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.

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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.

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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

comparison of the timing of melatonin secretion. Finally, the depressed patients, but presumably not the control subjects, were given benzodiazepines. Kabuto *et al* (1986) recently reported suppression of the nocturnal melatonin surge by benzodiazepines in normal subjects. Although this observation must be taken cautiously, because the reported plasma melatonin concentrations are much higher than in most laboratories (Arendt, 1985), the potential effect of benzodiazepines on melatonin secretion should not be ignored. One might even speculate that these drugs could have a paradoxical effect, increasing melatonin concentrations in depression and thus explaining Thompson's results.

In conclusion, the relationship between melatonin secretion and depression remains undetermined.

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SIR: Dr Cavallo's letter raises three interesting questions about our study. Firstly, the relationship between a family history of depression and abnormalities in melatonin secretion is speculative. We tested the more cautious hypothesis that low melatonin secretion would be a state marker of depression. Since we were able to find no evidence in support of this, it would be illogical to search for an association with family history (i.e. genetic marker status).

Secondly, melatonin is not a sleep-related hormone. The sleep-wake cycle should not be equated with the light-dark cycle, since the two cycles may clearly dissociate from each other. Since we controlled for month of testing, the light-dark cycle was not a confounding factor.

Thirdly, the question of benzodiazepine use appears to strengthen the findings rather than diminish them. It is speculation indeed to suggest a paradoxical effect of benzodiazepines on melatonin in depression, one for which we know of no evidence. We agree that the relationship between melatonin secretion (volume, timing, and suppression by light) and depression remains of great interest, and there are still a number of important hypotheses to be tested. However, we also believe that it is important in such studies to control for all the relevant variables as we have endeavoured to do. We will be lead to doubt our study's findings if mistakes in design can be levelled at it rather than speculation.

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Self-Esteem: A Psychiatric View

SIR: Robson (*Journal*, July 1988, **153**, 6–15) mentions some of the views on self-esteem taken by cognitive therapists. While referring to the work of some rational emotive therapy (RET) therapists, he fails to mention the current position of self-esteem in RET theory, namely that self-evaluation, whether positive or negative, accurate or distorted, is a source of emotional disturbance. Ellis has delineated this position on many occasions (e.g. Ellis, 1972; Ellis *et al*, 1975).

Briefly, RET theory regards self-esteem as the individuals' rating of self as being either good or bad, based on the presence or absence of certain traits, behaviours or attributes. It regards such evaluation as irrational and self-defeating on the basis that human beings are simply too complex to be accurately rated. Ellis recommends that people rate only their abilities to perform specific tasks and give up rating themselves completely. He advocates that people accept themselves, *a priori*, as fallible human beings who like all human beings do some things particularly well, some things poorly, and many things adequately.

The confusion in the literature concerning the definition, measurement, and relationship to mental illness of self-esteem, to which Dr Robson draws our attention, would tend to support Ellis' contention that humans are too complex to logically rate their own worth.

I would wish those still seeking a scientific measurement of how people perform this illogical evaluation good luck in their task.

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