Lessons from falling coronary heart disease mortality in the United States

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Summary

The marked decline in coronary heart disease mortality in the United States since the late 1960s is most likely due in large part to successful efforts at primary prevention, attributable in turn to improved eating habits, better control of blood pressure and a reduction in smoking. Another part of the decline can probably be explained by an improvement in prognosis. It remains to be established if there has been a reduction in morbidity as well as mortality. There are other countries which have experienced declines, though of a lesser degree, while several countries have recorded increases in coronary heart disease mortality. These downward and upward turns, in general, are likewise compatible with the theory that they are caused by corresponding favourable or unfavourable changes in life styles.

Few events during the past years have created as much stir as the entirely unexpected decline in mortality from coronary heart disease (CHD) in the United States since the late Sixties, which was first recognized in the mid-Seventies. Incidentally, the downward trend is still continuing. A conference to explain this trend was convened in Bethesda in 1978 (Havlik and Feinleib, 1979). Taking a bird's eye view of the proceedings, there was a high degree of unanimity that primary prevention must have influenced this development but that the additional improvements in medical care, i.e. secondary prevention, could by no means be excluded. Actually, it was somewhat surprising that the clinical cardiologists present at the meeting did not promote secondary prevention as might have been expected. The events in the United States have stimulated a systematic look at the trends in other countries (Epstein and Pisa, 1979; Epstein, 1983), culminating in a major international effort, co-ordinated by the World Health Organization, to monitor and try to explain coronary heart disease mortality and morbidity trends on an international scale prospectively over

time—the 'MONICA' project (Pisa, 1982). Parallel activities on a co-ordinated cardiovascular community surveillance are carried out in the United States, following up on the observations which originated in that country.

Background data

As a point of departure, it is recalled that the mortality from coronary heart disease between 1968 and 1978 in the United States in terms of age-adjusted rates has declined by 25% for white men, 27% for white women, 24% for non-white men and 38% for non-white women. The declines in each of these four groups have been markedly greater in younger rather than older age groups (Havlik and Feinleib, 1979; Thom, Kannel and Feinleib, 1983). Among men, somewhat lesser but still substantial declines have been observed in Australia, Japan and Israel and also, on a smaller scale, in Canada, New Zealand, Norway and Belgium (Epstein and Pisa, 1979; Epstein, 1983; Thom, Kannel and Feinleib, 1983). The international implications of these findings will be dealt with later, after reviewing the situation in the United States.

The case for primary prevention

There is no question that the major coronary heart disease (CHD) risk factors have become less frequent in the United States during the time mortality has been declining (Havlik and Feinleib, 1979). Serum cholesterol levels have decreased on the average, if only slightly, but the percentage of persons with levels above 260 mg/dl has decreased by 13% among men and 23% among women between 1960-1962 and 1971-1974 respectively. Hypertension has been detected and treated more efficiently so that the prevalence rates for hypertension for ages 25-74 years dropped by some 2% amongst whites and between 4-8% amongst blacks, while current smokers above age 20 years declined by some 25% amongst men and 14% amongst women between 1965 and

1980 (Thom *et al.*, 1983). There remains a lingering question if those changes could have sufficed to produce so profound a change in mortality but a calculation, based on a multiple logistic function estimate derived from the Framingham Study data, suggests that the *predicted* effect of the observed risk factor changes on CHD mortality is of the same order of magnitude as the *observed* change in mortality, as reported already at the Bethesda Conference in 1978. Indeed, the increasing emphasis on the 'mass strategy' of coronary heart disease prevention (World Health Organization, 1982) is based on the premise that the cumulative effect of relatively small risk factor changes may be great, that 'a little goes a long way ...'.

Nutrition plays a key role in CHD prevention. The changes in dietary habits which have taken place in the United States in the 1970s and earlier, are in the direction which would be expected to lead to a reduction of serum cholesterol levels (Havlik and Feinleib, 1979) and there is a correspondence between predicted (from the Keys or Hegsted formulae) and observed changes in serum cholesterol, using the observed changes in eating habits as a base (Liu *et al.*, 1982; Arteriosclerosis, 1981). In absolute terms, between 1963 and 1980, milk and cream consumption decreased by 24%, butter consumption by 33%, animal fats and oils by 39%, while the consumption of vegetable fats and oils rose by 58% and fish by 23% (Walker, 1983). These are big changes and it would, indeed, be strange if they were inconsequential as far as the metabolism of fat is concerned.

