While analysis of the amniotic fluid provides valuable information in some conditions there is sufficient cause for concern about its safety to limit its usefulness for the time being. Possibly the prior localisation of the placenta by ultrasound may prove to be unnecessary, but we still need to know the actual risk of rhesus sensitisation and to obtain more extensive experience with suprapubic amniocentesis in late pregnancy.


### Low-protein diets in chronic renal failure

Restriction of dietary protein relieves symptoms of uremia—the nausea and vomiting in particular. No doubt dietary restriction could postpone the onset of symptoms if started early on, but since their appearance is unpredictable such a policy makes little sense, except perhaps in patients with progressive anaemia. Imposition of an unpalatable diet solely in an imperious attempt to improve the “biochemical profile” is neither reasonable nor kind.

Symptoms may be minimised, but sooner or later nutrition suffers. How soon depends partly on the progress of the underlying disease and partly on the singleness of mind with which the patient follows the diet. The chemical identity of the products of protein metabolism which cause the clinical syndrome of uremia remains as elusive as ever, but it is important to realise that the poor nutrition of uremic patients may as often be caused by an inadequate diet as by the toxic effects of retained compounds.1 A low-protein diet does not improve renal function; it only reduces the accumulation of potentially toxic metabolites. The progression of renal insufficiency depends on the nature of the kidney disease; the prevention of saline depletion; and the control of hypertension, urinary tract infection, and a handful of other correctable factors.

Doubts remain about the ideal composition of low-protein diets, and patients continue to have considerable difficulty in stomaching them however exemplary their design. Although uremic patients have maintained nitrogen balance on as little as 20 g of protein daily,4–4 many do not easily do so, at least not within a few weeks. Only when about 35 g of protein is allowed can nitrogen balance be reasonably well assured.5 Not only must the diet contain both high-quality protein to ensure sufficient essential amino-acids and a disproportionately large energy supply as carbohydrate and fat, but the patient needs to be persuaded to eat it. Predictably, the more restricted the diet the poorer is the patient’s compliance.6

A low-protein diet postpones the need for regular dialysis or renal transplantation, but it may compromise the success of those treatments if the patient’s nutrition deteriorates meanwhile. Measurement of nutritional status is infuriatingly imprecise; in the end sequential measurement of body weight (in the absence of oedema) is probably as good as any other variable. Plasma albumin is often moderately reduced, but its concentration conceals the extent to which the extravascular pool has been reduced to maintain the intravascular albumin.7 8 Albumin turnover is low because catabolism is proportional to the intravascular concentration.7 These changes give little information about the adequacy of a diet, for they happen soon after protein is restricted and are not usually progressive.8

Not surprisingly, low-protein diets are most useful in those with very slowly progressive chronic renal failure. More complex treatment may often be postponed indefinitely in patients with chronic renal failure as a result of relieved obstructive nephropathy, healed renal tuberculosis, or chronic pyelonephritis. For these, and for the hard core of patients either unsuitable for or unable to obtain transplantation or dialysis, further refinement of dietary treatment must be pursued. Diets based on essential amino-acids have yet to be proved advantageous, but their keto- or hydroxy-acid precursors have shown initial promise.9 Originally suggested10 for improving nutrition on very restricted renal diets (by channelling retained urea nitrogen into protein synthesis) they seem instead to exert a protein anabolic effect greater than their transamination alone can explain.9 11 These compounds deserve further study, but for most patients with progressive renal failure conventional diets containing less than 30 g of protein will be used briefly, if at all, as soon as regular dialysis and transplantation become widely available.

3 Giovannetti, S, and Maggiore, Q, Lancet, 1964, 1, 1000.

### Immunotherapy for multiple sclerosis

The perivascula cuffing and oedema seen in acute lesions of multiple sclerosis (MS) suggest that some sort of immune allergic phenomenon may play a part in causing it.1 Controlled antibody surveys on patients with MS have thrown suspicion on many viruses, including measles, type C influenza, herpes simplex, parainfluenza 3, mumps, varicella zoster, and vaccinia; any part they may play in the production of the disease process is now under close scrutiny.2–9 Virions and nucleopodids of parainfluenza and paramyxoviruses have been found in brain tissue from patients with MS by cocultivation techniques and by electronmicroscopy.2–9

During replication some viruses incorporate cell constituents into their new coat, which alter the antigenicity of the new virions and may also modify the antigenicity of the surface of the infected cells. Changing the cellular antigenic determinants in this way might cause neuronal or glial cells to be regarded as “non-self” and so provoke a damaging allergic reaction.