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Summary

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We heard, to begin with, an excellent description of the pathology of endomyocardial disease with regard to the endocardium, the myocardium and the gross pathological appearances at microscopy and so on. It appeared certain from Professor Hutt's paper that there are differences in epidemiology in the tropical as opposed to temperate zone varieties.

We discussed the questions of the season of the year, the amount of rainfall and aspects of that type all suggesting, as Prof. Falase says, the possibility of an infection and an insect vector. Multiple infections from parasites and other organisms, probably in areas where there is a great deal of water, fungus and very poor social conditions would be worth investigating.

We then heard from Dr Spry about his exceptionally interesting and very imaginative research, both in man and in the laboratory, on the abnormal eosinophil. This work and the work we subsequently heard from Dr John Davies is of great interest. It has ramifications far beyond those of EMF or even of heart disease and I think this introduces a whole new area of abnormalities that may develop in otherwise normal constituents of the blood and produce devastating disease. We heard from Dr Spry about the toxic proteins produced from various parts of the eosinophil, crystalloid and the membrane and how toxic these proteins were. The earlier data that he showed indicated that the amount of this protein in the serum was more than in normal controls in people with eosinophilia but without EMF, and was greatest in patients with EMF. All of which is very persuasive evidence put together that the abnormal eosinophils as they degranulate release their toxic proteins which get into the serum and then damage the heart. How they damage the heart I think is still unknown, and the acute experiments done by John Davies show that in the rat heart the endocardium was not damaged by the proteins, but the myocardium was. It is extraordinarily interesting and it does not destroy the theory for one moment because the endocardial damage might well follow the myocardial damage and something of this sort probably happens in patients with daunorubicin myocardial damage.

We also heard from Dr Spry a very significant clinical fact which is that in the patients with hypereosinophilia in a temperate climate, steroids given over a long period do seem to slow down the process of the disease. I think this is very important therapeutically.

The question of early diagnosis then came up. Of course, we always seem to see these patients very late. Professor Hutt made the point that it was almost impossible to find an early case at post-mortem, and that people come to hospital who are usually very ill especially in Africa and India. How can you hope to find the early stages of something as rarified as endomyocardial fibrosis? I think perhaps we all agree that clinical diagnosis, even if all the methods of investigation are at your fingertips, is extremely difficult in the early stages and the most promising method to me seems the apical two-dimensional echocardiograms described by Dr Harry Acquatella. It seems with this technique you could probably see some impairment of movement of the apex or even some loss of the cavity of the ventricle which might indicate early development of the endocardial plaques. Dr Eckhardt Olsen suggested that you could do a blood smear and see if the eosinophils were degranulated, and if they were assume that the patient had early EMF. Even if they do not have early EMF, this would identify a group perhaps at risk which should be further investigated. I realise, as Professor Falase said, that you really cannot expect people to do blood smears in ordinary rural village life, but it should be possible to do a pilot study and try to identify a group or a particular area at risk. We heard the experience of Dr Puigbo in Venezuela and it seems there was not any really significant difference between those patients and the patients that Dr John Davies has studied in Brazil and in India and those that Professor Hutt has told us about. So that all adds up to the fact that basically we are all dealing with the same syndrome of endomyocardial disease with fibrosis which goes through various stages. I think the evidence is very strong that degranulation of the eosinophil is important, but I would make the plea that there must be other factors which may be different according to where the patients come from, the environment in which they live, the conditions to which they are exposed and possibly genetic factors as well.

From the point of view of surgical treatment, we have a fascinating description from Professor Dubost who has, I suppose, the greatest experience of surgical treatment of this disease in the world, and added to that some of us were able to see Professor Bertrand in Moscow who described exactly the same types of disease and came to very much the same conclusions. His surgical mortality was about 40%, if I recall correctly, which is higher than that of Professor Dubost, but Professor Bertrand did produce a survival curve that suggested that patients who were operated on did actually do better than those who were not. This was not a randomized study, so it is very difficult to put any great weight on this as he was the first to point out. Nevertheless, it does look as if the patients who are carefully selected, and we have discussed the points on which selection is based, probably will do better treated surgically than medically. I do not think there is any magical medical treatment in terms of heart failure or general management that one has to recommend for this disease. The approach is presumably to get at the cause and deal with the eosinophil and to perform surgery on those who have got to an advanced stage and

have developed mechanical disorders which cannot be removed by pharmacological manoeuvres. That brings us back to the question of early detection. This is made more difficult because good function is maintained until a fairly late stage and so you do not get the changes of impaired contraction and regional abnormalities of the wall in the early stages that you might expect to get in other forms of heart disease that affect systolic function.

When I became President of the International Society and Federation of Cardiology it occurred to me that one of the things the Organization ought to be doing was to look very carefully at how it could help research into heart disease and cardiovascular disease in the developing countries and the tropics. Thanks to Dr Eckhardt Olsen's work, initiative, drive, energy and competence, this idea is now really coming off. The Year of Tropical Cardiology is being immensely successful and is generating a tremendous amount of interest and meetings like this and the one in Moscow which have brought together a number of expert people. I would like to take this opportunity to congratulate Dr Eckhardt Olsen for his magnificent service to cardiology and cardiological research in developing countries.