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Symposium on

Nutrition and the Kidney

Some of us who enjoy the piquant flavor of broiled kidneys wrapped in bacon—and other dishes made with this organ—have learned that they are rich in protein, thiamine, vitamin B_{12}, and other important nutrients. The reasons for this are obvious, for the kidneys—besides controlling the homeostasis of the *milieu intérieur*—are second only to the liver in synthetic activities and in other biochemical functions which serve the body. The kidneys also have an endocrine function, and their wealth of enzymes serve a variety of structures and cells. Despite their need for protein and nutrients to carry on the metabolic activities described above, these small organs seem to be relatively resistant to the effects of nutritional deprivation. Approximately 3600 liters of blood course through the renal vessels each 24 hours, and the kidneys are in the unique position of being able to tap for their exclusive use one-sixth of the nutrients which are pumped around the body each day by the heart. Whether this is the reason for their immunity to nutritional bankruptcy is, of course, speculative.

In animals, disturbances of renal function and structure have been produced with some difficulty by dietary manipulations. Nocturia, hematuria, renal calculi, and tubular abnormalities have developed as a result of inadequate food intake. Thus far, deficiencies of ascorbic acid, vitamin K, vitamin A, alpha-tocopherol, linoleic acid, choline, potassium, magnesium, and chlorine have been shown to affect the kidneys of animals—usually the tubules.[1] The pathologic changes have not always been clear-cut, and this is perhaps due to present-day difficulties in interpreting structural abnormalities in tubular cells by tinctorial techniques. In man, scurvy[2] and hemorrhagic hypoprothrombinemia[3] are the only deficiency states known to produce hematuria, and until recently there was no evidence to hand that deficiencies of other nutrients seriously affected renal function or structure. On the other hand, diseases of the kidney—be they congenital[4] or acquired—are notorious for the frequency with which they compromise the nutrition of the body.

The symposium on *Nutrition and the Kidney* in this issue of the American Journal of Clinical Nutrition deals in the main with methods of protection of the body from the nutritional ravages of kidney disease. An international panel of physicians—whom we hasten to greet—give practical

advice, but they also indicate that the kidney of man may not be as protected from the ill effects of malnutrition as we have suspected. Drs. Sargent and Johnson call attention to alterations of renal function in man produced by changing the proportions of nutrients in the diet. Dr. Conn gives us a new and useful name—"kaliopenic nephropathy"—for the recently described and sometimes serious renal disease produced by potassium deficiency. And Dr. Davies from Central Africa indicates that renal tubular degeneration and isosthenuria are found in some infants ill with kwashiorkor (tropical protein malnutrition). Davies' observation may have some bearing on the etiology of lipoid nephrosis in children and of a recently described abnormality in adults ill with the nephrotic syndrome.[5] In both of these conditions—as in kwashiorkor—tubular degeneration seems to be the outstanding renal abnormality.

The usual forms of glomerulonephritis are probably the result of streptococcus infection. But two afflictions of man which involve the kidneys may be the result of a disturbance of nutrition: these are diabetic nephropathy and certain forms of hypertension, such as eclampsia and essential hypertension.

It has long been suspected that an excessive intake of common table salt was a factor in the production of eclampsia and other forms of hypertension. The relationships between sodium intake and the kidney have been explored recently by Meneely *et al.*,[6] who have produced renal lesions and hypertension in rats by diets containing large amounts of salt; and also by Hartroft and Hartroft,[7] who—in a most important communication—have demonstrated interrelationships between levels of dietary salt, the activity of the juxtaglomerular apparatus of the kidney, and the glomerulosa (aldosterone-producing) layer of the adrenal cortex. The empirical use of diets low in salt in pre-eclampsia and eclampsia and in essential hypertension may eventually be placed on a sound basis by the observations cited above.

Best and Hartroft[8] have produced hypertension and renal disease in adult rats by an acute deprivation of choline during their infancy, the rats being fed at a luxus level during the rest of their life span. This observation, coupled with Hartroft's finding of intercapillary glomerulosclerosis-like deposits in the glomeruli of choline-deficient rats,[9] raises again the question of the nutritional causation of the vascular complications of diabetes, long suspected of being dietary in origin. More exciting, perhaps, in this regard are the recent observations of Brontë-Stewart *et al.*,[10] indicating that a high dietary intake of saturated or hard fat raises blood cholesterol levels in man, while a large intake of unsaturated fatty acids can reduce these. It is interesting to recall the investigations of Burr and his associates[11–13] on linoleic acid deficiency and renal lesions in rats and to speculate that a high intake of hard fats—forced on us in recent decades by the food industry—may have produced hardening of the arteries not only in the kidney of the diabetic patients, but elsewhere in their bodies.

It should be obvious to the reader that a great deal needs to be learned about the effects of malnutrition on the function of the kidney. We hope that the symposium in this issue of the AMERICAN JOURNAL OF CLINICAL NUTRITION will stimulate work in this field.

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