PANEL DISCUSSION
Guest Editor: HERBERT POLLACK, M.D.

Present Status of Lipid Metabolism and Atherosclerosis

The Panel was comprised of the following members: HERBERT POLLACK, New York, N.Y., Moderator; ROBERT E. OLSON, Pittsburgh, Pa.; Oglesby PAUL, Chicago, Ill.; and HERMAN E. HILLEBOE, Albany, N.Y.

The Panel Discussion was conducted at the Thirtieth Scientific Sessions of the American Heart Association held at the Sherman Hotel, Chicago, Illinois, on Sunday, October 27, 1957.

M ODERATOR POLLACK: This panel was designed to try to answer some of the questions that are being raised today in connection with the subject of the etiology of atherosclerosis. Cardiologists have been so concerned with the diagnosis and treatment of clinical cardiologic disorders up to fairly recently that they did not have time to occupy themselves with the etiology of one of the principal causes of myocardial disease, that is atherosclerosis.

The reopening of this question of the etiology of atherosclerosis has been rather bewildering because it brings up new terminologies and technics foreign to the electronic principles that have engaged many of the clinical cardiologists. They have been forced to listen to such terminology as poly-unsaturated fatty acids, iodine numbers, beta sitosterol, and many other names that are new in the cardiologic literature.

Another new aspect of this subject has been the epidemiologic one. Here again the cardiologist as a rule has not kept pace with the development of epidemiologic technics, the study of the community as a whole rather than the patient as an individual.

To start the panel today, I should like to ask Dr. Herman E. Hilleboe, Commissioner of Health of the State of New York, to open the discussion on the etiology of athero sclerotic heart disease.

DR. HERMAN E. HILLEBOE: I should like to express a few views on the epidemiology of atherosclerosis, particularly some of its terminal effects in the heart. In coronary artery disease, for example, we are dealing with a disease entity that appears to have multiple factors in its causation as well as in its manifestations. We feel that by observing its natural history among people under varying environmental conditions and mode of life, we may be able to unmask some of the hidden secrets. This we call the epidemiologic method of study. It is no different from that used in the study of the infectious diseases in the past except that we are dealing here with diseases that are of unknown etiology, and we are therefore at a distinct disadvantage.

We must use indirect methods of study. Consequently it is necessary for us to talk about associations and degrees of associations and not about cause and effect. Certainly our work with tuberculosis seemed complex, even though we knew the cause was the tubercle bacillus and we could classify pulmonary lesions as minimal, moderate, and far advanced. We cannot do this clinically with atherosclerosis, which manifests itself in the heart and brain and in the peripheral circulation. I think that we need not know the cause of diseases in order to do something about them. If we can learn enough about their epidemiologic characteristics to detect their occurrence early and to prevent their progression then public health workers and private practitioners can proceed collectively to do something about them.

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The biggest criticism that we would make of some of the work going on—and I hope that this is considered as constructive criticism—is that we are dealing with diseases difficult to define. In the literature and in the work that is done, we find that defining coronary artery disease itself or the manifestations of atherosclerosis are in quite a confused state. We find that in the living individual, because of the difficulty of determining the incubation period of coronary artery disease, we are at a loss to study its natural history. We know, too, that postmortem studies present difficulties in classification.

In addition, as we view some of the studies throughout the world on the natural history of atherosclerosis we find great difficulty in bridging the gaps that occur. Specifically in coronary artery disease we think first of the diet itself, the components of the diet, particularly fat, the fat content of the blood, the fat composition of the arteries in which we are particularly interested, and the terminal episode.

It is very easy to relate the diet to several constituents in the blood. We cannot by inference jump from the relationship between these 2 things to the relationship between the constituents in the blood and the cause of impairment and death. Our greatest gap occurs at this point.

