsies, progress from the completely healthy one at left to the one almost consumed by atherosclerosis—hardening of the arteries—at right. Dr. Moses is medical director of the American Heart Association, and the picture in his office dramatizes the stealthy action of the nation's No. I killer. The full story, however, is in the statistics:

- Cardiovascular disease will kill a million Americans this year—54 per cent of all deaths.
- 675,000 will die from heart attacks, 176,000 of them under 65. These are "premature" heart attacks; their victims are primarily men at the peak of their productivity.

These facts are well known. Not so widely appreciated, however, is the fact that we know how to stop much of the dreadful toll. "Perhaps we can't prevent heart attacks completely," says Dr. Moses. "But we could push most of them back into the 80's and 90's instead of having people struck down in the prime of life. Most people would consider that quite an improvement."

Moses's optimistic statement doesn't mean that victory is complete. There are still gaps and tangled threads in the cloth of evidence. But for the first time, the bulk of informed scientific opinion has swung, with an almost audible click, into something like agreement. During the past two years, most of the country's principal scientific and public health agencies have reached the conclusion that premature heart disease — at least a

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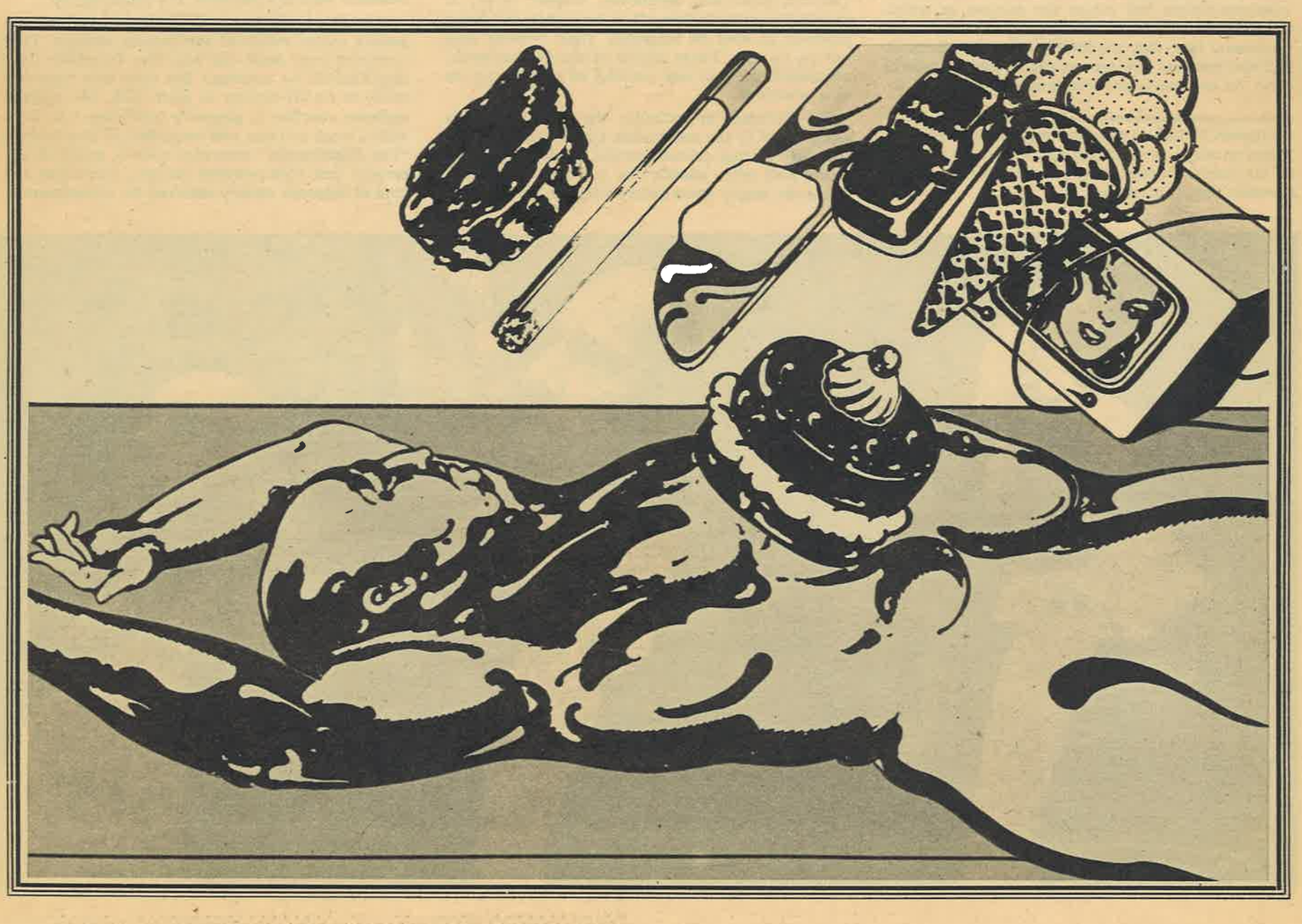

ILLUSTRATION BY CHRISTIAN PIPER

Heart disease

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good part of it—can be prevented.

