PREVENTIVE CARDIOLOGY

Edited by
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Preventive Cardiology

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Edited by

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Foreword

On the official WHO-list of age-specific death rates for cardiovascular diseases Sweden is rather down the scale in Europe. When we consider the major causes of death however cardiovascular diseases, dominate the picture. Therefore it is natural to take measures in an effort to control these very common diseases. An important step was then the establishment of a Planning Group in Preventive Cardiology under the auspices of the Swedish Medical Research Council. The aim was to start heart control programs in different areas of Sweden.

In association with the Council on Epidemiology and Prevention of the International Society of Cardiology the Planning Group organized this symposium on Preventive Cardiology.

The proceedings of the symposium are published with the feeling that their usefulness will not be limited to those interested in one particular report. It is hoped that the volume also will attract those readers who are interested in the general problem of benefits and drawbacks of prevention of heart diseases.

As the organizers of the symposium it is our privilege to thank all those having made possible this conference and the publication of the proceedings. Our sincere thanks are directed towards the contributors to this volume and to our collaborators at the Section of Preventive Cardiology, Department of Internal Medicine, Sahlgren's hospital, Göteborg, who all supported the enterprises in connection with this meeting. (They are Drs. Lars Wilhelmsson, Dag Elmfeldt, Anders Vedin and Claes Wilhelmsson.) The Swedish Medical Research Council and "National Association against Heart and Lung Disease" have generously sponsered the conference. Mrs Inga-Lisa Ljungberg has been our most efficient secretary at this meeting.

G. T. L. W.

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Welcoming address

by Lars Werkö

The Council of Epidemiology of the International Society for Cardiology has made a concentrated effort to increase the knowledge regarding cardiovascular epidemiology all around the world. One very important part of this effort has been the yearly courses of epidemiology that has seen held in August or September in some parts of Europe. This year Sweden was selected as host for this course and we deemed it natural to grasp the opportunity and arrange a conference on preventive cardiology where we could update the situation having so many eminent scientists from all over the world with us.

In this setting I do not need to dwell on the importance of the problem facing us. Most of you have been so deeply involved during so many years that you may be somewhat astonished to hear that the Swedish authorities have not quite understood the magnitude of the impact of the cardiovascular diseases on the society, both outside the hospitals and for the clinical medicine proper, until lately.

However, two years ago the Swedish Medical Research Council created a committee on preventive cardiology with the aim of increasing and coordinating the efforts of combating cardiovascular diseases in the society, especially ischemic heart disease. This conference is sponsored by this committee as well as by the Swedish Association against Heart and Lung disease. The whole project had not started had it not been for the efforts of WHO, that especially in Europe has had a comprehensive programme running for the last couple of years. Much of the presentations today will center around that programme.

The real missionaries for the cause of international cooperation within this field are also with us during this time and it gives me great pleasure to welcome Ancel Keys and Jerry Stamler among us. Without their dedicated work neither international cardiovascular epidemiology nor the ISC Council had occupied the firm position it now has. The present chairman of the international council. Jerry Morris, is representing the council but to a much larger extent himself and the British Medical Research Council. Similarly we are happy to have amongst us Zbynek Pisa, who has been of such importance for the European WHO Control Programme.

I have mentioned a few people in this introduction. This does not mean that you are not all equally welcome. We are very happy that you all have

been able to come and hope that you will take part in to-days deliberations with the same intensity that I am quite sure that for example Jerry Stamler will do. So let's start with our formal programme.

You may think that we have been a little provocative using as title for the coming panel discussion "risk factors, cause or effect?" This was done in order to have a good starting point for a real debate not only of the import of serum cholesterol and high blood pressure, cigarette smoking and physical inactivity in predicting the future event of myocardial infarction but also of factors like psychosocial instability, status incongruity, personality types and other less studied variables.

There are several problems that still need solution—some of them even a well formulated hypothesis. Let me list a few that we may address us to.

Is the serum cholesterol the best predictive factor for IHD?

And is the reduction of serum cholesterol the most important presentive measures that we should aim at?

What about other lipids?

