SIR,—The minimum dietary sodium requirements have never been formulated satisfactorily. Whether a required level of 10 mmol/day has been inherited from primate and hunter-gatherer ancestors, as stated by Dr Trowell, is still open to argument. In the most gluttonous meat-eaters it could indeed have reached 60 mmol/day if they consumed something like 3 kg of lean meat per day, but this must have been much, much less in non-lean-omnivore herbivores. The common food plants contain very little sodium.

It is not necessary to look for remote ancestors or for hunter-gatherers. These herbivores may be contemporary skilful cultivators. I refer to the highland Papuans in New Guinea for over a million of whom the staff of life is the sweet potato.1

The highland sweet potato provides some 1-3-1 mmol Na/kg. It is often responsible for 80-90% and over of the total food intake and it is improbable that other vegetable sources have higher rates. The contribution of animal matter does not count. The chance of imported salt reaching the autochthonous consumer is comparable to that of finding caviar on a European dining-table.

I had the opportunity to determine electrolytes in 24 h urines of a highland community.2 In groups of male and female adults and adolescents Na excretion was 1-7-2-1 mmol/kg body weight/24 h. The lowest single value was 0-3 mmol. Three adult males had unusually high values (6-5, 8-8, and 26-0 mmol) but they had had access to the scanty resources of the local missionary which did include imported salt. A control group of Dutch student-nurses consuming an institutional diet excreted 154-177 mmol/24 h.

Although these highland Papuans were healthy and functional according to local standards, goitre was endemic. The people were small (adult males about 52 kg/155 cm, females 44 kg/146 cm). Protein intake was very low. The altitude was about 2000 m; garden plots were on steep slopes and rainfall was heavy.

This community may not be the best example. However, these findings do show that the numerous sweet potato farmers in the New Guinea highlands, deprived of outside contacts, can hardly achieve more than 6-8 mmol Na/24 h (350-460 mg NaCl) in their customary diet. Such a level cannot be stated to be inherited because it is simply dictated by the ecological situation. If "inherited" is meant in an evolutionary sense such levels are shared with many herbivore animals.

It is true that the Papuans have a craze for salt but they also have one for tobacco. A distinction should be made between salt hunger and salt requirement. Hipsley, studying Papuans on the shore villages which are built on piles above the sea-water, recorded 24 h urinary excretions of 8 mmol Na (range 1-35).3 One spoonful of seawater would provide that quantity.

Blood pressure in highland Papuans tends to be on the low side;4-6 and, more significantly, no increase with age occurred. In these highland communities potassium intake and excretion would be very high. We found levels between 105 and 180 mmol K/24 h in urine. It would be very interesting to study electrolyte balances of diarrhea patients in such extreme conditions.

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A 12-year-old girl presented in July, 1977, with a clear history of temporal lobe epilepsy confirmed by EEG. After 2 weeks on sodium valproate she was readmitted with further episodes of confusion, perseveration, and aggression. Following a short period of observation she was discharged on sodium valproate 800 mg (30 mg/kg) daily. Subsequently she had no further seizures, her general behaviour improved, and her appetite became normal. Liver-function tests before treatment were normal.

Some 9 months later seizures recurred, associated with diarrhoea, vomiting, and anorexia, and she was admitted with a possible diagnosis of sodium valproate toxicity.

Neurological examination revealed generalised hyperreflexia and some tremor. There was no oedema or hepatic enlargement. The serum valproic acid level was 127 μg/ml (therapeutic range 50-100). The drug was withdrawn. Some 36 h later the patient collapsed with circulatory failure, and died 2 days later.

The gross histology of the liver was normal. Neuropathological examination of the brain showed acute lesions, consisting of widespread “hypoxic” changes in nerve cells with foci of spongy necrosis in grey-matter, and the grey-matter contained enormous numbers of enlarged, pale astrocytic nuclei and scattered large cells of Opalski type. The lesions corresponded closely with those observed in fatal cases of hepatic encephalopathy, the principal association being with hyperammonaemia.

Case 2

A 41-year-old mentally handicapped girl with poor epileptic control was changed to a combination of carbamazepine (100 mg in the morning) and sodium valproate (400 mg at night). This was subsequently readjusted to sodium valproate alone (800 mg daily, a dosage greater than 40 mg/kg). For a short period primidine 125 mg at night was added and the dosage of sodium valproate was reduced stepwise to 400 mg daily because peak blood levels of valproate were 219 μg/ml. The valproate level fell to 118 μg/ml and at this stage an EEG showed a “disorganised record with sharp and slow complexes” corresponding closely with those observed in fatal cases of hepatic encephalopathy. The record was diffuse with a possible localisation on the left side post-centrally, with sharp waves in the mid temporal lobe area. The record was diffuse with a possible localisation on the left side post-centrally. Seizure control was poor and the child seemed confused.

Pursuing a possibility of a urea cycle enzyme abnormality, we measured her plasma ammonia level and found it to be 85 μmol/l (reference range 4-25); this abnormality was confirmed 2 and 4 weeks later. A small dose of phenobarbitone was introduced; on this combination (valproate 400 mg daily with phenobarbitone 60 mg at night) valproate levels were in the therapeutic range (75 and 74 μg/ml) yet ammonia concentration is raised.

RENAL FUNCTION AND 1,25-DIHYDROXYVITAMIN D THERAPY

Sir,—Dr Paterson’s report (May 31, p. 1164) on hypercalcaemia due to overdose with vitamin D prompts us to report a case which illustrates the dangers of therapy with 1α-hydroxycholecalciferol (1α-OHD).

The patient presented aged 48 in 1972 in renal failure due to bilateral hydropnephrosis and an atriope left kidney. The surgeon found a retrocaval ureter on the right. After transposition of the ureter renal function was well maintained. In July, 1977, he started to complain of back pain. Investigations revealed plasma ura 27.7 mmol/l, creatinine 0.41 mmol/l, calcium 2.00 mmol/l, and phosphate 1.73 mmol/l, and alkaline phosphatase 206 IU/l. X-rays showed some sclerosis of the upper and lower regions of the vertebral bodies (“rugger-jersey” spine). Hand X-rays revealed subperiosteal erosions. Bone biopsy showed a gross excess of osteoid and active osteitis fibrosa. He was treated with aluminium hydroxide and, after the plasma phosphate had been controlled, 1α-OHD 2 g daily was started. In May, 1978, plasma calcium was 2.94 mmol/l and plasma creatinine had increased to 0.74 mmol/l. 1α-OHD was immediately discontinued. In March, 1979, he again complained of back pain and the lumbar area was tender. At that time his plasma creatinine was 0.60 mmol/l. In April, 1979, he was restarted on 1α-OHD 0.5 μg daily. He continued on this therapy until December, 1979. In July, 1979, when plasma calcium was 2.29 mmol/l, plasma phosphate 1.7 mmol/l, and alkaline phosphatase 90 IU/l, plasma creatinine had already increased to 0.65 mmol/l. Subsequently, plasma phosphate 10%. In parallel with the biochemical and haematological improvement there was a marked clinical improvement; the child became more alert and less ataxic. Subsequently seizure control has been maintained on carbamazepine and ethosuximide.