The changes in risk factors and eating habits which have been described are averages, which presumably do not apply equally to all segments of the population. Their impact could be assumed to be greatest if they applied mostly to the social groups with the highest incidence of CHD. However, it is probable that the changes were most marked among the higher social classes who show the lowest frequency of the disease. Nevertheless, it could be argued that the effect in those groups might be so great that it overshadows whatever goes on in other classes, thus strengthening the inference of a causal relationship. Regrettably, in the United States unlike in Britain, mortality statistics do not lend themselves to an analysis by social class. Life insurance data do, however, suggest, that the decline in CHD mortality in the United States has been greater in the higher social classes (Metropolitan Life Insurance company 1979).

There are a few studies in the United States which address directly the question whether the incidence of acute myocardial infarction including both mortality and morbidity has changed in the 1970s. The Kaiser-Permanente Study, based on hospital admissions, suggested that the incidence had, indeed,

declined (Friedman, 1979) but the Rochester, Minnesota, Study indicated that, in this city, the incidence of acute myocardial infarction has remained unchanged (Elveback, Connolly and Kurland, 1981). Likewise, no change could be detected in Framingham (Kannel and Thom, 1979). The Du Pont Study on the other hand, indicated clearly that incidence had decreased in this industrial population (Pell and Fayerweather, 1982). The evidence, therefore, is inconclusive.

Before leaving the subject of primary prevention temporarily, it must be recalled that post-mortem studies in New Orleans have provided evidence that coronary atherosclerosis did become less common in the 1970s, as compared with earlier decades (Strong and Guzman, 1980). Though the representativeness of post-mortem data is always open to question, the magnitude of the observed change is such that it cannot be dismissed. Up to this point, the data at hand make a plausible case in favour of the view that mortality from CHD in the United States declined because there was a decrease in the factors predisposing to it. The case would be strengthened if it could be shown that there was no concomitant improvement in the survival of myocardial infarction patients while in hospital during the acute phase or after their discharge. The available evidence will be considered in terms of secondary prevention.

The case for secondary prevention

Concerning survival during the first month after the acute onset of acute myocardial infarction, there are four studies which report an improvement in prognosis (Elveback *et al.*, 1979; Kannel and Thom, 1979; Pell and Fayerweather, 1982; Nanas *et al.*, 1982). In two studies, the prognosis during the first month was unchanged (Friedman, 1979; Goldman *et al.*, 1982). The National Hospital Discharge Survey (see Thom *et al.*, 1983) similarly provided no evidence that discharge rates from myocardial infarction had changed for men or women at any age during three time periods (1972, 1975, 1978). On the face of it, this would mean under a number of assumptions that the incidence of acute heart attacks has remained the same between 1972 and 1978. Amongst several alternative interpretations, it would be conceivable that incidence has, indeed, diminished so that fewer patients are admitted but that, due to better care, a correspondingly greater number are discharged, leaving the discharge rate unaltered.

It is evident that it cannot be stated with reasonable assurance whether or not the prognosis during the first month after myocardial infarction has improved during the 1970s. Four studies are in favour and three are against but, among the latter is the careful and representative survey of essentially

the entire Boston metropolitan area. There are three studies which deal with the prognosis past the first month and in none of them is there any indication of improvement (Goldberg *et al.*, 1979; Elveback *et al.*, 1981; Weinblatt, 1982).

Primary or secondary prevention: which is at work?

With the present gaps in knowledge and the inconsistencies in the evidence, it is not possible to say with certainty whether the decline of mortality from CHD in the United States in the last 10–15 years has been due mostly to a reduction in the incidence of the disease, i.e. primary prevention, or to an improvement in prognosis. On the other hand, the available evidence is more than sufficiently compelling to state that primary prevention *must* play a substantial role. Assuming various degrees of reduction in incidence and mortality, it is possible to calculate how different 'admixtures' of these two forces would affect the death rate (Epstein, 1983). If, for example, incidence and mortality both declined by 10%, the calculated decline in the death rate over a 10-year period among middle-aged men would be a little under 20% and thus within the observed order of magnitude of what has actually happened.

