The authors draw only careful conclusions. The lesson I learned, however, was that diagnoses in at least some of the cases concerned had in fact the predictive value claimed by those in favour of classification. The prognosis of e.g. an autistic child is indeed poorer than the outlook for a child of the same sex, age, IQ, Behaviour Screening Questionnaire (BSQ) score and social background but with no more than an unspecific behaviour disturbance; and even the best of day centres has less of an impact on the natural history of autism than we would wish (Rutter, 1967). A correct diagnosis should indeed tell us which treatment to choose and what success to expect of it. For example, drug trials of antidepressants match experimental and control patients not only for their scores on depression scales, age, sex, etc. but also and most importantly for diagnosis, e.g. primary depression rather than schizophrenia complicated by depressive symptoms. Perhaps a fairer picture of the day centre's long term achievements would have been painted if the children could have been matched for diagnosis as well.

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JASPERS: GENERAL PSYCHOPATHOLOGY

DEAR SIR,

The article by Professor Michael Shepherd (*Journal,* 1982, **141,** 310–12) quotes "the verdict of one influential British textbook" but does not give reference to this volume or to its author. The textbook referred to is that of Myre Sim's *Guide to Psychiatry,* 4th Edition (Churchill-Livingstone, London and Edinburgh 1981).

Professor Shepherd described my verdict on Jaspers' *General Psychopathology* as an "egregious assessment". As he was highly critical of it I assume that he did not use the word 'egregious' in the archaic sense when it meant 'outstanding' but in its modern sense of being 'uncommonly bad'. To criticize an author severely on his textbook and not mention the author or text by name is in egregious taste.

I do not see how he "can testify" to the impact of this "egregious assessment" on candidates for the M.R.C. Psych. for neither I nor he raised this issue of Jaspers in our examinations and neither did the candidates.

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ARE EATING DISORDERS FORMS OF AFFECTIVE DISORDER?

DEAR SIR,

Hudson and colleagues (*Journal,* February 1983, **142,** 133–38) have recently described a study in which a high prevalence of affective disorder was found amongst the relatives of patients with eating disorders. They concluded that this added to 'the evidence from studies of phenomenology, course of illness, response to biological tests, and treatment response, that the eating disorders may be forms of affective disorder'. In our opinion none of these lines of evidence stands up to close scrutiny.

With regard to *phenomenology,* depressive symptoms are indeed common in both anorexia nervosa and bulimia nervosa. However, clinical evaluation of these symptoms is complicated by the direct effect of the eating disorder on appetite, weight, energy, interests and concentration. Furthermore, since in anorexia nervosa it is well recognised that restoration of body weight is associated with a decrease in depressive symptoms, it is possible that the depression is a non-specific product of the malnutrition (Eckert *et al,* 1982). In bulimia nervosa, since the depression lifts in response to measures which enhance control over eating, the mood disturbance is likely to be a secondary phenomenon (Fairburn, 1982).

The findings of most studies on the *course* of anorexia nervosa have failed to support the contention that the condition is a form of affective disorder. Although many patients do exhibit depressive and anxiety symptoms at follow-up, the most striking observation is that the characteristic psychopathological features of the disorder (the pursuit of thinness and a morbid fear of fatness) tend to persist (Hsu, 1980). With one exception, the outcome studies have found that the eating disorder does not evolve into an affective disorder: instead, as Russell (1970) has noted, the illness 'breeds true'. As yet, there have been no studies of the course of bulimia nervosa.

The *biological tests* Hudson and colleagues refer to, relate to dexamethasone suppression. Several studies have found that a proportion of patients with anorexia nervosa or bulimia (DSMIII) have responses to dexamethasone similar to those found amongst patients with affective disorder (Gerner and Gwirtsman, 1981; Huson *et al,* 1982). However, it is possible that this abnormality represents a secondary