

THE MINNESOTA SYMPOSIUM ON PREVENTION IN CARDIOLOGY

Reducing the Risk of Coronary And Hypertensive Disease

Edited by:

Henry Blackburn, M.D.

and

Jennifer Willis, M.B.Ch.B.

Laboratory of Physiological Hygiene

University of Minnesota

Minneapolis, Minnesota

Sponsored by:

The Minnesota Heart Association

In Cooperation With:

The Mayo Clinic

The Mayo Foundation

The University of Minnesota

and

The Council on Clinical Cardiology

American Heart Association

May 2-3, 1968

Heritage Hall, Kahler Hotel

Rochester, Minnesota

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Welcome

Ladies and gentlemen, good morning. It is my very pleasant responsibility to welcome you to Minnesota, to Rochester, and to the Mayo institutions. At a time when coronary heart disease has reached epidemic proportions in the western world, it is particularly appropriate to spread the gospel of preventive medicine and to disseminate all available information about reducing the risks of atherosclerosis. We are beyond the stage when this type of information can be regarded as of interest only to the epidemiologist or the clinical investigator. It must be made available to, and used by, the medical practitioner who now spends his time almost exclusively with diagnosis and treatment rather than prevention. And that is what this symposium is all about.

The Minnesota Heart Association is proud to be a cosponsor of this symposium on prevention in cardiology. Dr. Henry Blackburn, chairman of our association's Professional Education Committee, deserves great credit for organizing the program and assembling the distinguished participants from this country and abroad. Others who have worked on the local arrangements are Drs. Kenneth G. Berge, Stewart L. Nunn, and John A. Spittell, Jr. With a "Thank you" to these gentlemen, I once more bid you "Welcome" and turn the podium over to Dr. Henry Blackburn.

John L. Juergens, M.D.*
Rochester, Minnesota

*Former President, Minnesota Heart Association. Associate Professor of Medicine, Mayo Graduate School of Medicine (University of Minnesota), and Consultant, Section of Medicine, Mayo Clinic, Rochester.

Introduction*

THE ANALOGY is far from perfect, but I like to think of the development of the epidemiological study of cardiovascular diseases as occurring in Classical, Romantic and Modern periods, like those of serious music. The Classical Period provided an important base, with classic epidemiological compilation and study of International Vital Statistics on Causes of Death. The apparent national differences found in cardiovascular mortality gave an initial impetus to, and clues for, further study. The Romantic Period was one in which a few distinguished physicians and scientists, led by Paul Dudley White and Ancel Keys, made their peripatetic rounds in many varied and exotic places. They obtained important clinical impressions of cultural differences in the frequency of cardiovascular diseases and focused world-wide attention on the great implications of such differences. The Modern Period is characterized by systematic and long-term studies of cardiovascular manifestations in natural populations, measurement of personal characteristics, and active experimental intervention on factors of risk.

Follow-up study of apparently healthy persons has led to estimates of future risk of coronary attack, based on measurement of predisease attributes. The relative importance of these factors and their interaction are now being elucidated. Here, today, the means and the effects of modifying factors of risk will engage us.

The time is appropriate for a call to greater investigation into the possibilities of prevention of cardiovascular diseases and the wider application of current knowledge in attempts to reduce their toll. It is appropriate to sound this call in Minnesota, where so many have been concerned with preventive cardiology, and in Rochester, where the Mayo Clinic holds the respect of all for its practice of diagnostic and therapeutic medicine.

We are most grateful for the pleasant host arrangements in this quiet, single-minded medical community. We owe these arrangements, and the gracious hospitality of the Clinic, to Kenneth Berge and John Spittell, Stewart Nunn and John Juergens, as well as to Helen Bies of the Minnesota Heart Association. The Mayo Clinic and Foundation, and the staff of the Minnesota Heart Association, are available to assist you.

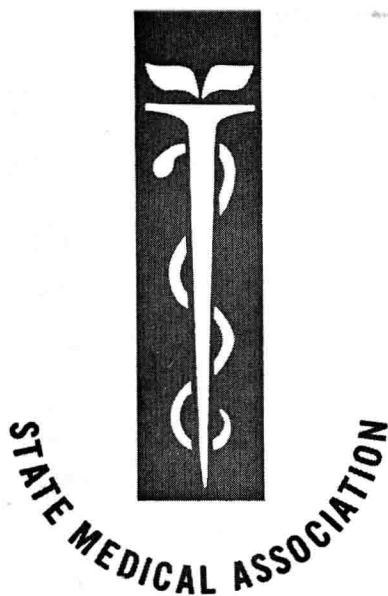
We are pleased now to introduce a distinguished faculty, one which has the richest experience available in the evaluation and attempted reduction of the risk of coronary and hypertensive disease.