Finding the answers wasn't easy. Suspected at one time or another of being the principal villain behind the heart-attack epidemic were such diverse items as fat, cholesterol and sugar in the diet, lack of certain vitamins and trace minerals in food and water, excessive coffee drinking, overweight, high blood pressure, diabetes, stress, personality, a sedentary life, body build, cigarette smoking, even soft water. One study even correlates the heart attack rate with lack of regular church attendance, another with too much sleep and a third with ownership of radio and TV.

The problem

Heart disease comes in many forms. The heart muscle itself can simply fail or become infected. Valves that regulate the flow of blood in and out can leak or refuse to close when they should. The timing mechanism that keeps the beat of life going can become erratic and slow down or speed up dangerously. But the great killer—the one behind what we call heart attack—is atherosclerosis.

The beginnings of atherosclerosis, one of a family of blood-vessel diseases called arteriosclerosis, are still mysterious. When children go off mother's milk and onto cow's milk, they develop what investigators call fatty streaks—long, yellowish markings—on the inner linings of their arteries. Fatty streaks in early life are not important and usually disappear. But there is evidence that in some people fatty substances such as cholesterol are deposited in the fatty streaks, and in later life can produce damage to the arteries. Gradually, the lining of the arteries becomes heavy and irregular, and displays thick deposits called "plaques." This whole process severely restricts blood flow through the artery, and the roughened spots can break loose, cause rupture by weakening the blood-vessel's walls, or provide ideal spots for the

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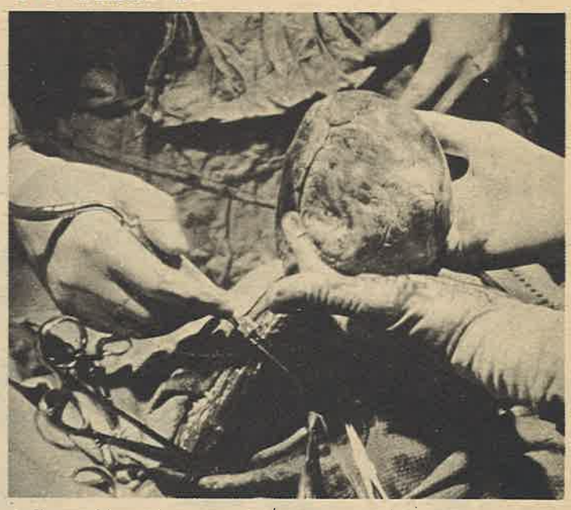
Vital muscle



A diseased heart is removed during transplant surgery.

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formation of blood clots that choke off blood flow entirely. It can happen in any artery, but it is most common in the large and middle-sized arteries in the body — in the brain, the kidneys, the aorta, the legs. When blood circulation is cut off, or an artery breaks in the brain, the result is called a stroke. But the damage takes its greatest toll when it affects the arteries of the heart—the group of three, 5-inch long, soda-straw sized blood vessels that sit atop the heart and wrap around it like a crown (hence their name: the coronaries).

The process of arterial injuries, or lesions, is slow, probably taking 20 to 40 years on the average. Most Americans have moderately severe atherosclerosis by the time they reach 50.

For many years, hardening of the arteries was thought to accompany only old age. Then, in the Korean war, came a stunning discovery: A team of doctors performing autopsies on young soldiers killed in battle found that nearly 8 out of 10, most of them in their late teens and early 20's, had appreciable coronary atherosclerosis. As the realization grew that the disease was not restricted to the aged, many investigators began to examine their other preconceptions about the disease. Main question: Was atherosclerosis really an inevitable process—or could it be a preventable disease?

The disease of affluence

Studies of the world's population have revealed a remarkable fact: The richer the country, the more likely it is to have a high rate of coronary heart disease. A study in England a few years ago showed that even within a country the relationship between wealth and heart disease holds; in fact, the single facet of that affluent society that correlates best with the heart-attack rate is the number of radio and TV sets in a home. Nobody seriously believes that radio and TV sets bring on heart disease. Rather, the blame must lie with a host of changes in life-style that accompany the acquisition of these material wants. But what elements are responsible, and might be changed to prevent the disease? In the history of the growing awareness that heart attack is associated with certain traits and habits characteristic of an affluent civilization, there stands out one great scientific landmark — the Framingham study.

Framingham, Mass., is a gray factory town surrounded by pleasant suburbs 21 miles from Boston. As a town whose 28,000 residents in 1949 were ethnically and sociologically representative of the American population, it was a logical candidate for the Heart Disease Epidemiology Study of the National Heart Institute. Two and a half decades ago, more than 5,000 healthy men and women between the ages of 30 and 59 and free of any sign of heart disease were selected for the experiment in Framingham. In the 23 years since, each subject has come in for biennial examinations. During the course of the experiment, some 900 subjects

have died. And from these deaths have come a series of scientific facts where only general impressions and suspicions had existed before.

The "risk factors"

The study's overwhelming accomplishment was the identification of a series of "risk factors." It proved that men have more and earlier heart attacks than women, and that people who have high levels of cholesterol in the blood, high blood pressure, diabetes, electrocardiogram (EKG) abnormalities or low vital capacity (the amount of air that can be expelled from the lungs), who smoke cigarettes or have parents and grandparents with heart problems are more likely to have heart attacks than those with none of these factors. And the more factors, the higher the risk.