It would be unfortunate at this stage to play, as it were, primary and secondary prevention against each other. On the basis of what is known already, it is most unlikely that an improvement in prognosis alone could account for the profound change in CHD mortality which has taken place. In order to reduce overall death rates among middle-aged men by 25% through improvement of prognosis alone, mortality during the first month would have to be reduced by 30% and the mortality among the survivors by a quarter. This is not unthinkable but very unlikely to have happened during the 1970s on the basis of all the available evidence! By the same token, a reduction in incidence alone by 20% might have reduced overall mortality by 25% which is not unthinkable either, if one assumes that the relationship between risk factor reduction, and reduction in mortality is totally causal, recalling the calculation based on the multiple logistic function discussed earlier. While this may be approximately true, it would not be easy to substantiate this assumption scientifically to an entirely satisfying extent. In order to avoid unnecessary and unproductive controversy, it will be scientifically and tactically preferable to give both primary and secondary prevention their due in having brought about the most welcome downturn in CHD mortality.

Links between cardiovascular and other chronic disorders

Sight is sometimes lost of the fact that the decline

of CHD mortality in the United States was accompanied by a reduction of most other major causes of death (Thom *et al.*, 1983). Indeed, there is much reason to believe that the preventive measures which protect against CHD have a favourable influence on the prevention of disease and the preservation of health (Epstein, 1982; Epstein and Holland, 1983). This consideration adds weight to practical lessons to be drawn from the experience as far as CHD is concerned.

Furthermore, neither the trend data from the United States nor those on an international scale lend any support to the notion, sometimes expressed as part of the discussions concerning the relationship between low serum cholesterol levels and cancer, that the dietary measures advocated for the prevention of CHD might increase the risk of cancer. On the contrary, as a rule, the trends in heart disease and cancer go parallel or rises in mortality from heart disease are associated with only minimal trend changes in mortality from cancer other than lung (Epstein, 1983 and unpublished data).

Lessons to be drawn: an international view

It is clear that there have been changes in the environment and living habits in the United States which have diminished the burden of coronary heart disease in the population in terms of mortality. It is as yet unknown if there have been concomitant changes in morbidity but this would seem likely if a major part of the decline can be attributed to primary prevention, also shown to be most likely. The only question is whether changes in nutrition in the direction of improving serum lipid patterns, better control of hypertension and less smoking can be held largely responsible for the change in mortality. This is indeed highly probable unless one is willing to take recourse to mysterious forces which have hitherto escaped recognition. Possibly, stronger social support systems affect health favourably, but there is no adequate reason to think that such a shift has been at work in the United States. In spite of all these positive pointers, it would be hazardous to accept them if they were inconsistent with the international picture. While the case for positive alterations in living habits is less strongly supported in other countries with downward trends, available evidence points in the same direction, specifically in Australia (Dwyer and Hetzel, 1980), New Zealand (Beaglehole *et al.*, 1981) and Israel (Palgi, 1981). There is a need for information from Canada and the data from Japan are difficult to understand at face value. In that latter country, however, the absolute, as opposed to the percentage change, is small because mortality rates are so low and the old and vexing problem of

changes in the fashions of death certification cannot be excluded.

For consistency, not only the downward but also the upward trends require explanation. In most Western and Central European countries, these increases in the 1970s have been modest and are not too difficult to understand, considering that, in general, efforts at coronary heart prevention have either not been consistent or have not spread to the population at large. The greater economic well-being, going along with more widespread overnutrition, presumably accounts largely for the steep upward trends in Eastern European countries. The upward trend in Sweden supposedly takes place in spite of improvements in life style, similar to the United States (Welin *et al.*, 1983) but for diseases with multiple aetiology, epidemiological evidence does not require total consistency for the acceptance of a plausible theory. The Finnish experience is consistent with theory since the recent downward trends in this country with initially very high rates can be adequately explained in terms of successful preventive efforts (Pyörälä and Valkonen, 1981). The British experience is discussed in detail elsewhere in this publication (Marmot, 1984).

Conclusion

During World War II, R. A. McCance wrote a remarkable sentence in a mimeographed report dealing with salt balance and survival at sea: 'For those who have faith in their physiology, preventive measures speak for themselves'. The CHD mortality experience in the United States and also in other countries is but one piece of a large amount of evidence that the recognized risk factors for CHD are causally related to the disease and that their control has preventive value. The current wave of attacks against the risk factor theory is due in part to the fact that people dislike being overwhelmed, in this case by a compelling weight of scientific findings. There are, of course, other and more important reasons for a widespread reluctance to accept or act upon the preventive lessons from three decades of atherosclerosis research. Such reluctance may be relatively less pronounced in the United States, being probably one of the explanations why modifications in life styles are more widely practised in that country. This is more than ever the time, to paraphrase McCance, to have faith in all that has been learned about the causes of atherosclerosis over the years and let the resulting preventive measures speak for themselves.

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