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# DIET IN RELATION TO RENAL AND CARDIO-VASCULAR DISEASE

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## Introduction to the Subject of Diet in Hypertension and Renal Disease

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Dr Roscoe (1952) deals fairly comprehensively, as far as one can in a short space, with questions of diet in chronic renal disease. As to diet in hypertension, I feel there is very little to say. The effect of salt depletion (with which Black (1952) deals) is interesting but, as he says, it is doubtful if it has much practical importance in treatment. The only other dietetic measure, which is sometimes important, is to reduce the weight of hypertensive patients if they are fat. Sometimes this brings down the blood pressure, but even when it does we are not certain that this is a true effect because it is known from intra-arterial readings of blood pressure that measurements taken with the ordinary cuff round an obese arm are very inaccurate, and the effect of the diet may simply be that we are now obtaining blood-pressure readings more consistent with the facts. In any case the reduction of weight does good by relieving the heart of some of its work.

There are two main determinants of water excretion under normal circumstances. The first is the solute load, that is the total amount of dissolved substances which the kidney is called upon to excrete. McCance (1945–6) showed, and Rapoport, Brodsky, West & Mackler (1949) confirmed, that when osmotically active substances such as salt, urea or sucrose are given to a person deprived of water, the amount of urine increases very greatly. As it increases, its concentration, or osmolarity, becomes less, approaching the osmolarity of blood plasma. This is the condition known as osmotic diuresis. The second factor determining water excretion comes into play when water is present in the body in excess. This is what we know as water diuresis, and in this case the concentration of the urine approaches that of distilled water.

In renal failure—I am referring to patients in the late stages of renal disease without oedema—the excretion of solutes continues to be efficient in spite of the fact that perhaps only 10 or 20% of the renal tissue is still functioning. Thus the solute load per nephron or per unit weight of renal tissue is greatly increased and the kidney behaves as if it were continually under the stress of an osmotic diuresis (Platt, 1951). A concentrated urine therefore cannot be produced and, as is well known, the urinary specific gravity approaches that of the plasma less its proteins (about 1009).

On the other hand, water diuresis is inefficient and the kidney has difficulty in getting rid of a big water load such as that of a litre of water.

Thus, in determining the amount of fluid that we should give to these patients we have to bear in mind that the kidney needs water in considerable quantity in order to excrete its solutes with the minimum of energy expenditure. About 3 l./day is probably the optimum quantity. On the other hand, excess of water should be avoided. Fortunately the thirst mechanism guides the patient effectively in most cases, and so long as he is provided with fluid in sufficient quantity he is usually quite capable of judging for himself the amount that he should drink.

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## Salt and Hypertension

By D. A. K. BLACK, *Department of Medicine, Royal Infirmary, Manchester*

Both the substantives of my title stand in some need of definition. 'Salt' is used not as a chemical expression, but as the ordinary description of sodium chloride; there is fairly general agreement that sodium is more important than chloride in sustaining blood pressure, and this is not without practical importance, as some of the 'salt substitutes' on the market, though free from chloride, contain sodium citrate. 'Hypertension' denotes a physical finding, that of raised blood pressure, and not a disease state; as clinical knowledge advances, we are able to distinguish patients in whom renal disease, suprarenal disease, or disease of the central nervous system accounts for the elevated blood pressure. Most patients with raised blood pressure have, however, no demonstrable primary disease to account for their hypertension; they are said to suffer from 'essential hypertension', and it is with this limited group of hypertensive patients that I propose to deal. Sustained hypertension can in itself cause damage to the kidneys, heart, and brain; most of the patients from whom we have derived data have a normal excretory function, but in one this was reduced to about a quarter of normal.

The practice of salt restriction in hypertension antedates considerably any acceptable rationale for the procedure. Widal and his school seem to have introduced it about 50 years ago, but its use in the English-speaking world dates from the enthusiastic advocacy of Allen in America in the nineteen-twenties. Hypertension is a syndrome in which the actual blood-pressure level is very variable, and there may be a considerable fall in blood pressure when the patient is removed from active life, especially if he is rested in bed. It is also a syndrome which is quite compatible with long survival after its initial discovery. Each of these considerations makes it difficult to assess the value of any proposed therapy, and until a few years ago the general opinion seemed