but, in the meantime, some of his questions can be answered.

ACTH, cortisol, Na, prolactin, T4, T3 and T4f measures came from blood samples drawn at a standardised time, 08.00 h the first day after admission. 17-OHCS measures came from 24 h urine samples collected the first day after admission. Our clinical observations in diabetes and literature reports led to the introduction of some measures of glucose kinetics. "Blood glucose" measures are the means of 12 different samples for each patient, 4 samples each from one of the three consecutive days after admission, with the blood drawn before the four daily meals or snacks, at 08.00, 12.30, 16.30 and 19.30 h. 'Blood glucose dispersion' refers to the standard deviations of the above means. 'Ketone bodies' measures are the means of 12 different urine samples collected in each patient precisely at the same times as the samples for blood glucose. Finally, 'ketone bodies dispersion' refers to the standard deviations of the above means.

The correlations found between GHQ-28 total scores and endocrine or metabolic measures in diabetes and Addison patients tend to support our hypothesis. One possible explanation for the failure to demonstrate a significant correlation with hyperthyroidism parameters may come from the fact that all these patients had severe pathology (psychiatrist's global CIS severity scores 3 and 4) and, therefore, the range of GHQ scores was small.

Dr McGauley's suggestion, based on the report by Starkman & Schteingart (1981), is probably the best one to interpret the failure to demonstrate significant correlations with GHQ scores in Cushing's patients. Firstly, 8 of them (57.1%) had adrenal adenomas and 6 (42.8%) were patients with pituitary ACTH-dependent disease. Secondly, we can now report that in the group as a whole the mild cases (psychiatrist's global severity CIS score of 2) had significantly lower ACTH levels when compared with both the cases of moderate intensity (severity score of 3) ($P=0.0353$) and the severe cases (severity score of 4) ($P=0.0126$) (non-parametric Mann-Whitney U-test). Thirdly, this relationship between psychiatrist's severity CIS scores and ACTH levels could not be demonstrated with cortisol levels in the Cushing's patients.

A. LOBO
M. J. PEREZ-ECHEVERRIA
A. JIMENEZ-AZNAREZ
M. A. SANCHO

Departamento de Psiquiatria
Hospital Clinico Universitario, Planta 11
50009 Zaragoza, Spain

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Geographical Error

SIR: McCreadie *et al* (*Journal*, August 1988, **153**, 174–177) refer to my study as carried out in England. Perhaps Dumfries is now in England, but Cardiff certainly is not. It is still the capital of Wales.

I. G. PRYCE

Whitchurch Hospital
Cardiff
Wales

Melatonin Secretion in Depression

SIR: Thompson *et al* (*Journal*, February 1988, **152**, 260–265) compared melatonin secretion between depressed patients and individually matched control subjects. They also reviewed most of the related literature, highlighting flaws in study designs that might question the validity of published results and conclusions. In concordance with several of the reports critically reviewed, we also have reported lower melatonin concentrations in a group of depressed hospitalised boys, compared with ambulatory control subjects (Cavallo *et al*, 1987). We considered our data as preliminary, as we grouped together patients with various subtypes of depression, and we failed to examine the effect of hospitalisation *per se* on the results. In contrast to our findings and the other studies reviewed, Thompson *et al* demonstrated no difference in mean nocturnal plasma melatonin concentrations between depressed and control subjects. Also, they observed no difference in the timing of melatonin secretion.

Several issues need to be addressed in their carefully designed study. Firstly, it is unclear whether the control subjects were screened for family history of depression. Secondly, studying melatonin secretion in individuals with diverse sleep/wake (and consequently, diverse light/dark cycles) prevents a valid