As epidemiologists we start out initially by studying the principal factors. For example, in coronary atherosclerosis we are interested in any relationships that there might be between the available food, as for example the fat in the diet, and death from various types of coronary artery disease or associated heart conditions. It is easy to select a half dozen countries and to show a linear relationship between the available fat in the dietary of the countries and the mortality from arteriosclerotic heart disease. But if we look more extensively into the available food and the mortality from heart disease, we at once learn that the picture is not so simple as it might appear.

The first comment that I would make as an epidemiologist is that we must not alter the facts that should be considered before constructing a hypothesis that we will try to prove.

It has been shown by a study of the data from as many as 22 countries that if we review the figures concerning the available food supplies and the mortality, the apparent association observed when applied to a limited number of countries is greatly reduced; also that the basic data are subject to considerable limitations. This caution applies both to the components of the diet in the different countries and to the mortality, especially to the classifications of causes of death. In addition the presumed association is not specific for fats in the diet nor for diseases of the heart. For instance, the association of mortality from heart disease with diet is stronger when the correlation is with animal protein. There is a negative association that we find for both animal protein and fat with mortality from noncardiac disease. It is not enough simply to rack up a series of positive instances where those things that we would like to show are available. We need to think of the negative instances and to explain them at the same time.

Purely on a statistical basis we can conclude from the available data, poor as they may be, that the suggested associations between national death rates from heart disease and the percentage of dietary fat available for consumption cannot at the present time be accepted as statistically or epidemiologically valid. From this conclusion we can proceed to the additional studies in the chain effect, i.e., where inferences are drawn from the constituents of the diet, particularly fat, to the constituents of the blood, to the constituents of the vessels of the arteries, to the effect that we will call coronary artery disease or some other type of disease such as cerebrovascular disease.

Finally, in looking over the work that is going on in this field of atherosclerosis and its effect on the morbidity and the mortality of the population, we see certain areas where more work needs to be done. Certainly a tremendous amount has been done on the relation between elements in the diet and some ele-
ments in the blood. But in the effect of the diet on the coagulation factors in the blood, there may well be a relationship of greater importance than that of the mechanical basis of fat in the blood and fat in the vessel walls themselves.

There are other factors to be considered, i.e., the relationship to occupation and to mental stress.

In conclusion, as epidemiologists concerned with this complicated study, we are perhaps the critics who stand back and look at the disease as a whole as it affects the population. Those of you who study groups of individuals must give us good data and we will attempt to put together the array of facts in an orderly fashion for mutual interpretations. Until such time as we can find reasonable justification for affecting the population as a whole, I think we must keep many of our activities purely on an experimental level.

Moderator Pollack: Each of the panelists will be asked to give a brief discussion of one aspect of this problem and then questions from the floor will be invited. When honest men disagree, as the late John Peter said, then obviously there was something in the conditions under which they were working that differed. It is up to us to find out just why these conditions appear to be the same but are basically different.

As the next part of this topic, Dr. Robert Olson, of Pittsburgh, will discuss the physiologic and biochemical aspects of blood lipid and the dietary relationship involved.

Dr. Robert Olson: The idea that atherosclerosis results from a disorder in lipid metabolism probably dates from the very early observations of German pathologists. They noted that the lipoidal material that they could isolate from the atheroma resembled in general the composition of the serum. These observations were followed by the classic experiments that showed that by feeding cholesterol to rabbits experimental atherosclerosis could be induced. Since this time the preoccupation of the biochemists with the idea that atherosclerosis is a disorder of lipid metabolism has increased. The search for truth has opened up in a marvelous way many avenues of research, including the establishment of new techniques which I think are going to revolutionize the biochemists’ concept of fat metabolism, such as column chromatography, gas phase chromatography, and many other techniques that have attracted a number of workers in this field.

I think it would be amiss to be so preoccupied in the field of lipid metabolism to believe that it is the only factor involved in the pathogenicity of atherosclerosis. The weight of my remarks will be directed toward the idea that we must not lose sight of many other factors involved in the pathogenesis of this disease, some of them certainly completely nonlipoidal and foreign to the area of fat metabolism.

