Some Aspects of Prevention of Coronary Artery Disease*

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Introduction

LADIES & GENTLEMEN, I consider it a great honour to have been invited to deliver this lecture before such a distinguished gathering, The subject of my talk this morning is "Some aspects of prevention of coronary heart disease". As you are all aware, the whole subject of prevention is an immense one, and cannot justly be covered in 20 minutes. I shall, therefore, only highlight some of the impressions I have had over 20 years, practising cardiology.

About 10 years ago I met an old friend, a prominent cardiologist, at a conference like this, and during a discussion on the management of coronary artery disease he commented, "I fear that our therapeutic measures are more impressive than helpful to our patients. We should consider this whole problem in terms of preventive cardiology and seek to obtain the help of the paediatricians".

I recall pondering over these words at the time, because it appeared strange, coming from him, a practising physician like myself, whose philosophy and training have traditionally involved attempting the cure of a disease as and when it presented itself. Yet it was true, that though we saw hundreds of patients with the disease, and our hospital statistics were beginning to show this to be the leading cause of death and distressing morbidity, we were adopting a stereotype attitude which underscored our helplessness. Many of us did realise that there were many unsatisfactory aspects about our management, but we consoled ourselves that we were doing the best under the circumstances, and no other physician could do more.

Over the last two decades, as a result of intensive work in the Laboratories and in the field, it has become quite clear, that what we were seeing in our patients, was the end-stage of a disease process, that started many years earlier. We have come to realise that moderately advanced lesions appear to be obstinately irreversible, and cannot regress, despite the intense application of current methods of therapy. It was also clear that for every patient who developed symptoms, there were hundreds more who showed no overt clinical evidence of this dangerous disease.

Our performance record, also in terms of salvage of lives of these patients was a dismal one, in spite of rapidly improving medical and surgical capabilities, often involving the application of expensive technology in coronary care units and other areas.

Logically then, our attitudes and our emphasis had to move towards those areas in time, when control or manipulation of probable causes may be effectively applied. I hope to deal in this paper, within the time available with the question of primary prevention of atherosclerosis, by which I mean, the attempt at control of those aspects of the disease that begin early in life, and are probably responsible for the later serious coronary artery lesions in adult life. It would be impossible to provide a simplified formula, or a dognatic programme, for general application. And as I proceed,

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it may be obvious to you, that the recommendations I make, would be coloured by my own experiences and philosophy, in dealing with this problem. For I speak, not as an epidemiologist or a public health worker, but as a practising cardiologist.

First, we need to ask our pathologists if they can tell us when the initial lesion, if any, begins. To me, one of the most amazing aspects of the anatomy of the coronary blood vessels is that their location, unlike that of any other artery elsewhere in the body, causes them to undergo twisting, turning, buckling and telescoping, with every single heart beat, so many thousand times per day. I cannot think of any metal, or man-made tube, that could withstand this type of pressure for more than a reasonable length of time. There must be little tears or injuries that occur over the many years of a life time. But repair and regression does occur, in an orderly fashion. We are told by some, that atherosclerosis is a result of a possible abnormal reaction to such injuries.

In the view of most investigators, the evolution of the adult plaque starts from fatty streaks, probably in the second decade. They can reduce in size or even regress, to judge from experimental lesions. The critical time of life, for such possible regression, appears to be towards the end of the second, or perhaps, the third decade. In the presence of certain known risk factors, the critical point in the evolution of atherogenesis appears to shift into the paediatric years. Hence, all measures to control or reduce the factors that fuel the progression of such lesions, would have to take into consideration these age groups.

To me, there appears to be a strong genetic factor in the causation of coronary artery disease. In the words of Sir William Osler we inherit a 'tube', by which he means the vascular tree.

We have considered marriage guidance, for genetic reasons, for people with thalassaemia and other genetically transmitted disease – why not then, for possible risks of coronary heart disease? Perhaps in the light of modern knowledge, we should advise against intermarriage amongst families, in whom there are two or more coronary risk factors, and we should strongly discourage families, with hereditary hyperlipoproteinemias from intermarrying into similar groups.

Perhaps there is something to be said in the old fashioned practice of "arranged marriages" in this part of the world, where the background of prospective partners, both socio-economic and medical are studied by the elders of the families concerned, to see if there is "compatibility". It is common knowledge, that these family elders, scrutinise the family histories of the intending bride and bridegroom carefully, looking for diseases like diabetes, hypertension and so on. It would, thus, appear unfortunate, that as western influence erodes further into our society, this practice becomes less common.

