who had had a renal transplant for at least 6 months, and who had a serum-creatinine of less than 3 mg. per 100 ml., no difference was found between two groups in the percentage of hyperlipæmic patients, fasting serum-triglycerides, or pre-β-lipoproteins. Only serum-cholesterol levels were higher in patients on daily steroid, but the mean level was still in the normal range (see accompanying table).

These results do not confirm, therefore, the action of alternate-day steroids in preventing post-transplant hyperlipidæmia, even if our patients had a shorter post-transplant period than Beaumont et al. patients (mean 17 months *v.* 43 months). The role of steroids in this complication requires further definition; it might be best elucidated by serial determination of the lipid patterns after transplantation.

But steroid therapy may not be the only cause of transplant hyperlipæmia: for instance, after successful transplant many patients, for psychological and physical reasons, tend to overheat, which on its own or in combination with prednisone can predispose to lipid disorders. The observation that a low-calorie/low-starch diet restores the lipid pattern to normal in transplanted patients [3] means that other factors besides steroids might explain the hyperlipidæmia.

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BLOOD-LIPIDS IN RENAL DISEASE

SIR,—The interpretation that Cramp et al.[4] give to the work of Gutman et al.[5] is not in agreement with the conclusion those authors reached. Heparin administered during hæmodialysis activates lipoprotein lipase, which splits triglycerides in free fatty acids and glycerol.[6–8] The effects of heparin wear off in a few hours and triglycerides and lipolytic activity return to predialysis values. Gutman et al.[5] showed that these effects did not occur with regional heparinisation and peritoneal dialysis, and therefore rightly concluded that metabolic improvement occasioned by dialysis did not seem to be responsible for these triglyceride and postheparin lipolytic activity changes.

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BODY POTASSIUM DURING PROLONGED THIAZIDE THERAPY

SIR,—If Dr Wilkinson and his colleagues (April 5, p. 759) are right in concluding that potassium supplements are not necessary in the treatment of essential hypertension with a thiazide, this is important. However, they say nothing about the dietary potassium intake of their patients. It seems relevant to know whether this was in the middle of the normal range for all of them. Secondly, although the mean total body potassium (T.B.K.) changed little, it is not clear from the data whether there was a consistent and continued fall in T.B.K. in any of the individuals studied. If some individuals show a clinically significant fall in T.B.K., a means of identifying them would be needed, so that they could be given potassium supplements.

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*** We showed this letter to Dr Wilkinson, whose reply follows.—ED. L.

SIR,—All our patients had potassium intakes around the middle of the normal range, with the usual seasonal variation. Only one patient showed a persistent fall in T.B.K. to 80·7% of his pre-treatment value after 12 months; however, at 15 months his T.B.K. was 85·2% and at 18 months 92·5%, without potassium supplements. We noted this stabilisation of T.B.K. in all the other patients, but it usually occurred after 6 months of therapy. We would like to emphasise that *routine* potassium supplements are not necessary in the treatment of essential hypertension with a thiazide.

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NEUROBLASTOMA, IMMUNODEFICIENCY, AND CATECHOLAMINES

SIR,—Your editorial on neuroblastoma (Feb. 15, p. 379) commented on the high urinary level of vanillylmandelic acid (V.M.A.) in up to 90% of children with neuroblastoma. This may be related to the poor prognosis in this disease.

The immune system may be very important in the prevention and in the response to treatment of malignancy. Patients with congenital immunodeficiency of various types have a markedly increased incidence of neoplasia.[1] Many human neoplasms, including neuroblastoma, have tumour-specific antigens which elicit an immune response from patients' tissue in vitro, despite the fact that the tumour grows in vivo. Cancer patients with immunological deficiency have a poor prognosis even if little or no detectable disease is present.[2] No matter how exuberant the immune response, it has been shown repeatedly that immunotherapy is of no avail if the tumour mass is as small as 1 c.cm. In patients freed of gross neuroblastoma by surgery, radiation, or chemotherapy, microscopic foci of disease may remain and immunocompetence may be important under these conditions. It is at this point that the excretory products of the neuroblastoma may be damaging.

Adrenaline can modify (generally by inhibition) a wide variety of immune processes by increasing leucocyte cyclic-A.M.P. levels.[3] The most familiar example is its use in the treatment of asthma. Adrenaline and other drugs that increase leucocyte cyclic-A.M.P. levels reduce the production or secretion of antibody,[4] suppress T-cell-mediated cytolysis,[5] and reduce D.N.A. synthesis in both T and B lymphocytes after mitogen stimulation.[6]

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