

the chance to examine schizophrenia discordant MZ twins from other centres for this and other aspects of our study.

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#### RESEARCH DIAGNOSTIC CRITERIA FOR PRIMARY AFFECTIVE DISORDER

DEAR SIR,

The St Louis Criteria (Feighner *et al*, 1972) and the Research Diagnostic Criteria (Spitzer *et al*, 1978), although widely used in research on depression, have been recently criticized. It has been stressed that this diagnostic approach does not provide homogeneous groups (Nelson *et al*, 1978; Feinberg *et al*, 1979; Nelson and Charney, 1980).

One classical way to test the consistency of a noso-

logical grouping is to follow the course of the disease over time.

Therefore a sample of patients previously diagnosed as having a primary affective disorder (PAD) has been followed up for a period of four years. One hundred and fourteen patients that met the criteria for definite PAD (and also meeting the diagnosis of major depressive disorder, primary subtype) were hospitalized at the Department of Clinical Psychiatry of the University of Florence during the years 1975–6. Seventy-eight cases out of the total were subsequently followed up as out-patients for at least 4 years; at the end of this period new assessments, on the basis of the newly acquired knowledge, were made by experienced psychiatrists not informed about the earlier diagnoses. The inter-diagnoser agreement was satisfactory ( $k = .92$ ,  $n = 44$ ).

The diagnoses of 63 patients (80.8 per cent) were still consistent with the former ones: 25 patients relapsed into episodes again diagnosable as PAD, whilst 38 had a four year period of well-being. Conversely 15 cases (19.2 per cent) had subsequent diagnoses other than affective: 7 patients showed paranoid symptoms, 7 had clearly hysterical signs, and one became an alcoholic.

Whether further diagnoses of schizophrenia, hysteria or alcoholism are compatible with that of affective disorder, depends on one's view of the natural history of such a disease. In fact it is still controversial whether a patient suffering from a major psychiatric disorder, such as depression, can recover and then be affected by a different major psychopathy, e.g. schizophrenia. However, most biological or psychological theories of depression assume that this disorder is incompatible with non-affective states. In any case, as the RDC are aimed to select homogeneous groups for research purposes, the exclusion of false negatives ought to be preferred to the inclusion of false positives. Thus, diagnoses changing over time have to be seen as at least dubious. Indeed it appears reasonable that some depressive onsets of schizophrenia, some affective signs superimposed onto hysterical personalities, or other secondary dysthymias, may be misdiagnosed as PAD.

This lack of homogeneity is not surprising if one considers that the RDC are mere checklists of symptoms, which ignore other sources of clinical knowledge, such as premorbid personality, family history, physiopathological markers, longitudinal course of the illness, etc. These factors have been the basis of the clinical method and nosology since Sydenham onwards.

In my opinion, therefore, the RDC, although helpful for improving standardization and diagnostic agreement, still fail to provide homogeneous samples.

Furthermore, as the RDC are arbitrary pragmatic conventions, with no underlying theory, there is little chance of reaching, with these means, a natural classification of mental disorders.

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#### ENDOGENOUS DEPRESSIVE SYNDROME

DEAR SIR,

Matussek, Söldner and Nagel (*Journal*, May 1981, **138**, 361–72) were unable to diagnose 14 per cent of their depressed patients as either 'endogenous' or 'neurotic'. These authors supported Eysenck's (1970) contention that such patients can carry components of both depressive syndromes. We believe that the expression *during childhood* of varying degrees of inherited endogenous depressive illness contributes to the concurrent development of neuroticism. Endogenous (primary, major, melancholic) childhood depression has an incidence of at least 1½–2 per cent (Kashani and Simonds, 1979; Staton and Brumback, 1981), but is seldom diagnosed as a biological disorder. When this illness is treated in childhood with

tricyclic antidepressants, however, significant improvement occurs in cognitive function (Brumback *et al*, 1980; Staton *et al*, 1981) and in socially maladaptive behaviour (unpublished data).

Akiskal (1981) describes the high incidences of affective episodes and familial affective illness in adult patients given the diagnosis of borderline personality disorder, and concludes that borderline psychopathology can be a *secondary* manifestation of affective disorder. Akiskal suggests that chronically unpredictable mood changes, often subtle and having their onset before adulthood, lead to lifelong social maladjustment and impaired self-esteem. Our studies of depressed children support Akiskal's conclusions. Treatable affective disorder is often obscured by secondary symptoms of personality disorder or neuroticism in both adults and children.

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