Taking a lead from Dr. Hilleboe’s discussion of the epidemiology, I will consider the triangle which the epidemiologist deals with in the general analysis of infectious disease. He relates by a triangle the host, the environment, and the agent. The problem with atherosclerosis is that the agent is not easy to find. It may not exist as a single entity, but certainly we can say that circumstances which lead to the formation of atheroma arise by interaction between host and environment. The agent is endogenous. To complicate the picture further we can add those factors that seem to bear some relationship to the progress of atherosclerosis in the host: heredity, race, age, sex, endocrine glands, and even the psyche. The environmental factors that have been implicated in one way or another include diet, not only the fat content but other nutrients that have an effect on the fat metabolism, certain drugs among which we might include beta sitosterol, exercise, occupation, and possibly even culture and climate.

At the upper end of the triangle we can segregate some local factors and some humoral factors arising in the host as a result of interaction and environment. We might segregate factors such as serum lipids, lipoprotein-clearing factors, and coagulability of the blood, and even have local factors that affect the response of the artery. They include
hemodynamic factors, dealing with pressure and turbulence, metabolic factors dealing with the arterial tissue itself, with its structural composition and any trauma that it undergoes.

To highlight this multiple etiology further I relate diet, which is so widely discussed today, to clinical events that can be measured by the clinician or the epidemiologist. Between these 2 determinants there lies a tenuous route of association possibly because I am going from diet, to serum lipids, to the process of atherogenesis, and then to the clinical event. We know that this line of association is not a direct line, and the length and strength of the connections cannot be precisely defined at the present time for a population or for an individual.

We have to recognize that the effect of diet—and the caloric balance, the protein intake, the fat intake, the carbohydrate intake, and possibly even the mineral and the vitamin intake—may have some influence not only upon serum lipids but upon other events in this chain, and we have to recognize other factors operating at each level to produce this disease.

In discussing this problem I refer to the multiple factors even at the site of interaction between beta lipoproteins and serum. You will agree that there may also be a determinant in the process of atheromatosis in the artery itself.

One can see possible sites of breakdown in this system at the present time. Changes in the intima due to thrombus formation may be the first. Much emphasis has been placed upon changes in the elastic membrane, and even here we must invoke the concept of multiple etiology.

The relationship of serum cholesterol to clinical events, mostly of myocardial infarctions, has been analyzed in 5 populations of individuals studied by the Public Health Service over the past several years. There is a wide distribution of cholesterol values, from 150 mg. per cent up to very high values.

We cannot with this evidence make a 1:1 relation between serum fat and clinical disease. It is interesting, however, that many of the subjects with infarcts seem to cluster above the midpoint, between the 50 and the 75 percentile of the serum cholesterol distribution curve; this distribution confirms the fact that the serum profile is one determinant of atherosclerosis but its quantitative significance in a given individual, or even a given population, in my opinion has yet to be evaluated.

I present what is a reasonable summary of our concepts of fat metabolism at the present time relating to 3 organs, the gut as the site of absorption, the liver which serves as the site of transformation, and the depots which serve as the site of storage. The fat that is absorbed by the gut passes to the liver and the other tissue, and the liver transforms this chyle in large amounts to lipoproteins, etc.

Further, we know that in certain cases, such as diabetic acidosis and in starvation and in certain pituitary disorders, the depots can mobilize a great amount of lipid, for example, chylomicrons, and transform it to beta lipoproteins, and the response is often an increasing output of beta lipoproteins. I would like to call your attention to the liver, possibly a central organ in mediating the response of depots of fatty acids mobilized in various ways; also the liver may have an important influence in transforming dietary modifications of the lipoprotein profile.

In conclusion, I would say that at the present time the physiologist should look upon—I think is compelled to look upon—atherosclerosis as a disease of multiple etiology in which disorders of lipid metabolism may play a role. Secondly, beta lipoproteins appear to be a determinant in the process of atherogenesis but their quantitative significance is yet to be precisely evaluated. Third, dietary manipulations can alter the serum cholesterol and lipoprotein configuration. The extent to which this happens in individual cases and in individual populations in the presence of varying components of diet other than fat is still a subject for investigation.

