It is of interest that there is no significant correlation between GHQ-28 scores and either ACTH or cortisol levels in the Cushing's patients; especially as the authors cite the relationship between neuropsychiatric disability and hormonal levels in this subgroup as the basis for examining any relationship between these parameters in their heterogeneous sample of endocrine patients. One possible explanation for their failure to demonstrate a significant correlation between ACTH levels and GHQ-28 total scores comes from the work of Starkman & Schteingart (1981). They reported an increased prevalence of more severe neuropsychiatric disability in patients with pituitary ACTH-dependent Cushing's disease compared with patients with adrenal adenomas. However, the aetiological type of Cushing's syndrome in this subgroup is not specified.

It is difficult to know what conclusions to draw from the inclusion of the correlation coefficients between GHQ-28 total score and endocrine blood measures without additional methodological detail and further discussion of the results generated.

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Reference

STARKMAN, M. N. & SCHTEINGART, D. E. (1981) Neuropsychiatric manifestations of patients with Cushing's syndrome. *Archives of Internal Medicine*, **141**, 215–219.

SIR: The article by Lobo *et al* (*Journal*, June 1988, **152**, 807–812) uses the term 'endocrine psychosyndrome'. This term does *not* suggest that the psychopathology of all endocrine disturbances is the same: it reflects the clinical experience that the psychic alterations of endocrine patients concern in common *the same spheres of human inner life*, namely the biological background of general activity and of elementary moods, biologically-rooted trends (as for example, hunger and primitive sexuality), and biological rhythms. Within this frame of biological alterations due to endocrine diseases there are marked differences among different endocrine diseases. (In particular, the biological trends are influenced in different ways by different endocrine diseases.) If, however, an endocrine disease is complicated by a general metabolic cerebral alteration or by a general structural cerebral alteration, the psychopathology does of course no longer correspond to the term 'endocrine psychosyndrome'.

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SIR: Professor Bleuler, pioneer in the description of psychopathological disturbances among endocrine patients, further clarifies the meaning of the so-called 'endocrine psychosyndrome'. His comment about the "marked differences among different endocrine diseases" has to be welcomed. It has been his insistence on the common syndrome rather than the differences (Bleuler, 1967) which has influenced the content of some textbooks (Alonso-Fernández, 1976).

Our clinical experience, however, suggested that, firstly, the "changes referred to the impulses, mood states and different drives" in the 'endocrine psychosyndrome' (Bleuler, 1967) are frequently seen also in non-endocrine medical conditions, as it has been maintained recently (Gibbons, 1983), and secondly, consistent with present knowledge about the heterogeneity of endocrine diseases, we have been more impressed by remarkable differences between them, in the kind or severity of psychopathological phenomena. Aside from common knowledge about, for example, the predominance of anxiety in hyperthyroidism or depression in Cushing's patients, our clinical impressions, partially coincidental with literature reports, had suggested a relationship between the psychopathology observed and the severity of some diseases, such as Addison's and hyperthyroidism. The relationship seemed less clear in hyperprolactinemia and Cushing's. In type 1 diabetes, psychopathological phenomena had been observed particularly in patients with marked and quick blood glucose oscillations, especially with descending changes.

Therefore, it seemed reasonable to convert these clinical observations into a working hypothesis and submit them to test with standardised methods of assessment, which have rarely been used (Gibbons, 1983). Patients cognitively impaired were excluded, to minimise the risk of including the general metabolic or structural cerebral complications Professor Bleuler alludes to in his letter. Furthermore, in the attempt to trace the psychiatric disturbances to biological causes, it seemed appropriate to try to correlate the hormonal levels or related metabolic parameters with the psychopathology detected. The preliminary results tend to confirm the hypothesis (Pérez-Echeverria, 1985).

The present paper, part of our general study, was intended basically to validate the GHQ-28 in patients with severe endocrine diseases, with the hormonal levels or related biological parameters used as external validity criteria. To draw more specific conclusions, as Dr McGauley correctly suggests, additional details are necessary. We are in the process of reporting more data and pertinent discussions