

in part by Grant (1988) and, if we accept that Dr O'Neill really meant 'treatment' instead of 'cure', by Wood *et al* (*Journal*, July 1988, 153, 128). Section 2 of the Mental Health Act 1983 provides for the involuntary admission and *assessment* of patients: what is assessment but screening by non-invasive measures (observation, history-taking, and mental state examination)? The result of such an admission may well be the diagnosis of an 'incurable' condition such as schizophrenia – one might indeed say all mental disorders – or indeed 'untreatable' conditions such as psychopathic disorder and certain degenerative illnesses. The consequences for the patient of both the admission and the diagnosis may be devastating; far more so than for the HIV positive who is deprived of mortgage facilities and life insurance. Nonetheless, armed with the information derived from such an admission, substantial gains can result in terms of provision of appropriate management.

Dr O'Neill is concerned about the effect on the family of the finding of HIV positivity. Certainly this will be traumatic, but has she considered the possibility that this might well save the life of the spouse? Dr Connelly suggests that I might better understand the furor over HIV if I read Thompson's paper: I don't. As a psychiatrist, I am accustomed to dealing with catastrophic effects of life events and also with families ridden with guilt, anger and other emotions related to the condition of a member. Perhaps I could ask Dr Connelly to spell out what is so special about HIV that it merits consideration above and beyond that according to other life-threatening and stigmatising illnesses.

Dr Connelly refers to "the failure of countries to confront the social impact of AIDS". I am sure that this is true of the United Kingdom. The 'softly softly' approach was advocated on the grounds that it would prevent HIV being driven underground, yet the attitudes of the major financial houses seem to have done just this, although the medical profession remains fettered. How else can one reasonably explain a report rate for AIDS in the United Kingdom which is well under 50% of those for France, Australia, Switzerland, and Canada (Anon, 1988)? I find it quite remarkable that a civilised society permits a financial institution to refuse a potential client a service on the grounds that a test has been performed irrespective of its outcome. Surely legislation should be enacted against this victimisation rather than against the medical profession's pursuit of its time-honoured principles, and, in the absence of such legislation, surely the profession should actually extend HIV testing so that it achieves 'routine' status.

The legalities of the HIV problem have recently been reviewed by Dickens (1988), and interesting reading it makes. The erosion of medical values under the pressure of the HIV problem has yet to be reviewed, and will no doubt make the name of a future medical historian. At the moment, perhaps the last word is best left to Grant (1988): "Where will all this nonsense end?"

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#### Emotional Disturbances in Endocrine Patients

SIR: Lobo *et al* (*Journal*, June 1988, 152, 807–812) have correlated certain hormonal levels or related biological parameters with the total GHQ-28 score in their subgroups of endocrine patients; this aspect of their article needs further clarification. In particular, the terms 'blood glucose dispersion' and 'ketone body dispersion' are not explained. As only the correlation coefficients are presented, without the raw data, one is left to speculate as to the exact nature of these 'endocrine blood measures'. The only reference to their method is that these biochemical measures came from single assessments. Thus the reader can assume that blood glucose dispersion cannot refer to any measure of glucose kinetics such as its metabolic clearance rate or turnover. Have the authors, in fact, used blood glucose/ketone dispersion to denote the range of their sample values? Furthermore, it is not stated if the blood samples were taken at a standardised time or if the patients had fasted, variables which can heavily influence blood glucose concentration.

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 & Scheingart (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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STARKMAN, M. N. & SCHEINGART, 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-Echeverría, 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