The study put numbers on the risk factors. For example, cholesterol in the vicinity of 250 milligrams per 100 milliliters of blood had been considered perfectly normal; yet the study showed that a man with 240 had three times the risk of a heart attack of a man below 200. An individual with a systolic (pumping) blood pressure of 160 was four times as likely to have heart trouble as one below 120; if he had an abnormal EKG, his risk was two and a half times as great, and low vital capacity or cigarette smoking made him twice as likely a candidate for trouble. Surprisingly, being overweight was not particularly important—unless it was extreme, or connected with other risk factors. The study showed that a combination of risk factors increases the danger enormously. For example, men with moderately high cholesterol levels, moderately high blood pressure and certain EKG abnormalities are 23 times more likely to have a heart attack than those with none of these factors.

An important element to recognize about the Framingham study, and others since then, is that it demonstrated a clear *statistical* association between heart disease and a number of factors. But statistical association doesn't prove cause and effect. For example, cigarettes and heart disease are associated statistically. But one can either assume that the cigarettes cause the heart trouble, or that something that makes individuals susceptible to a heart prob-

lem also makes them more likely to smoke. Either hypothesis, in the absence of additional evidence, is equally good. The same reasoning applied to the other Framingham risk factors. The real question became: Will changing the risk factors help to prevent heart disease? Some of the answers were surprising—and not hard to establish.

(1) **Diabetes:** Study after study gave the same result: Controlling diabetes does *not* lower the risk of heart attack. But the point is moot; treating diabetes *does* clearly lower a patient's chances of dying from a variety of other ailments. So there is no question that diabetes should be treated vigorously.

(2) **High blood pressure:** Same situation. Lowering blood pressure does not reduce the heart attack risk. But it dramatically lowers the probability of death from heart *failure* and stroke.

(3) **Overweight:** Actuarial tables drawn up by insurance companies have shown that obese people die earlier from a variety of diseases—including heart problems — than those whose weight is normal. The Framingham study, by contrast, showed that *moderate* obesity—10 to 20 per cent over ideal weight—did not seem to be directly connected with heart attacks, *unless* the subject also had high levels of blood cholesterol, in which case his risk was far higher than that of a normal subject with the same cholesterol level. Somehow, the two worked together. So there has never been much argument that being thin is better than being fat, and probably gives some protection from heart disease.

(4) **Sex, heredity, vital capacity, EKG abnormalities:** Nothing you can do about these anyway, so no chance for reducing risk here.

(5) **Cigarette smoking:** There was still some argument until about the time of the Surgeon General's report on smoking and health in 1964. But since that time, despite a few holes in the evidence and the best efforts of the Tobacco Institute, the implication of cigarettes as a causative factor in heart disease has been almost completely accepted by the scientific establishment. Heavy cigarette smokers are more than three times as likely to die from a heart attack as nonsmokers; when they quit, their death rate declines almost immediately. It continues to decline slowly for at least 10 years, although even then it is somewhat

above those who have never smoked.

(6) Blood cholesterol: This is the one all the fighting has been about. Many investigators have conclusively demonstrated that the level of blood cholesterol can be reduced by diet. But does lowering the serum (blood) cholesterol reduce the risk of heart attack? Should Americans ease off on the products they like so well and consume in such enormous quantities—thick, well-marbled steaks, prime rib roast, ham, leg of lamb, ice cream, scrambled eggs and bacon, cheese, chocolate bars, rich, butter-filled baked goods—and turn instead to veal, chicken, fish and low-fat milk, in an effort to protect themselves from the great scourge?

Diet—the crux of the matter

In a way, the diet-heart argument deserves the central place it has received. While research has not resolved the prevention question to the satisfaction of everyone, it is clear that high blood or serum cholesterol is *the* central risk factor. And diet is central to serum cholesterol levels. In countries where the general level of serum lipids—blood fats—is low and the heart-attack rate a fraction of that seen in the United States (Chile, Japan, Italy and Greece, for example), other risk factors such as overweight and cigarette smoking are far less potent; in fact, some of them cannot be correlated with the probability of heart attack at all. Conversely, there is evidence from many countries that the individual with a *low* serum cholesterol reading is virtually immune to a heart attack.

But is cholesterol in the blood *causing* those clogged arteries? Or is some basic process, as yet not understood, causing both the accumulation of cholesterol in the artery walls—the atherosclerosis—*and* the high blood levels? And the other logical question: Will reducing the level of serum cholesterol help prevent heart attacks, or is that simply treating a symptom and not the disease?

Cholesterol is a white, odorless, tasteless, nearly ubiquitous fatty alcohol found principally in eggs, meats (especially organ meats) butterfat, shellfish (in moderate amounts), and practically all foods except vegetables. It is also manufactured in the body, primarily by the liver, and is essential for life. Cholesterol is used by the body, for example, to make hormones and bile acids, which play an important part in digestion.

Two substances in the diet largely control the circulating blood cholesterol level. One is dietary cholesterol. The more you eat of such foods as eggs and sweetbreads and, to a lesser extent, clams, scallops and oysters, the higher the cholesterol in the blood serum. Even more important in influencing the serum cholesterol are dietary fats. *Saturated* fats, the kind usually solid at room temperature, make serum cholesterol rise; foods such as butter, cheese, beef, pork, lamb and chocolate are high in saturated fats. *Unsaturated* fats tend to make serum cholesterol go down; they are usually liquid at room temperature and include corn, cottonseed, safflower and other vegetable oils. (An important exception is coconut oil, which, though a liquid, is one of the most saturated fats in existence. And there is a third, in-between category, such monosaturated fats as olive and peanut oil, which neither raise nor lower cholesterol level.)

