therapy in both groups. FT_4I levels correspond to the findings in the whole group of patients. TRAb values were undetectable in all the 20 patients randomly selected.

From a study measuring thyroglobulin shortly after ^{131}I therapy for non-toxic goitre, we know that this therapy is followed by a considerable increase in serum thyroglobulin after a few days, indicating antigen release from the thyroid. We suggest that this release in susceptible patients is able to produce a Graves'-like immunological response in a small proportion of those treated with ^{131}I for non-toxic as well as toxic multinodular goitre.

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Primary hyperparathyroidism and vitamin D deficiency

Sirs, The complex metabolic effects of liver enzyme induction and inhibition by rifampicin and isoniazid (Perry *et al.*, 1978) are emphasized by a recent case report (Kovacs *et al.*, 1994). As rifampicin has similar enzyme inducing properties to anti-convulsants there may be a further mechanism at work in terms of induced bone cell resistance to parathyroid hormone activity (Jenkins *et al.*, 1973). This would explain the fall in serum calcium producing normocalcaemic primary hyperparathyroidism, but the emphasis in the paper was on further 25-hydroxyvitamin D deficiency as the explanation. In Indian vegetarians at least (we are not given details in this patient) it has been particularly difficult to separate the effects of continuing D deficiency from any other exacerbating factors such as antituberculous therapy (Stamp *et al.*, 1980; Perry *et al.*, 1982). There has been no convincing report of late rickets or osteomalacia and in several hundred patients we reviewed at the chest clinic at St Mary's Hospital, Luton and Dunstable, no European patient presented with drug related osteomalacia although their average vitamin D intake was of the order of only 80–120 units per day. The effect in our view appears extremely small but there may be the occasional case as in the above report who does show an increased metabolism of 25-hydroxyvitamin D reflecting genetic susceptibility to rifampicin (Perry *et al.*, 1982) or a slow acetylation effect of isoniazid on cytochrome P 450 activity.

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Addison's disease

Sirs, We read the report of Kong and Jeffcoate on 86 cases of Addison's disease (1994) with great interest. The incidence of various endocrine, autoimmune, or relevant disorders accompanying the hypoadrenalism was similar to that reported in our series of 180 patients with Addison's disease (Kasperlik-Załuska *et al.*, 1991). However, we were intrigued by the very low prevalence of vitiligo in their group of patients. In our patients vitiligo has been one of the most frequent findings (14% in the group of non-tuberculous Addison's disease) and this observation led us to include it as a clinical marker of an autoimmune process in the polyendocrinopathies. Thyrotoxicosis was slightly more frequent in our series (Kasperlike-Załuska *et al.*, 1994); however, in three cases it was Hashi-toxicosis, not pure Graves' disease.

We collected a significant group of patients with Addison's disease of tuberculous origin, diagnosed mainly in the 1960s. Recently the greatest number of newly diagnosed cases have been of idiopathic type; metastatic destruction of the adrenals was also found in some elderly patients.