What is the situation of the "water story"?

How does smoking cigarettes enter the predictive picture in different part of the world?

What is the significance of psychosocial circumstances? And do they act by themselves—through neural or humoral pathways—or through more ordinary metabolic mechanisms?

Is the final catastrophe of a myocardial infaction due to *one* or to a set of factors acting throughout a life time? Or is there one set of events leeding to vascular disease and another acting on top of the former to release the killing blow to the myocardium?

Has the day come for a general preventive effort? If so, how should this be organized?

You can comprehend that we have quite a lot to discuss so let's start with some facts collected in several international or national studies.

The challenges and possibilities for prevention in mass community efforts to control the major coronary risk factors

By Jeremiah Stamler

The challenges and possibilities inherent in mass screening to detect coronary-prone persons—the implications for the effort to curb the coronary epidemic—can be fully comprehended and appreciated only when considered in the light of specific and concrete facts. In my later presentation, I will cite the data available from the U.S. National Cooperative Pooling Project on the impact of the three major coronary risk factors, singly and in combination (1, 2, 2a–l). It is relevant to our present theme to examine the data once again, this time in terms of the potential for prevention. While this is U.S. experience, it clearly is meaningful for other countries, at least the industrialized ones.

Let us examine the risk factors one by one, beginning with hypertension. At the present in the USA of all the hypertensives—totalling 20 to 25 million persons—almost half are undetected. Of the half who realize they are hypertensive, about half are receiving no treatment. Of the remaining quarter, about half are being treated inadequately, so that they remain hypertensive (3–7). Given this situation with regard to undetected, untreated, and inadequately treated high blood pressure, the rough estimate is that only about one-eighth—1/2 x 1/2 x 1/2—of the U.S. hypertensives are being given sufficient treatment to lower their blood pressure appeciably, i. e., to diastolic level less than 95 mm. Hg.

Let us assume that the level of effective treatment can be increased to embrace 50 per cent of the hypertensives, and let us explore the possible consequences, in terms of mortality from coronary heart disease and all causes for men age 30–59 of the type studied in the Pooling Project. The age-adjusted ten-year CHD death rate for these men with diastolic pressure $\geqslant$ 95 mm. Hg at entry was 54 per 1 000; the all causes mortality rate, 109. Let us assume that the CHD mortality rate of the well-treated hypertensives can be lowered from 54 to 30 per 1 000, a reduction of 44,4 per cent. This rate of 30 per 1 000 is still above that of the normotensive subgroups (26 and 20 per 1 000). From the limited experience of the U.S. Veterans Administration Cooperative Study on Antihypertensive Agents, this is a reasonable estimate of effectiveness of therapy (1, 8). It is reasonable to make a further assumption as to effectiveness of anti-

hypertensive treatment against cardiovascular mortality overall—e. g., against stroke, in addition to CHD—so that all causes mortality for the well-treated hypertensives is lowered to 70 per 1 000. This would mean a lowering of the all causes death rate of 35.6 per cent for the hypertensives, once again, a reasonable assumption (1, 8). For the total population of men age 30–59, the net result of effectively treating half the hypertensives (i. e., just under 10 per cent of the total population) would be a reduction in overall CHD mortality rate of 6.3 per cent, from 32 per 1 000 to 30 per 1 000, and a reduction of all causes mortality rate of 5.6 per cent, from 72 to 68 per 1 000. Lest these decreases seem insignificant, I hasten to add that no improvement in life expectancy for middle-aged white American males has occurred in the 20th centuty (9). A 5.6 per cent reduction in all causes mortality for the 35 million U.S. men age 30–59 would mean saving of about 14 000 lives per year!