Researchers have for years suspected that diet is involved in atherosclerosis, simply because arterial plaque is made up largely of cholesterol. But the evidence was only circumstantial, and a good argument could be made that the cholesterol in the plaques was manufactured by the cells in the artery walls and had little to do with the supply circulating in the blood stream. In fact, the question of how cholesterol is deposited in the artery walls is still one of the most mysterious aspects of the problem.

The first evidence of the importance of diet came in 1913 when a Russian pathologist named Nikolai Anitschkov fed rabbits an experimental high-cholesterol, high-fat diet. The animals quickly developed severe hardening of the arteries, a condition never found naturally in proper lettuce-leaf-nibbling bunnies. Since then, researchers have shown that it is possible to induce atherosclerosis in almost any animal by a diet high in cholesterol, saturated fats, or a combination of the two. But despite numerous such experiments, heart specialists remained unconvinced. What happens in animals may not happen in man. Rabbits, for example, are normally vegetarian, and their metabolic response to

substances they usually do not eat may be quite different from the human response.

Of Trappist monks and priests

As animal work progressed, other investigators looked into the epidemiology of heart disease for clues to its cause. European workers, for example, noticed that coronary death rates dropped precipitously in Holland and Norway during the Nazi occupation—when the normal fat and cholesterol-rich diets were drastically changed.

A researcher named Mary Helen Goodloe spent 11 years studying religious orders with varying dietary patterns. Frugal Trappist monks, who eat plain, low-fat foods, have on the average only 90 heart attacks per 100,000 man-years—a commonly used measure. Trappist fathers, a bit up the scale in dietary permissiveness, have 290. Brothers of the less austere Benedictine order have a heart attack rate of 290, same as the Trappist fathers. But the rate among Benedictine priests, who eat an extremely rich diet, soars to 920. (Mrs. Goodloe also noted that brothers in both orders perform considerable manual labor; priests lead scholarly, inactive lives.)

Suggestive, indicative, persuasive to some, but not proof. Maybe there are one or more factors other than diet—heredity, national lifestyles, exercise patterns or characteristics of unknown origin—that produce the high serum cholesterol and heart-attack rates in some countries. The evidence seems to rule these out. One experiment, for example, studied Japanese men with common ancestry living in three different places: Kyushu, Hawaii, and Los Angeles. Average serum cholesterol levels were, respectively, 160, 210 and 245. Heart attacks are virtually unknown to the group in Japan, up to American levels in the Los Angeles group and in between in Hawaii. In both Hawaii and Los Angeles, moreover, increased levels of cholesterol in the blood and the death rate were also exactly proportional to the percentage of fat calories in the diet.

By the early nineteen-sixties, the medical profession had taken the position that the diet-heart theory was interesting, but unproven. Many thought it might be a good idea to change the diets of high-risk patients—those with multiple risk factors, for example, and those who had already survived one or more heart attacks. Nevertheless, when it came to making recommendations to the public at large, most of them balked. For one thing, nobody was sure—really sure—that it would do any good. Second, nobody could be absolutely certain that changing the basic American diet would not be harmful to some people. And third, encouraging a large-scale switch of dietary habits might wreck whole segments of the food industry, for example, the meat, egg and dairy producers.

Dream study

There was a way to settle the problem: Take two large groups of people and let one continue to eat the super-rich typical American diet for 10 years or so, while the other changes to a low-saturated-fat, low-cholesterol schedule. If Group II has substantially fewer heart attacks and other complications of atherosclerosis, the point will be proved beyond a doubt. Thus, more than a decade ago, many researchers began calling for a 10-year, $100-million national study of 100,000 people to settle the matter once and for all.

The National Heart Institute came up with $750,000 for a feasibility study in the early nineteen-sixties. Half of 1,500 men who were selected in five cities ate a diet especially prepared by food processors for the experiment. They ate ice cream containing vegetable fat, for example, rather than the usual butterfat; steaks were special lean, tenderized cuts; cakes and pies were made with unsaturated fats and no egg yolks. The other subjects ate an apparently identical diet, but it was closer to the typical American fare in saturated fats and cholesterol. No one knew until the experiment was over which diet he had been on. And, of course, all 1,500 men were free to live their lives without any restrictions other than those on their diets.

The purpose of the study was not to see if the so-called "prudent" diet would prevent heart attacks, but rather to see if (1) it would lower serum cholesterol in those who were on it, and (2) whether the experimental group would stick to the diet.

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If those two questions were answered affirmatively, then it would be possible to go ahead with a mammoth 100,000-person, 5- to 10-year study.

The Diet-Heart Feasibility Study was a success. The men did stick to the diet for the planned two years, and their average serum cholesterol readings dropped by about 10 per cent. In addition, the low-fat group had only about half as many heart attacks as the control group. Doctors were quick to point out that the number of subjects was too small and the time too short for any firm conclusions. Moreover, many men had become interested in preventing heart attacks and had lost weight, stopped smoking and taken other steps that were perhaps good for them but made the apparent improvement in heart-attack rate meaningless. Nevertheless, the investigators said that the study had clearly proved the feasibility of a big study.