Finally, I would make the plea that every physician taking care of middle-aged patients
consider himself a clinical investigator in this particular period when data are needed about the relationship of the diet and of the lipid levels and of the natural history of atherosclerosis, to define the problem further so that some reasonable position at the public health level can be taken.

Moderator Pollack: Dr. Oglesby Paul will now discuss the clinical aspects of this problem.

Dr. Oglesby Paul: Dr. Olson has brought the clinician into the story, and what can one say in ten minutes about the clinical phases of coronary heart disease that we don’t already know? I doubt if we can say anything new, but we can perhaps emphasize certain points that may be helpful.

The multiple-factor theory is helpful for the clinician to keep in mind. We all have patients who are fat—overweight—who go on until the mid-eighties and never get into trouble. We have lazy patients and patients with high serum cholesterol who likewise do not get into trouble. If we think in terms of a disease process in which different weightings of these factors may be important, we have an approach that helps us to understand some of the clinical variables we see in our offices and in hospital and clinic practice.

I hope that this multiple-factor theory does not exclude the possibility that the control of 1 or 2 critical factors may permit the control of the disease as such. If it were necessary to control each and every item Dr. Olson listed, this, indeed, might be a hopeless problem.

As clinicians, I think it is important that we recognize that we are not making a diagnosis of atherosclerosis as such except under rare instances. When we make a diagnosis of atherosclerosis with involvement of the coronary bed, what we are really diagnosing, as you know, is the complication of atherosclerosis. It is only when infarct or ischemia occurs that we are able to say with any certainty that an individual has coronary heart disease. But it isn’t even this simple, because we have, as the coronary disease progresses, a similar development of collateral circulation. When a patient has symptomatology due to coronary disease, it represents the inadequacy of both the collateral circulation and the coronary circulation with which he was endowed.

The variable course of this disease is one of the problems that is sometimes forgotten, and in too small a group of patients, particularly patients who are followed without adequate controls, I think that we get utterly and totally lost. This is particularly true of small groups that are treated by physicians in the office and in clinic and hospital practice, where the relatively good outlook over a short period of time would make one consider that one is getting satisfactory results.

Just to remind you how variable is this disease with its remissions and exacerbations, I will present very simply the course of a few patients illustrating some aspects of this fact. First is an individual going from 1944 with a course that is now actually associated with less angina than he had for a period of many years. Next is another individual who 21 years ago had his first infarct, which is well documented. He had another infarct, a ventricular aneurysm, and advanced angina decubitus; and he is now free of angina. Next is an individual who has gone along with essentially the same amount of symptomatology for a period of 12 years.

We are deluding ourselves if we think of this disease as being either a malignant disease with a standard predictable progression or that it is a disease with a standard stable course. It is neither, and it will take a good many cases, followed over a good many years, by each of us, before we can get satisfactory data regarding the course of the disease and the effect obtained by any agent.

Finally, I would like to remind this audience that all of us who see patients are having difficulties with the publicity that attends the subject of fat in the diet. If the American public has a weakness, it certainly has a weakness for going into a panic at certain times. You will remember the mock invasion from Mars that struck New York some years ago when everybody headed for the hills. I think that maybe Sputnik has had a similar effect on the American public. The medical litera-
ture and the drug literature relating to the subject of fats and cholesterol, and the papers you have heard here today, all serve to create a wave of public interest that creates a problem in the hospital, in the clinic, and in the office. It requires patience and conservatism to withstand the onslaught of patients, press, radio, and television, and to withstand the temptation to make a dogmatic statement and to give treatment with a certainty not justified.

**Moderator Pollack:** The panel will now entertain and invite questions from the floor for further discussion.

While we are waiting for some of the people in the audience to gather their thoughts, Dr. Hilleboe, I would like to ask you if you would care to comment briefly as to whether or not we are running into an increasing epidemic of coronary heart disease.

