

It has been claimed that overproduction of salt-retaining adrenal steroids is concerned in the production of essential hypertension, and Perera & Blood (1946) relate the failure of hypertensives to lose salt and water on a salt-free diet to this. In experimental hypertension (Floyer, 1951) the effect of adrenalectomy in bringing down the blood pressure can be prevented by supplying salt. It would seem from this and from previous work that the ability to conserve salt is essential to maintenance of blood pressure; but our present results do not support the view that the 'cause' of essential hypertension is an overproduction of the hormones which mediate salt conservation.

SUMMARY

1. The practice of salt restriction in the treatment of essential hypertension is discussed.
2. Five hypertensive patients did not conserve sodium more efficiently than normal subjects, when placed on a low-salt diet.
3. This study does not support the view that an excess of salt-retaining steroids is present in essential hypertension.

I am indebted to Professor R. Platt for the results on sodium output on the rice diet.

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Diet and Chronic Renal Disease

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Chronic renal disease includes a number of conditions that must be diagnosed before treatment can be carried out, but here only two will be considered; chronic type 2 nephritis and the chronic renal failure which develops following acute type 1 nephritis, chronic pyelonephritis or malignant hypertension.

Chronic type 2 nephritis

This is the only variety of chronic renal disease in which recovery occurs, so that treatment is particularly worth while. But although dietetic treatment would clearly appear to be indicated and may improve the symptoms, we can seldom be certain that it has had any curative effect.

The condition is characterized by marked albuminuria, which results in lowering of the plasma proteins, especially albumin, and in protracted cases in normochromic anaemia (Roscoe, 1950). There is also oedema, often of a very severe degree and, with this, sodium retention.

Treatment consists of a high protein intake to improve the plasma-protein concen-

tration, and a low salt intake in the hope of reducing the oedema. The water intake does not seem to be important, and there is one form of treatment, which consists of giving 7 pt. of fluid a day, which sometimes results in diuresis with loss of the oedema.

Patients that do not recover sometimes lose their oedema and pass into a stage of chronic renal failure. The dietetic treatment must then be changed.

Chronic type 1 nephritis, chronic pyelonephritis and renal failure in malignant hypertension

The renal lesion in these conditions is always progressive, but the course may be prolonged, especially in chronic pyelonephritis, and the patients may lead a normal life for months, or even years, so it is important to prescribe any treatment that delays deterioration. There is degeneration of the kidney nephrons affecting both glomeruli and tubules and resulting in a quantitative loss of functioning kidney tissue (Platt, Roscoe & Smith, 1952) which becomes greater with time. At first the remaining nephrons are able to maintain excretion, but as more nephrons are lost kidney function fails.

It is not obvious why the degeneration of nephrons should continue when in many cases the original destructive process is no longer active, but a possible explanation is as follows: each remaining nephron has to carry out more than a normal amount of work in order to excrete an undue proportion of waste products, this leads to hypertrophy and thence to degeneration. This theory is supported by both pathological and experimental evidence.

Hypertrophy of the remaining nephrons in diseased kidneys is marked (Oliver, 1939) and it also occurs in animals subjected to partial nephrectomy (Addis, 1940) so that it is clearly a response to a reduction in kidney tissue. The extent of the hypertrophy, moreover, varies with the amount of work which the kidney remnants have to perform. This has been shown in two ways, both with partly nephrectomized rats. Firstly, the more kidney tissue is removed, the greater the work that each remaining nephron has to do, and Addis (1940) has shown that smaller kidney remnants hypertrophy more than larger ones. Secondly, the work of the kidneys can be increased by increasing the protein in the diet and so the amount of urea to be excreted, and Addis (1948) again showed that the hypertrophy was proportional to the protein in the diet. There is also experimental evidence that increasing the work not only produces hypertrophy but also hastens kidney failure. When a high-protein diet is given renal failure develops more rapidly and death occurs sooner both in partly nephrectomized rats (Addis, 1940) and in rats with nephrotoxic nephritis, an experimental condition resembling type 1 nephritis in its pathology (Farr & Smadel, 1939).

If, as the above results suggest, hypertrophy and degeneration are dependent on the work that the nephrons have to do, our treatment should be to reduce this work.