Paul Dudley White of Boston, and of the World at Large, once stood alone with a depth of clinical knowledge and a remarkable breadth of understanding of heart diseases. Because he stood strong and with curiosity, vision, and diplomacy, he no longer stands alone but is rather conjoined in a large and important medical enterprise, Preventive Cardiology. Each of us here owes him a professional debt for our own emerging understanding of the distribution, causes, and control of heart diseases. Many more people will come to owe their well-being to Paul White. This is because he so effectively raised the professional challenge of prevention in cardiology and because he provides such a personal challenge to all for living more balanced lives.

Henry Blackburn, M.D.†
Minneapolis, Minnesota

*Presented at the Minnesota Symposium on Prevention in Cardiology, May 2-3, 1968 in Rochester, Minnesota.

†Professor, Laboratory of Physiological Hygiene, University of Minnesota.



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|| Cardiology*

The Challenge of Prevention

PAUL DUDLEY WHITE, M.D.
Boston, Massachusetts

IT IS MY FUNCTION to set the stage for this symposium historically, and perhaps romantically, and to outline some of the major problems of the challenge.

We have doubtless always had cardiovascular diseases and much the same kinds or etiologic types that we have today, but also in all probability in different total prevalence and relative incidence. We can do little but guess how common heart disease was in toto or in each separate kind before the days of autopsies. However, in the Bible and especially in the writings of Pliny the Elder sudden deaths were recorded which were instantaneous enough to have been due to coronary insufficiency and ventricular fibrillation. At other times there were recorded more slowly evolving deaths due quite likely to myocardial infarction.

With the descriptions of post-mortem examinations in the sixteenth and seventeenth centuries we find other cardiovascular diseases identified, especially infected and deformed heart valves, enlarged atria and ventricles, aortic aneurysm, and pericarditis. In the eighteenth century congenital lesions and coronary disease were described, as well as the symptom of angina pectoris and the signs and symptoms of heart failure. Another century went by, however, before hypertensive heart disease came into its own as an important and quite common type.

Heart Disease in the Twenties

When I was a very young doctor we were still in the dark ages; then in the 1920s we began to move ahead trying to learn the overall prevalence of cardiovascular disease and the incidence of the various types.

Infectious Heart Involvement

Rheumatic heart involvement, both acute and chronic, made up about half of all our

*Presented at the Minnesota Symposium on Prevention in Cardiology, May 2-3, 1968 in Rochester, Minnesota.

cases. Syphilis and aortic aneurysms were still common, and hypertension ranked second as an etiological type. Diphtheria was still a potent threat to the myocardium.

The Electrocardiogram

Clinically significant atherosclerosis of the coronary arteries was low on the list, although we had become acquainted with its diagnosis both symptomatically and objectively, especially with the help of the electrocardiogram. Relatively rare in our figures were congenital heart patients who infrequently sought help because none was available. A statistical study by Duckett Jones and myself in the 1920s noted the incidence of congenital cardiovascular defects as 1.5 per cent, while 25 years later in a similar study we found it responsible for 7.9 per cent of 3,000 so-called cardiacs. There were also 3.0 per cent thyrocardiacs, now rarely seen.

Heart Disease in the Sixties

We have made considerable strides, sometimes fortuitously, in the reduction of the prevalence, incidence, and mortality from the infectious types of heart disease, especially the rheumatic, which has dropped far down the scale, though it still exists, especially in parts of the world where preventive measures are few or nil, as in some parts of Central Africa where one sometimes sees tight mitral stenosis in children.

Other infectious types have also been controlled in large part: diphtheria, syphilis, and subacute bacterial endocarditis. Hypertension of certain recognized etiologies and even "essential" hypertension have declined a good deal in the mortality column, under medical treatment.

This leaves two major causes of cardiovascular disease almost untouched, at least so far as their prevention is concerned: congenital heart disease and coronary heart disease. In this program we are concerned with coronary heart disease, but I hope that when we know more about genetics and pay more attention to the health of the human fetus, and even before we have reduced in major degree both the prevalence and the mortality of coronary heart disease, especially in young and middle

aged males, we may begin to plan more of an attack on congenital defects. These may eventually constitute our major cardiovascular challenge.