Since the observations of the Framingham Project, much data has been extracted by so many different groups, and so many statements made, that I wonder sometimes if a state of confusion is produced not only the lay public, but also to doctors.

Briefly however, to look at these studies positively, significant contributions have been made, establishing an association between certain characteristics of individuals and environmental factors in the development of atherosclerosis. These "risk factors" have been evaluated as indicators of atherosclerosis, and the generally accepted view is that their elimination will alter the course of the disease.

A careful look at these risk factors reveals that, though much remains unknown, the etiological relationship of *elevated serum lipids, cigarette smoking, hypertension, diabetes* and *obesity* in coronary heart disease can no longer be doubted. The applicability of adult risk factors to children, at present rests on *belief,* but not on absolute proof: *belief* that life-long reduction of serum lipids and hypertension, will carry with it reduced risk of atherosclerosis: *belief* that childhood habits of *diet, exercise* and *abstinence from smoking* will carry over into adult life. Thus, primary prevention will call for the identification of a particular type of child and the young adult, at risk.

Such a search implies a more thorough form of medical examination, including attention to family history. There should be at least one medical examination of all children at primary, secondary and tertiary levels of education. This should also apply to those seeking employment. It is surprising that, although, these are usually done in most countries, nobody has given serious attention to estimation of serum lipids, the blood sugar and the serum uric acid, in these medical examinations, especially when all that is required is only one specimen of blood. The possible expense and value, in terms of cost benefit in developing countries, like ours, for any type of screening, has to be considered, but to me this is not prohibitive. These are practical, albeit, very necessary measures, if we are at all serious, in tackling the very grave problem of coronary artery disease. Biochemical and other disorders, detected at these examinations should be

treated on an individual basis. The long term effects of such therapy on the growing child cannot be known with certainty. It may also carry implications we have so far not considered, because our ideas of abnormal values in young age groups, is not well established. We must also remember that the consequences of diagnosing an abnormality, could affect career choices, employment possibilities and even insurance premiums.

Inspite of the tremendous work done, and the evidence that has been gathered, as to the role of saturated fat and cholesterol in the incidence of coronary artery disease, it is shocking to note that most nutrition programmes in our countries, advocate a diet that encourages the development of atherosclerosis. Perhaps our public health colleagues should be orientated towards current views on nutrition.

There have also been recommendations, for a general change in diet for the entire childhood population. Laboratory and field studies leave no doubt, that we could lower cholesterol and lipid levels, and control obesity, by diet alone. Such a general change in diet, as opposed to a selective change, for those with abnormal lipid levels and other metabolic abberations, is based on the belief, that at the adult level, established habit patterns are notoriously difficult, if not impossible, to change.

To me the best results are obtained, if health education on the role of an appropriate non-atherogenous diet, is aimed primarily at the mother and housewife, who has the greatest influence on the education of their children and their diets. In this connection, radio programmes boomed into the kitchen, often produce salutary effects. I hold the belief, that no serious harm can result, if we advocate for our people, a commonsense diet – i.e. a diet moderate in calories, moderate in total fats, moderate in polyunsaturates, and low in saturated fats and cholesterol. Further, I feel there is more than sufficient evidence to show, that advocacy of such a diet from infancy or early in life, will contribute to good health generally, and lower the incidence of coronary artery disease.

I have over the last 10 years or so, talked to student groups, civil servants' organisations, teachers' institutions, Rotary Clubs and so on, on the importance of a non-atherogenous diet, so much so, that in the city where I live in, particularly everyone is now on low animal fats and on moderate calories. Practically every family you ask in Kuala Lumpur will tell you, that they use corn oil or sunflower oil for cooking, instead of coconut oil, which is much cheaper, and has been for centuries the cooking oil of choice in Asia. Kuala Lumpur has become now so medically sophisticated, that patients come to my clinic and demand, an estimation of fasting lipids. In fact, estimation of fasting lipids, blood sugar and serum uric acid, has now become a routine procedure in the annual examination of all government servants over the age of 40. In addition an electrocardiograph is always included for the 40+ groups. We have also to persuade our public health colleagues not to be unduly preoccupied with infections and communicable diseases – in view of the rising incidence of coronary artery disease.