The actual energy expended in urine formation cannot be determined, but it is possible to calculate the minimum amount of osmotic work needed for the excretion of each solute (Rhorer, 1905). This is:

$$W_{\min} = R \times T \times V(U \times \ln U/P + P - U),$$

where R and T = pressure and temperature constants, V = volume of urine/min,

U = concentration of the solute in urine and P = concentration of the solute in plasma. Now the work will become less the more nearly the urine approaches protein-free plasma in composition, and it is in fact found that in renal failure urine concentrations do not differ as widely from plasma concentrations as normally, which suggests that the available energy is limited.

When we consider the effect of different metabolites, it is found that urea and nitrogen and chlorine are together responsible for about 85% of the work and so are the only ones which need be taken into account. Urea is present in urine in much higher concentrations than in plasma, so that much energy is needed for its excretion. However, when the kidney tissue is reduced, at first all the urea formed cannot be excreted so the plasma concentration rises. This finally results in a normal rate of urea excretion, and it also means that the difference between plasma and urine concentrations is reduced and less energy is expended in excreting the same amount of urea, but even so the work involved is considerable. It is possible to reduce this work by decreasing the protein intake and so the urea excretion, and increasing the fluid intake so that the urinary urea concentration is reduced. Sodium and chlorine are usually consumed in such amounts that their urinary concentration is less than the plasma concentration, so here treatment should be by increasing their intake and reducing the fluid intake. Thus in the one case a high fluid intake is indicated and in the other a low. However, Newburgh (1943) has calculated that the best compromise between these conflicting needs is obtained when the daily fluid intake is about 2 l., and we now give our patients this amount of fluid and a low-protein diet with about 5 g of extra salt added to the food after cooking.

A few cases of chronic renal failure develop a condition characterized by marked loss of sodium chloride, and these must be given much larger amounts of salt (Thorn, Koepf & Clinton, 1944).

The need for a high salt intake must be remembered when renal failure develops in cases of hypertension treated with a low-salt diet. Here a salt deficiency may be more dangerous than a high blood pressure.

Anaemia of renal failure

In conclusion, the anaemia of renal failure should be mentioned. This increases in severity with increased nitrogen retention (Roscoe, 1952). It is not, like the anaemia of chronic type 2 nephritis, due to protein deficiency, so it is not adversely affected by a low-protein diet. It is normochromic in the majority of cases, so there is no point in giving iron. In fact, no effective treatment is known.

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Diet in Heart Disease

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Undernutrition is not an important factor in the causation of heart disease in this country, although elsewhere severe deficiency states such as beriberi may result in heart failure. A similar picture is seen in chronic alcoholics in whom there is a conditioned deficiency of vitamin B₁, but such cases seldom occur nowadays. Undernutrition may be a factor in the incidence of rheumatic fever, and a decline in incidence has been noted in countries like Sweden where standards of living have improved. It is probable, however, that better housing has been as important as improved nutrition in producing this reduction.

Diet has some part to play in the treatment of heart disease. It is important to remember, however, that a patient with heart trouble is of necessity limited in many ways, and it is unjustifiable to impose further restrictions unless there is good reason for them. Needless restriction, by producing introspection, is indeed often harmful. In a well-compensated ambulant patient there is no evidence that any particular kind of foodstuff is harmful, and we can advise as Sir James Mackenzie did, 'the kind of food the patient likes, so long as it does not disagree with him' (Mackenzie, 1908, p. 263).

When the heart fails, however, some modification of this attitude is necessary. Congestion of the liver and alimentary tract impairs the normal processes of digestion and absorption. The cardiac output is reduced, and the demands of the digestive tract on the limited amount of blood available must be curtailed. Finally, there is diminished excretion of sodium by the kidneys, with a tendency for the retention of salt and water in the tissues. In the presence of decompensation, therefore, one must advise small, frequent meals which can be easily digested and absorbed, with adequate rest after food. Restriction of salt is advised in order to avoid the retention of fluid in the tissues. A really salt-poor diet, containing less than 2 g daily, may be achieved in hospital, but it is unappetizing and difficult to prepare. At home, the most one can expect is for the patient to avoid salty foods, beer, and added salt at the table. This reduces the daily intake to approximately 5 g of salt and, when the salt intake has been previously high, may be sufficient to prevent the accumulation of oedema. In most cases, however, digitalis and mercurial diuretics are required as well.

Perhaps the most important contribution of dietetics to the treatment of heart disease is in the control of obesity, for the reduction of excess weight is a very valuable weapon in the treatment of all forms of heart disease, but particularly of coronary artery disease (angina pectoris and coronary thrombosis). Although obesity produces no actual structural damage to the heart, it may itself give rise to all the symptoms of