We must now concentrate on atherosclerotic and thrombotic disease, not only of the coronaries but also of the aorta, the cerebral and renal arteries, and those to the legs, for it is the same process which affects them all: both sclerotic and thrombotic.

Coronary Heart Disease

Why is there a greater challenge now than when I was a young cardiologist nearly 50 years ago? Let me list five of the various reasons for our concern:

Prevalence of the Disease

There has been an increasing prevalence of coronary disease as well as an increased incidence. Austin Flint in 1866 and Osler in 1892 called angina pectoris rare, and they knew the symptom well. I am quite sure that I was as able to determine by careful history in the 1920s as I am in the 1960s the presence of symptoms and signs of the disease, including the diagnosis of angina pectoris, and to recognize the electrocardiographic abnormalities and the collateral signs of acute myocardial infarction long before coronary angiography. I was specializing in the whole field of cardiology as I am now and seeing the same number of, or somewhat more, cardiacs, both new and old. In 1924 it took me two years to see 100 new patients with coronary heart disease in contrast to one year in 1965.

A careful scrutiny of my own ward patients in 1912 and 1913, before we had an electrocardiograph, has revealed very few cases of angina pectoris, and only one case of myocardial infarction found at autopsy. I have also looked over electrocardiographic glass plates of the first hundred of our patients in 1914 and found only one case with typical acute myocardial infarction and one with complete heart block. Another clue to the relative infrequency of coronary heart disease as a clinical problem compared to other subjects like rheumatic heart disease was that in the first 100 of my published papers only two concerned coronary heart disease, whereas from 1946 to 1957 in-

clusive there were 21 such papers out of 99 publications.

The Age of the Coronary Patient

We all have had the experience that of late we have been seeing younger and younger males with coronary heart disease. This was strongly supported by a report by Samuel Levine, who analyzed in 40 families the age of onset of angina pectoris in father and son. He found that the sons began having angina 13 years younger than had their fathers and that their deaths also occurred at a younger age. This fits in with my own experience. I have compared the ages of 100 new patients seen between 1924 to 1926 with those of 100 similar cases seen between 1964 to 1965. There were 5 per cent in the 1960s under the age of 40 compared to 1 per cent in the 1920s, and 21 cases under 50 in the later group compared to seven in the earlier. It was Sam Levine's belief, as it is mine, that something has happened in the last generation which may explain these differences.

Sex

One hundred years ago the wife in the U.S.A. outlived her husband by about one and a half years, but now this advantage has changed to an average of six years. It is clear that atherosclerosis and thrombosis of the coronary cerebral arteries and aorta are responsible, in the main, for this difference. The longevity of the husband must be increased to match, so far as possible, that of his wife. It may be that a shorter male life is a law of nature, but some improvement at least should be possible. In the series of 100 new cases of coronary heart disease of my own there were 72 men and 28 women; of the 21 patients under 50 years of age, 19 were men and only two were women; of the five cases under 40, all were male. In two separate series of 100 cases under the age of 40 years, studied years

ago by Sam Levine and myself, 193 were men and only seven were women.

Heredity

How much of the responsibility is to be credited to the genes and how much to the environment is not known. It is possible that the relative responsibility of these factors may vary from family to family and from person to person. Studies of a few thousand coronary-ridden families might determine what changes in their environment would improve their longevity and health.

Occupation

Years ago when I was a young doctor we had a good many real laborers in this country. Most of their hearts were sound, unless they had had rheumatic fever or syphilis, but I recall that their radial arteries were beaded. It was the affluent white collar executive and capitalist who occasionally had coronary heart disease then. Now the situation seems to be reversed. The 'laborer' no longer labors but owns an automobile and gets the richest cuts of beef and puts on pounds of weight; in middle age his coronary arteries become beaded and occluded while his radial arteries remain smooth.

Meanwhile the executive has become wiser and, with the help of his wiser doctor, tries to keep physically fit and is beginning to benefit. Incidentally, I have been trying to persuade groups of young people in this country and overseas to rebel not against their parents but against their parents' bad habits. Sometimes I am viewed with concern as a Don Quixote sparring with windmills; but my experience over a long life in seeing and following up a great many patients makes me believe that the next generation may benefit from our efforts, though it may be difficult to rehabilitate the fathers, even with transplants.

Smoking

A custom loathsome to the eye, harmful to the brain, dangerous to the lungs, and in the black stinking fume thereof, nearest resembling the horrible Stygian smoke of the pit that is bottomless.—James I of England

Our models are lenses, coloring the world with as many different hues.—Emerson