A similar set of calculations can readily be made as to the estimated gains from a successful effort to reduce the proportion of men in the population who are cigarette smokers. Let us estimate the consequences of converting 30 per cent of current cigarette smokers, again men age 30–59, to ex-smokers, with a ten-year CHD mortality rate reduced to 24 per 1 000, and an all causes death rate to 46 per 1 000, rates for men smoking pipe and/or cigars only. For these reformed cigarette smokers, this would represent a decrease in CHD mortality rate of 40.0 per cent, and in all causes mortality rate of 50.0 per cent. For the total population, this would be a decrease in CHD mortality rate of 12.5 per cent, and in all causes mortality rate of 11.3 per cent. This would mean an annual saving of about 30 000 lives among the 30 million U.S. men age 30–59. No wonder that public health experts have characterized a successful mass campaign against cigarette smoking as the single most important thing that could be done to improve life expectancy in the industrialized countries.

Let us repeat the calculations, now estimating the fruits of a successful effort to identify 50 per cent of U.S. hypercholesterolemic men ($\geqslant 250$ mg./dl.) and lower their serum cholesterol by 10 per cent by safe feasible nutritional procedures (1, 10–12). Previous calculations permit the estimate of a 24.4 per cent lowering of CHD incidence from a 10 per cent reduction in serum cholesterol (1, 14). Let us assume a similar effect on CHD mortality rate. The impact on all causes mortality rate for the 50 per cent of hypercholesterolemic men so influenced nutritionally is projected to be 6.9 per cent, assuming no impact on any other type of atherosclerotic mortality (e. g., stroke) other than CHD (1, 2, 2a–l). For the total population, the net effect on CHD mortality rate would

be a reduction of 6.1 per cent, and on mortality from all causes, 2.9 per cent, with a saving of over 7 000 lives per year.

The inference from the prospective epidemiologic studies is that an across-the-board improvement could be attained by fat modification of the diet to lower serum cholesterol of the whole population (1, 2, 2a–l). The National Diet-Heart Study unequivocally demonstrated that such a reduction in serum cholesterol is feasible (11). It is therefore worthwhile to estimate what the effects on mortality rates would be if various proportions of the general population of adult males changed diet habits enough to lower serum cholesterol 10 per cent. Again, let us take the estimate that a 24.4 per cent reduction in CHD mortality would result, with no effect on mortality from severe atherosclerosis of other arterial beds. From the data, it can readily be calculated that if 25 per cent of U.S. white males age 30–59 were to make these changes, the net effect on CHD mortality rate for the population would be a 6.1 per cent reduction, and on all causes mortality rate, a 2.9 per cent reduction. If 50 per cent of U.S. white males age 30–59 were to make these changes in diet habit and serum cholesterol, the net effect on CHD mortality rate for the population would be a 12.2 per cent reduction, and on all causes mortality rate, a 5.7 per cent reduction, with a saving of over 14 000 lives per year.

Finally, let us assess the preventive potential of combined intervention against the three major risk factors (hypertension, cigarette smoking, hypercholesterolemia) in highly coronary-prone men with any two or all three of these dangerous traits. Obviously such an approach has a great deal to recommend it; it makes great public health sense, theoretically and practically (1, 10, 12). Again, let us use the Pooling Project experience as our guide. Note the typical American situation, product of our way of life; only 1 249 of the 7 342 white males age 30–59 at entry –i. e., only 17 per cent–were classified not high for all three factors. All the rest had one or more risk factors; 45 per cent with one, 30 per cent with two, 8 per cent with all three. These latter two subgroups, with any two or all three risk factors (38 % of the total group), accounted for 57 per cent of coronary deaths, 55 per cent of all deaths.

By multifactor intervention, it is reasonable to anticipate a substantial reduction in mortality rates for these very high risk men. The estimates are based on the assumption that both CHD and all causes mortality rates can be reduced by over 50 per cent, to levels slightly below those for men with one risk factor only, but still substantially higher than those for men with none of the three traits. If these changes were successfully accomplished for half these very high risk men, i. e., for about 20 per cent of the population, this would yield an 18.7 per cent net

reduction in CHD mortality and a 17.8 per cent reduction in all causes mortality for the total population, with a saving of almost 45 000 lives per year.

These would indeed be substantial achievements, a major turn in life expectancy for U.S. adult males. These then are the stakes–the challenges and possibilities for primary prevention and public health advance–in the projected mass community efforts to control the major coronary risk factors.

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