Unfortunately, the money was not available. But other, smaller investigations, some of which had been started even before the National Diet-Heart Feasibility Study, began to produce results. One of the most famous was launched in 1957 by Dr. Norman Jolliffe in New York, and has been carried on in recent years by Dr. George Christakis of the Mt. Sinai School of Medicine. To date in the Diet and Heart Disease Study Project (which was quickly dubbed the Anticoronary Club), more than 1,000 men between the ages of 40 and 59 have volunteered to eat a diet of no more than 30 per cent fat, rather than the usual 40 to 45. The results have been impressive. The men on the special diet have had heart attacks at the rate of 430 per 100,000 man years. But the controls—men who did not eat any special diet—had attacks at the rate of 1,025, or more than twice as many.

Dr. Christakis's critics say his work is not convincing because he is strongly committed to proving the virtues of a low-fat, low-cholesterol diet. Furthermore, he used highly motivated experimental subjects who were all, presumably, anxious to avoid heart attacks and got into the experiment for that reason. So, the argument goes, might they not also be taking generally better care of themselves in other ways that would upset the experimental results?

Another study published a few years ago goes a long way toward meeting those objections by avoiding volunteers. Some 850 of the elderly men at the Wadsworth Veterans' Hospital in Los Angeles were divided randomly into two groups. One group was assigned to a chow line in the hospital's dining room where they got standard food. The other group went through a line where the menu looked and tasted much the same, but was cooked almost entirely in polyunsaturated oils and avoided fattier foods. (The subjects were not told which group they were in.) The principal findings: Serum cholesterol levels in the experimental group quickly fell 13 per cent on the average and stabilized there. More important, 31 men in the group who ate the normal American diet died of atherosclerosis or its complications, while only 13 of those on the low-fat diet died of artery disease.

In recent years there is even some evidence—tenuous at this point—that a change of diet can actually reverse the progress of atherosclerosis to some extent. Dr. William E. Conner of the University of Iowa College of Medicine, for example, fed monkeys a high-fat diet and they all developed atherosclerosis. Then he switched them to a low-fat diet. Periodically, his team did autopsies, and found that not only did the growth of atherosclerotic plaque stop, but the plaques actually regressed.

This may happen in human patients. Drs. Donald S. Frederickson and Robert I. Levy of the National Heart and Lung Institute were studying a group of patients with extremely high blood lipids (cholesterol, triglycerides and other fats), a disease which causes peripheral vascular

problems—a clogging of the arteries in the arms and legs with atherosclerosis. When the patients were put on a strict diet and treated with a serum-lipid lowering drug called clofibrate, not only did their serum-lipid levels drop, but the circulation in their arms and legs—far easier to check than coronary circulation—improved dramatically, indicating that the atherosclerotic plaques had become smaller.

A number of other studies have tended to show the beneficial results from lowering serum cholesterol. Yet they still don't furnish the kind of conclusive, ironclad proof most practitioners would like to have. Diet seems to lower serum cholesterol effectively for most—but not all—people. Why? What about cholesterol-lowering drugs? Sometimes they work and sometimes they don't. Why? And the epidemiological evidence isn't quite as firm as it sounds. Members of the Masai tribe in Africa, for example, live on milk and blood, a diet hideously high in saturated fat. The Samburu people of northern Kenya eat milk and meat. Yet both tribes have extremely low levels of serum cholesterol and heart trouble is virtually unknown. Some researchers theorize that their active, outdoor life offsets the effects of diet, others have shown they have a hereditary ability to handle fats in the diet without increasing blood levels of cholesterol. But no one really knows the answer. The Japanese eat few fats and have few heart attacks. Yet their blood-pressure and stroke rate is high. Both strokes and heart attacks come from atherosclerosis. So how can you have one without the other? And how does diet fit the picture?

A wonder drug?

As most of the public attention in the heart controversy focused on diet, other researchers worked on serum-cholesterol lowering drugs to see if they could affect the heart-attack rate. In one experiment, half of a group of 1,400 United Airlines ground personnel each day took pills containing clofibrate, while the other half were given placebos. During the five years of the study, Dr. Louis Krasno of San Francisco reported, the heart-attack rate for the older men on the drug was 1.89 per 1,000 per year, *vs.* 6.6 per 1,000 without the drug—three and a half times higher. In the young men the results were even more striking—0.64 versus 5, indicating that the unprotected group had almost eight times as many heart attacks.

Other drugs have been tried, too, and have shown that they can reduce serum cholesterol, sometimes by large amounts. A combination of drugs and diet is particularly effective in many cases, and some doctors look forward to the time when practically everybody with a serum cholesterol of more than 200 might be put on a combination diet-drug regime to drop the level below this apparently magic number.

As work has progressed over the years, it has become increasingly clear that that definitive 100,000-patient diet-heart study that researchers have been calling for isn't going to happen. First of all, the money isn't in sight. And second, the data showing that something *can* be done about preventing heart attacks are reaching the point where many doctors feel such a study would border on the unethical. "We're not going to take thousands of 20- or 30-year-olds and follow them for years, not changing their cigarette habits, not controlling their high blood pressure, not controlling their general environment, just changing their cholesterol levels to see what happens," says Dr. Moses. Others who have no ethical reservations are beginning to think the program wouldn't work anyway. So much attention has been given to the risk factors that some slight changes are occurring in living patterns and the change may speed up somewhat in coming years. So it is at least possible that we could spend all the time and money and at the end

still not be absolutely sure that diet did it.