We must also realise that, in a consideration of the risk factors in the natural history of coronary artery disease, some important pieces of information are lacking. . *The first,* is what causes coronary artery disease in the 20 – 30% of patients, who during life, lack any of the major risk factors. Secondly, why is it that women who were supposed to be relatively immune during their reproductive lives are now beginning to show an increased incidence of the disease?

With regard to the first question, I recall one day telling the wife of one of my patients, that we have found none of the currently associated risk factors in her husband, who had just suffered an acute myocardial infarction. She replied, 'I told you right from the first that you would find nothing in all the tests that you have done. If you really want to know what has caused this heart attack, I will tell you'. I asked somewhat patronizingly, what this was, and she replied quickly 'it is stress, the stress of his work, stress of his social obligations and even the stress of his pastimes'.

There is no doubt that many of us do have patients, with a kind of aggressive, driving personality, that we believe are the characteristics of a successful person in modern society. These are individuals with excessive competitive drive, aggressiveness, impatience, and a sense of time-urgency. It is highly likely that such behaviour influences currently known risk factors.

Friedman has called this, the "Type A" personality and makes out an interesting and provocative thesis to show its major role, as an important risk factor. I feel we must include this type of personality, and the stresses of modern living, into our repertoire of risk factors. I have always believed that hard work never killed anybody, but we are developing into a group of individuals with the inability to relax in between such hard work. Although it is difficult to quantitate stress or psychological factors, in relation to coronary artery disease,

most of us have seen ample evidence of its role in our clinical practice. There is no doubt in my mind that the stresses and tension of modern living, in our urban society, are significant contributory factors, and that our contemporary mode of living, with its pace and turmoil, have additive effects on other risk factors.

With regard to the second question why there has been an increase in coronary artery disease in women of reproductive age, I suspect that the oral contraceptive pill may have a large role to play. While not decrying the immense amount of benefit that has resulted from its use, I cannot in all honesty believe, that the use of progesterogens and oestrogens for such a long period of time, is without serious harmful effects. Over the last 7 or 8 years I have seen women in the reproductive part of their lives with coronary artery disease who have had no other risk factors other than the fact that they have been on the 'pill' for many years.

I feel that the role of endocrine and sex hormones in the pathogenesis of coronary artery disease requires closer scrutiny, and much more research needs to be done. Besides, the 'pill' is not the answer to family planning. It would perhaps be preferable, to ask our young women to have the number of children they require in early life, where the risk of congenital defects in the offspring is much less, and then have their tubes ligated, instead of being for practically a lifetime on sex hormones.

In the short time available I have discussed some aspects of the prevention of coronary artery disease, largely from the experience I have gained and the impressions I have formed over a period of 20 years. I have proposed *medical examinations* or screening of children and adults at certain definite intervals.

I have suggested *genetic counselling,* in predisposed groups, and those with increasing number of risk factors. I have advocated *health education* of our public health personnel, our mothers and housewives, and our children, on the value of an *non-atherogenous diet*. I have made my observations as a clinician on the role of stress and tension as strong contributory factors, despite a lack of concrete scientific evidence. Lastly, I have made a plea to make a close look at the oral *contraceptive pill* as a contributory factor for the increase incidence of coronary artery disease in women.

At this juncture, I would like to sound a word of warning. If we are to prevent in this part of the world, the epidemic of coronary artery disease which has been ravaging the West, a *well planned* and *well organised,* preferably *government-sponsored* effort should be made now. We cannot afford to wait for 100% scientific proof. There is more than enough experimental and epidemiological evidence for us to justify embarking on a widespread social effort to curb this dreaded menace. The prevention of coronary artery disease will be the *No. 1 medical problem of the century* and is bound to tax the ingenuity and wisdom of the medical profession to the limit. The time to act is *now.* In the words of Samuel Johnson "Nothing will be attempted if all possible objections must first be overcome".

It is perhaps opportune for countries in the Asean region to pool their resources and conduct epidemiological and field studies in collaboration, for we do not have the finances to conduct research on such large scales, in our own individual countries.

Before I conclude, I would like to make a plea to our governments and universities in this region to make more funds available for fundamental basic research. Whilst money for applied research is not difficult to come by, we are faced with the reluctance of governments and institutions of higher learning to spend money on basic research. However, it is from this, and from this alone will our knowledge of this disease be further enhanced.

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