Dr. Hilleboe: This question must take into consideration several factors. First of all, we have an aging population and, as you know, with an aging population we have more disease of various types, including atherosclerosis and its various manifestations. So corrections must be made for the change in the age and sex distribution of our population.

Secondly, we must recall that in 1949 there was a change in the nomenclature of the disease, and that if we attempt to make studies of long-term trends of the disease going from a period of time at the turn of the twentieth century to the present we must have an adjustment at 1949, because we called coronary artery disease one thing before that time and another thing after that time.

Thirdly, we know that within the short space of the last 10 years there has been quite a change in the accuracy and the completeness of the diagnosis of coronary heart disease. I happen to serve on one of the advisory committees to the National Office of Old Age and Survivors' Insurance, and twice a year we review some of the samples of diagnoses received from physicians. Some of them have been made by older physicians, middle-aged physicians, and recent graduates, and certainly the types of conditions that are called coronary heart disease are legion, because we go all the way from simple angina to the classical picture with changes in the electrocardiogram and changes in the x-ray, with the history and physical findings and other laboratory findings.

So these are the factors that we must consider when we judge whether or not there has been an increase in coronary artery disease. I would say offhand that the increase has been more apparent than real. The extent to which it has increased is modified by the factors that I have mentioned. Properly considered they show that there has been not quite as proportionately great an increase as one would suspect from a cursory examination of recent figures.

**Moderator Pollack:** Dr. Paul, you were talking about the distribution of atherosclerotic lesions in the body. Would you mind commenting on why the pulmonary tree is free from atherosclerosis?

Dr. Paul: Of course, atherosclerotic lesions can occur in the pulmonary circulation where pulmonary hypertension exists. Under normal circumstances, however, the pressure in the pulmonary artery is about one quarter or less than that in the peripheral circulation and the mean pressure is similarly lower. So we have normally a difference in lateral pressure on the vessel wall in the two circulations, which is at least a partial explanation of this freedom from atherosclerosis in the pulmonary vascular bed. When the pulmonary artery has been subjected to increased pressure, atherosclerotic plaques may develop.

**Moderator Pollack:** Then you are still of the old school that believes possibly hypertension has little to do with atherosclerosis?

Dr. Paul: Yes. I think the studies done recently by the Public Health Service in Framingham, for example, demonstrate that hypertension is one of the factors that may predispose to atherosclerosis involving the coronary tree, and their data, which were collected over a good many years and under careful circumstances, would indicate not that hypertension per se is necessarily a critical factor but that hypertension in association
with overweight and hypercholesteremia is a factor.

Moderator Pollack: In all of this discussion the word vitamins has not been introduced. I cannot think of any discussion on nutrition that does not involve vitamins. Dr. Olson, do you want to discuss the role of vitamin B₆ or pyridoxine in atherosclerosis?

Dr. Olson: There is some good evidence on the subject and some additional speculation. We know from the work of several laboratories that vitamin B₆ deficiency in the primate—in the monkey—can cause atheromatous lesions in the large vessels and in the coronary arteries of that species. These are characterized more by intimal proliferation than by fatty infiltration or deposits in the intima. In fact, in the B₆ deficient monkey the serum lipids are quite low.

We also know that niacin at high levels does affect the serum cholesterol level. I am sure that the mechanism of this effect is familiar to pharmacologists. It is not nutritional in the sense of the usual intake of this vitamin, but it provides another interesting problem.

Moderator Pollack: All of which raises the question, Dr. Paul, what would your recommendation be with respect to some of these preparations on the market now that are mixtures of the poly-unsaturated fatty acids and pyridoxine, vitamin B₆, etc.?