Drs. Frederickson and Levy of the National Heart and Lung Institute may have a better answer anyway. Just now beginning under their direction is a study of 250 hyperlipoproteinemics — patients with an inherited predisposition toward extremely high levels of serum cholesterol and other blood fats. Without treatment, most of these victims would die of heart attack at a very early age—many before 30. During the study, they'll all go on strict diets; one-half will also receive drug treatment, but the other half will not.

At the beginning and end of the study, all the subjects will go through a process called angiography, in which physicians use a complex X-ray technique to actually view the blood flowing through the coronary arteries and gauge the degree of obstruction of each. When the test is over, the results will be analyzed to see if those who received the drug had significantly lower serum cholesterol levels (previous experiments indicate the levels should run 20 to 30 per cent lower) and if so, whether their artery disease progressed at any slower rate than those with higher cholesterol levels, stopped progressing altogether, or perhaps even regressed.

Because of the severe nature of the disease seen in these patients, the usual progress of atherosclerosis is, in the absence of treatment, speeded up enormously. So statistically valid results, showing whether the treatment is doing anything to prevent heart attacks, should be available within three years.

The personality variable

As the great diet-heart debate moved ponderously toward a final answer, research progressed quietly in other areas. For many years, for example, it was an article of faith among laymen that heart attacks happened mostly to tense, high-powered, hard-driving executives whose life-style and personality somehow contributed to their fate. Recent research casts doubt on the supposed connection. Dr. Lawrence E. Hinkle of Cornell University Medical College spent five years studying 270,000 men working in various jobs for the Bell Telephone Company across the country. The re-

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sults showed the highest incidence of heart disease among foremen and workers such as linemen, repairers, and installers. In direct opposition to conventional wisdom, the higher on the executive ladder he climbed, the less became a man's chances of heart attack.

Two investigators who believe that stress does count are Drs. Meyer Friedman and Ray H. Rosenman of the Harold Brunn Institute of Mount Zion Hospital in San Francisco, who until 1955, did research in most of the usual factors thought to be connected with heart disease—serum cholesterol, smoking, and so on. Then they noticed that practically all of their patients had something in common: "An upholsterer came in to redo our waiting room," says Dr. Friedman, "and pointed out that the only place the chairs were worn was on the front edge."

The two men began to look at their patients' habits and personalities more closely, and gradually came to the conclusion that most fall into one of two classes. One group that they dubbed Type A's are aggressive, competitive, ambitious, obsessed with a profound sense of time urgency. They walk and talk rapidly, and are men who "get things done." Type B may also be a man of considerable accomplishment, but he seems to be less driven by the pressures of time.

In 1960, Drs. Rosenman and Friedman selected 3,500 men with no sign of heart disease, classified them as Type A or B, and began giving them careful annual examinations. So far, 257 have developed coronary heart disease. The type A's suffered two and a half times as many heart attacks as the type B's. They also ran higher serum cholesterol readings as a rule, but Rosenman and Friedman, after analyzing their data, have concluded that Type A's have a higher risk of heart attack than can be accounted for by cholesterol readings or any of the other conventional risk factors. Thus, they conclude, a Type A personality is itself a risk factor.

Ten years ago, almost nobody but Drs. Friedman and Rosenman believed that stress was involved in heart attacks. Now the professionals are not so sure. Many think the evidence is growing too strong to ignore.

The sedentary life and other suspects

Hundreds of thousands of joggers fill the sidewalks, highways and gymnasiums of America, running in place is a national pastime, health clubs have sprung up and prospered across the country. Does it do any good?

There is some evidence that it may. Dr. Jeremy Morris of the University of London, for example, kept records on the city's bus drivers, who sit behind the wheel all day, and conductors, who run up and down the stairs of London's double-deckers collecting fares for their eight-hour shifts. The drivers had 30 per cent more heart attacks. Further, the conductors who did have attacks seemed to survive them better. Dr. Henry L. Taylor of the University of Minnesota kept track of thousands of railroad clerks and switchmen for 10 years, found 20 per cent more heart at-

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tacks among the less active clerks. Sedentary kibbutz workers in Israel have more heart trouble than their more active associates who live, eat and work on the same land.

Regular exercise has certain measurable physiological effects. It lowers pulse rate, increases vital capacity, causes the heart to pump more blood with every stroke, probably promotes increased vascularization—growth of a better network of blood vessels. Consistent exercise also tends to lower weight somewhat. Maybe that accounts for the apparent benefit. Maybe bus drivers are subjected to more stress. Perhaps railway clerks and sedentary kibbutz workers endure pressures that weren't accounted for in the study, smoke more because they're seated or are affected by some other unknown factor.

Thus, the Task Force on Arteriosclerosis appointed by the National Heart and Lung Institute concluded that the evidence was too incomplete and inconclusive to advocate a national program aimed at increasing the general level of physical activity for the purpose of preventing heart attacks. Yet many experts are loath to believe that an individual in good shape doesn't have some advantage over the individual who isn't. "We're all pro-exercise here, despite the fact that the evidence isn't overwhelming," says Dr. Frederickson of N. H. L. I.

