ROUND TABLE 2

Salt intake and the pathogenesis and treatment of hypertension

Chairman: Dr J. I. S. ROBERTSON
Opening speakers: Dr L. WILHELMSEN
Dr T. O. MORGAN
Dr F. O. SIMPSON

Discussants: Dr A. Hornych, Dr A. F. Lever, Dr F. Skrabal, Dr H. G. Langford, Dr L. Tobian, Sir George Pickering, Dr J. Brod, Dr B. Hood, Dr M. H. Maxwell, Dr J. Joossens, Dr N. Henningsen, Dr D. G. Beevers, Dr A. Malliani, Dr G. Berglund, Dr J. Fodor, Dr J. Tuomilehto, Dr J. Woods and Dr P. Weber

Introduction

J. I. S. ROBERTSON
MRC Blood Pressure Unit, Western Infirmary, Glasgow, Scotland, U.K.

In this session we have to address ourselves to two straightforward and possibly related questions: first, is there any evidence in man that a high dietary intake of sodium chloride for a longer or shorter time is responsible for hypertension? In other words, can a high salt intake push a person towards the upper end of that continuous distribution of blood pressure values which has been so often noted in population surveys? Secondly, can we in man, by reducing the dietary intake of salt within reasonable limits, produce a worthwhile reduction in arterial pressure? If there is such evidence, that clearly would be a very profitable line to pursue because we should have a means of treating hypertension which did not involve the taking of drugs.

Now it is remarkable that several people have said to me that they are surprised we are debating this issue at all. Their comments, however, have taken two rather contrasting directions. On the one hand there are those who say that they have no doubt that an unnecessarily high intake of sodium chloride is responsible for much, if not all, of what we rather arbitrarily call 'hypertension', and furthermore, hardly any question that it is possible to reduce arterial pressure with even quite modest restriction of the dietary intake of salt.

By contrast, others have said that, in their view, there is very little evidence to suggest that changes in dietary sodium chloride intake, within a range which can be achieved practically, are relevant to hypertension. A controversy of this sort is, of course, not unusual, and indicates that the available evidence is, on both sides, absent or defective. Fig. 1 illustrates what I think should be the proper correlation between the weight of experimental evidence and the vehemence with which it is asserted. The ideal relationship between these two
is illustrated by the straight line drawn at 45° across this Figure. I think that there are very few people in the world, and I am pleased to be one of them, who, when interpreting scientific evidence, pursue that 45° line, with only a few degrees of deviation to one side or the other. It is only when the evidence is conclusive that I become satisfied in my interpretation of it.

Now in relation to salt intake and hypertension, the matter is, typically, illustrated by the curve which swerves across the slide. When an idea such as this is first put forward it is usual for, initially, very few people to be interested. Most of those who do make a comment at all say that the whole idea is nonsense; in any case it has been fully covered in the early Scandinavian or German literature; furthermore, most of the work is poor and conducted with bad methods. After a time evidence may become more attractive and convincing and many more people publish papers on the topic. It is now that the curve takes a very steep upward swing. Now the force of assertion tends to exceed very considerably the available evidence. Lastly, when the evidence does become more or less conclusive, there is very little need for advocacy and little reward in being an advocate. Then the curve may swing back a little to the other side of the 45° line.

I know that there are many in the audience today who feel that this question of salt intake in relation to hypertension is at present on a point on the curve in the middle of the Figure; and that those who assert that salt is important in the pathogenesis of hypertension are exceeding the available evidence. I am also aware that there are many others in the audience who do not agree with that view.

I do think, therefore, that there is an important controversy here and one that is of very great relevance to clinical hypertension.

I propose that we should conduct the debate in the following fashion. First of all Dr Lars Wilhelmsen of Göteborg will discuss some methodological and epidemiological problems associated with studying this question. This will be followed by what we might describe as the evangelical approach from Dr T. Morgan or Newcastle, New South Wales, who will put forward his view that it is important to reduce dietary salt intake in treating hypertension. Lastly, we shall have what you may feel is a more agnostic presentation from Dr Olaf Simpson of Dunedin, New Zealand, who will outline what he believes are some of the difficulties in accepting the importance of dietary salt intake. After this we can throw the discussion over to all who wish to participate. I now ask Dr Wilhelmsen to give his introductory comments.