Dr. Paul: I can settle this very easily; as to vitamin B₆, no. As to the other preparations that you mentioned I am not aware of any solid evidence that any of them added to the diet affects the course of coronary atherosclerosis, and until I personally have some more solid evidence, I would be glad to have some other people, as has been mentioned in the 2 preceding papers, follow groups of patients and see how they come out. I think if each of us in this room give a little of this and a little of that, we are not going to learn anything and I am sure that we are just going to deceive the patient into thinking that we are really doing something for him. I do not believe, therefore, that the dietary supplements, the vitamin supplements, the fatty-acid supplements are going, in a general, free distribution fashion, to give us any useful information and I would prefer that they not be employed except under rather rigid and carefully controlled experimental conditions.

Moderator Pollack: I would like to ask a loaded question of the panel and give each one a chance to answer it. That is, what is your advice today to patients with respect to the fat content of their diets? Do you tell patients to eat low fat diets, do you disregard fat all told, or just how do you approach it?

Dr. Olson: I would like to answer that loaded question in a circuitous way by commenting upon the attention of physicians to diet therapy in general. Many of our colleagues think that diet therapy is something measurable, that you put the patient on a diet and then he is going to be better, and in many instances, the diet, no matter what it is, is not studied from the point of view of the effect upon the patient's lipid pattern, general well-being, incidence of angina, or future course.

I think that physicians in general have to accept clinical nutrition as a very important part of their armamentarium, a specialty of medicine really, and really learn themselves something about the problems of diet prescription and diet adherence.

With that preface, I would say that in my mind the problem again is centered at the individual patient-doctor level at the present time.

In our own clinic we are studying several combinations of diet therapy for patients with clinical evidence of atherosclerosis. My own feeling is that the evidence today suggests that the unsaturated fats provide the best avenue of diet therapy for controlling serum cholesterol. In our clinic we are studying the effect of incorporating unsaturated fat in various proportions with saturated fat at a level of about 40 per cent of the calories in diets that will maintain weight in these patients. But, as I say, this problem is unsettled and I think it is premature to say without further evidence of this kind, which we all should participate in, that this course or that course is
the preferred one for prevention of further progress of this disease.

Dr. Hilleboe: We attempt to approach something of this kind on the basis of the best advice we can get from the experts. By the experts we mean the cardiologists, the laboratory people, the epidemiologists—anyone who can contribute to basic knowledge for the control of a chronic disease. Our problem amounts to working with the state and the local medical societies in putting out health information that people understand and with which we have some hope of succeeding in affecting the population.

When we use these two means in an attempt to get people to cut down the fat in their diets we fail in both of them. In the first place we do not yet have sufficient reason to state from the available scientific literature and the studies that have been made, including our own studies, that if you cut down the fat in the diet over a period of time you will reduce the amount of morbidity and mortality from heart disease, cerebrovascular disease, peripheral artery disease, or the various manifestations of atherosclerosis. If we cannot give good reasons, it does not seem to us wise to go forward.

Secondly, it is exceedingly difficult to get people to cut down their weight just on general principles, particularly if they are well. It is one of the most difficult problems in medicine. A patient may diet, and reduce 20 pounds, but most of the time he has those 20 pounds back very soon, unless the individual has an incentive related to himself or to his family. This is a task on which a great deal of effort is expended and very little success is obtained.

So we would take this position. At the present time we would rather concentrate our efforts with organized medicine and volunteer health agencies, particularly the Heart Association, to get people who are overweight to cut down their weight by proper reduction of total calories. There is good evidence that by doing so general well-being is improved and that mortality from various types of heart disease is affected. This we can do and this I think is intelligent. In the present state of our knowledge if we attempted to do otherwise and it turned out later that our advice had been bad what would people expect of us in the future? I think that we need to be conservative in this particular respect.

Dr. Paul: I think that there are essentially three groups of people that you are interested in here. For people in good health, if they are overweight, you are on solid ground in suggesting a diet that will reduce their total weight and that will reduce their total calories and that will include necessarily a reduction in their fat intake.