If the case for exercise must be judged unproven, the cases implicating a host of other suspects that have been indicted are even more tenuous. For example, Dr. E. Cuyler Hammond of the American Cancer Society, who followed some 800,000 men and women for six years starting in 1960, found, to nobody's great surprise, a correlation between death from heart attack on the one hand and heavy cigarette smoking, lack of exercise, overweight and high blood pressure on the other. But he made a completely unexpected finding, too: The death rate from arteriosclerotic disease (mainly stroke) was almost twice as high for men and women between 60 and 79 who slept 10 or more hours a night as from those who slept 7. The conclusion of most experts: Interesting, but inconclusive.

A British investigator has presented data that suggests sugar consumption may be the real culprit behind heart disease, but most researchers cite the lack of laboratory proof and believe he is on the wrong track. Heavy coffee drinking—20 cups a day, for example—has also been associated with heart risk, but this case, too, is far from proven. Other researchers have at one time or another seemed to find that various trace minerals, such as potassium, calcium, copper, zinc and lithium—or their lack — were associated with heart attack. While there could be some yet-undiscovered link between heart attack and one or more of these substances, the evidence is, again, so far highly tenuous. One doctor who has been trying to associate vitamin E with protection from heart attacks has failed to present any evidence strong enough to convince his colleagues. Another researcher, Dr. George W. Comstock of Johns Hopkins School of Hygiene and Public Health may have uncovered the strangest clue of all. Choosing a county in Western Maryland, he sought to test the theory, which some studies tend to confirm, that people living in areas where they drink hard (high mineral-content) water have fewer heart attacks than those who drink soft water. He was unable to find any differences between those from hard- and soft-water areas. But in analyzing the details from the socioeconomic questionnaires, he found that men who did not go to church regularly were twice as likely to have a heart attack as those who went once a week or more. Nobody is willing to call this anything but clear coincidence.

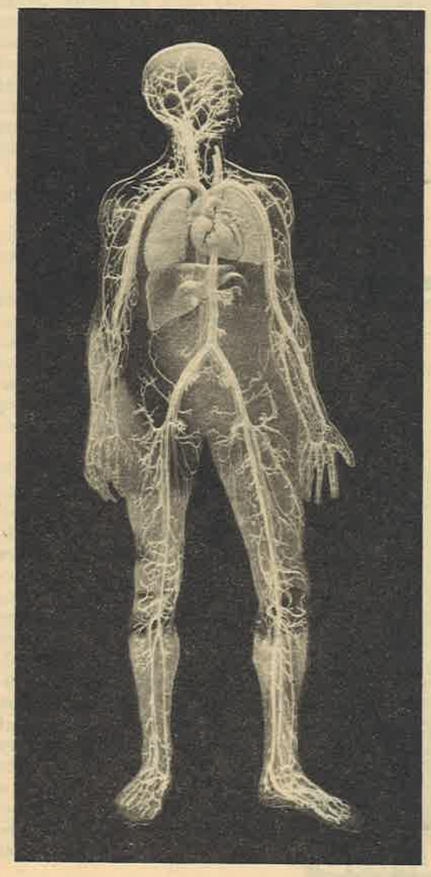
The bandwagon

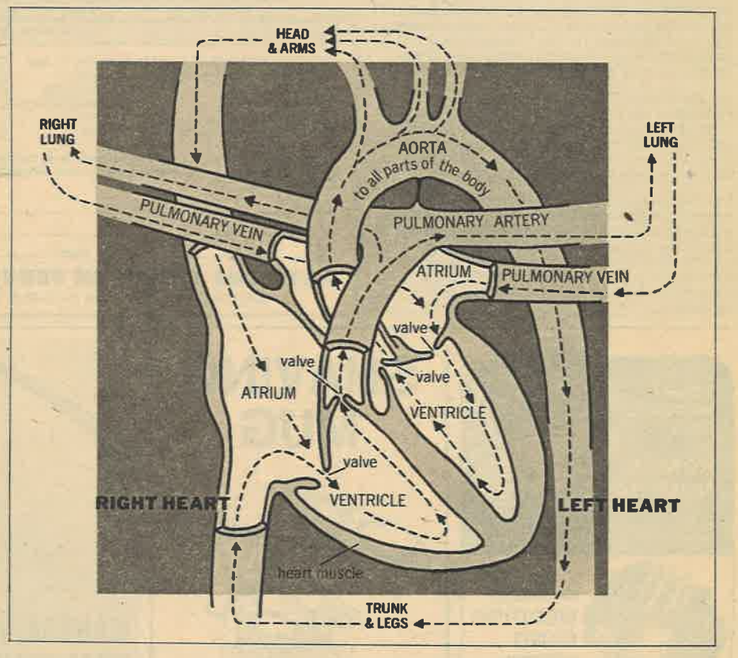
Even before the results from the various studies now under way are in, the great debate seems to be ending. Most researchers now accept as a working hypothesis that the prime causative factor in heart disease is high serum cholesterol. Jumping on the bandwagon early was the powerful American Heart Association, which in 1961 came out in favor of a cholesterol-lowering diet for high-risk patients, and in 1965 recommended that all Americans cut down on saturated fats and cholesterol, and adapt the "prudent diet." The A. H. A. position was hardly more popular with the medical profession than ants at a picnic. Cardiologists tend to be conservative. "They deal with a disease that kills unexpectedly; that makes them conservative," says Dr. Campbell Moses of the A. H. A. They weren't convinced that diet would do any good for the general population, or that it did not contain hidden hazards that would show up only after years of use, after the damage had been done. And there *were* holes in the theory.

Nevertheless, one by one the great scientific organizations have been coming around the conclusion that efforts to combat heart disease must begin with a better diet. The switch began just over two years ago when the Inter society Commission for Heart Disease Resources, a group of more than 100 of the country's foremost heart experts came up with a sweeping set of recommendations, including a warning to the public to cut sharply the consumption of saturated fats and cholesterol. In June, 1971, a Task Force on Arteriosclerosis of the National Heart and Lung Institute came out squarely on the side of prevention: "It would appear prudent for the American people to follow a diet aimed at lowering serum lipid concentrations. For most individuals, this can be achieved by lowering the intake of calories, cholesterol and saturated fat."

One year later, in July, 1972, came a thin but important report from two highly prestigious bodies that had been fighting among themselves for years over the diet-heart controversy: the Council on Foods and Nutrition of the American Medical Association and the ultraconservative Food and Nutrition Board of the National Academy of Sciences-National Research Council (which sets "minimum daily requirements" of all nutrients known to be necessary for human health.) "The evidence now available is sufficient to discourage further temporizing with this major national health problem," the joint report said. Then it went on to recommend that everyone with a plasma cholesterol level of 220 or above—which the board estimated includes two-thirds of the men and an unknown number of women—change their diets to bring the level down. That just about made it unanimous.

The emphasis is now switching to youth. While a





Left, the cardiovascular system, the heart and the network of blood vessels that carries blood through the body. The heart is a hollow organ divided by a strong membrane into two parts that pump blood by contracting. Blood flows into the "right heart" after delivering nutrients and oxygen to the body. From there, the dark, bluish-red blood is pumped to the lungs, where it picks up a fresh supply of oxygen and turns bright red. The reconditioned blood then returns to the "left heart," which pumps it through the great trunk-artery—the aorta—and other arteries to be distributed again in the body.

change of diet and modifying other risk factors will apparently help almost anyone, the younger the age, the more good it does. "The problem in my opinion is a pediatric problem," says Dr. Stephen Scheidt, a cardiologist at New York Hospital-Cornell Medical Center. Most of the professionals I talked to have already banned butter from their homes, switched the kids to low-fat milk, and severely cut consumption of steaks and roast beef in favor of fish, veal and chicken.

"Managerial" prevention

Can we stop heart attacks completely? It isn't possible, maybe not even desirable. "We know death rates are going to remain 100 per cent from living," says Dr. Moses. "A heart attack may be as good a way to die as any. And I would be very happy to see the heart-attack rate rise to 100 per cent, so long as it happened at age 95." Finally, at the end of an incredibly long and complex search, we do know how to prevent many —perhaps most—premature heart attacks. The prescription is deceptively simple:

(1) Have an annual physical that includes blood-pressure measurement, a blood-sugar test and a cholesterol assay. If any of the readings are high, bring them down immediately with diet and drugs, or by any necessary means.

(2) Maintain ideal weight, or something close to it.

(3) Do not smoke cigarettes.

(4) Get some regular exercise.

(5) Eliminate as much stress, tension and deadline-associated activity from your life as possible. If you must work in a stressful occupation, try to avoid combining high stress levels with inordinate fatigue.

Most doctors make little real effort to get their patients to follow preventive medical advice. They probably don't because they are fairly cynical about the effectiveness of such advice to their patients, although there is no way to document such a claim. Says Dr. William B. Kannel, medical director of the Framingham study: "There is the patient who is a recognized coronary risk. His doctor tells him to watch his weight, go on a low-fat diet, stop cigarettes. And the patient says to himself that he could get that kind of advice from his mother-in-law. What he wants is a pill to counteract the effects of his bad habits."

The pill idea, for the moment, is fantasy, but it may not be for all time. If a really safe, effective drug could be developed for keeping serum cholesterol low, such an advance might indeed allow people to do whatever they liked and still be relatively immune to heart attack. Other researchers have hypothesized that if we ever really learn what causes the blood clotting that brings on fatal heart attacks and strokes, we might be able to prevent them—perhaps with a pill.

Meanwhile, organizations responsible for the nation's health, recognizing that most individuals will not take the necessary steps to cut their risk, are recommending so-called "managerial" prevention. The Intersociety Commission, for example, has called for an "orderly phasing out" of the cigarette industry, a major national program to detect and control high blood pressure, a program to breed cattle with a smaller percentage of fat to meat (an Australian team recently reported that it had sharply reduced the saturated-fat content of steaks by special feeding of the cattle) and a repeal of all laws that make it illegal to do such things as manufacture meat products containing polyunsaturated vegetable oils.

The task force of the National Heart and Lung Institute recommended that the country's food industry be persuaded to influence the composition of the American diet by all means possible. Great reductions in average intakes of saturated fats could be achieved, for example, if food processors switched to vegetable oils in the preparation of all snack foods, TV dinners and similar prepackaged items. One company is already test marketing a cholesterol-less egg product. Nutritionists who have tried it tell me that scrambled eggs made from it are indistinguishable from the real thing.

Major changes in laws, habits and customs take time, and any large-scale reduction in the over-all heart attack rate by Government fiat or industrial revolution will be years in the making. In the meantime, those who want to protect themselves and their families finally know what needs to be done. ■