The second group is those patients who have a very bad family history of coronary disease and have perhaps also hypercholesteremia. Should we do nothing for those individuals? Should we have them ride a bicycle around the block 10 times a day? Should we have them see a psychiatrist? Or should we give them a diet? If they are not overweight and they still have this bad family history and hypercholesteremia, I myself give those people a try at a moderately low fat diet.

Third, if you have a patient with coronary disease either with angina or myocardial infarction do you do anything for him? I believe that there is some evidence, which many of us have confirmed clinically, that the patient who eats a high fat meal tends to have angina after it. There are some good data indicating that this may occur, and certainly I have had patients of my own in whom a high fatty meal has resulted in the appearance of angina. For that reason as well as for the control of weight itself, I think that caution in the amount of fat taken in is desirable. It does not recommend that these people go on a strict, low fat diet.

Moderator Pollack: One question has been brought up from the audience. Can essential steroid functions be harmed by effective reduction of cholesterol?

Dr. Olson: It has been shown that cholesterol can be a precursor for a number of steroid hormones, but the percentage of body cholesterol diverted for hormone synthesis is minimal. We know also that these hormones
can be synthesized de novo from acetate, so
that the body pool of cholesterol over any
dietary regime would produce hypocholester-
emia would not in any way interfere with the
synthesis of the steroid hormones.

MODERATOR POLLACK: Are there any other
questions from the audience?

DR. SIDNEY STRAUSS: In this whole meet-
ing I have heard only one etiology for coro-
nary disease: atherosclerosis. I want to call
your attention to the fact that there may be
an arteriosclerotic lesion of the coronary ar-
tery with hardening and gradual narrowing
until even without a thrombotic formation you
get an infarct. What about the fats in that
type of coronary disease? I think some of
these anginas which Dr. Paul showed were
probably arteriosclerotic lesions.

MODERATOR POLLACK: Dr. Strauss’ ques-
tion serves to re-emphasize the extreme diffi-
culty involved in this whole question. As has
been pointed out by several of the speakers,
the question of diagnosis is almost impossible
at times. The principal part of the disease
starts long before it is diagnosed. The pro-
gress of the condition goes on without being
detected. Differentiation, as now introduced
between atherosclerosis, arteriosclerosis, and
so forth, is still wide open and I do not know
the answer to it. Would anyone on the panel
care to pick up Dr. Strauss’ question?

DR. PAUL: I think that when we use the
term coronary atherosclerosis we are using it
interchangeably with the general term used
in the standard nomenclature, arteriosclerotic
heart disease and that is what we are referring
to.

DR. HILLEBOE: The chairman has presented
a question to me that really covers a lot of
territory. Before I answer it I want to know
whether you want to hear it. I will read the
question and if you want to hear something
about it, I will be glad to tell you. If you
do not think it is pertinent, say so. This is the
question from the audience, ‘Wouldn’t the
Public Health Service be doing a more effi-
cient job if they disseminated information to
the press regarding a weight reduction pro-
gram for the obese rather than spending so
much time on the Asian flu problem?’ (Ap-
plause)

I take it for granted that you are clapping
for the person who posed the question.

MODERATOR POLLACK: If there is no fur-
ther discussion, we will terminate this part
of the program.

Froment, R., Blanc, J., Gallavardin, L., and Perrin, A.: An Exact Sign of Massive
Left Auricular Thrombosis: Permanent Disappearance of the Murmur and Thrill

A 30-year-old man with mitral stenosis showed a very loud systolic murmur and
thrill near the heart apex. Between the ages of 36 and 40 there was progressive
diminution of the intensity of the murmur, and between the age of 44 and death at
the age of 48 no murmur could be heard. Signs of cerebral embolization appeared at
the time the murmur began to disappear. Autopsy disclosed pure mitral insufficien-
acy and thrombosis of the entire left atrium with the exception of a channel of about
the same width as the mitral orifice. A second patient, who is still living, showed an
almost identical clinical picture. The disappearance of the murmur is attributed
to the disappearance of a major discontinuity of lumen in the path of the regurgi-
tant blood stream.

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