Social competence and adolescent psychosis

SIR: Dalkin *et al* (*BJP*, February 1994, **164,** 202–207) suggest that premorbid personality may shape the expression of symptoms in first-onset psychosis. Lack of social competence may be indicative of schizophrenic vulnerability, even in the absence of schizotypical symptoms or represent the earliest manifestation of illness (Strauss *et al*, 1974).

We performed a retrospective case study (Resch, 1992) of 74 patients with schizophrenic and schizoaffective disorders (diagnoses according to DSM–III–R; 33 males, 41 females; mean age 16 years), in which premorbid social competence was rated on the Premorbid Adjustment Scale (PAS; Cannon-Spoor *et al*, 1982). Premorbid adjustment was compared with clinical outcome after eight weeks of neuroleptic treatment. Criteria for clinical outcome were the same as those of Pearlson *et al* (1989) (complete remission, partial remission and no response). Raters of the PAS were blind to clinical outcome. Raters of clinical outcome were blind to information regarding premorbid adjustment. Statistical analysis was made by non-parametric analysis of variance. Patients were grouped by their diagnoses and their recovery.

Patients who showed complete remission after eight weeks had significantly lower PAS scores (good social function) during childhood than patients with poorer clinical outcome. The median PAS scores of the schizophrenic and schizoaffective patients with a good outcome were, respectively, 0.25 and 0.23; with partial remission 0.44 and 0.38; and no response 0.46 ($P<0.05$) and 0.42 ($P<0.01$).

In early adolescence patients experiencing complete remission had the lowest median PAS scores: schizophrenic patients 0.43, patients with schizoaffective psychosis 0.38; schizophrenic and schizoaffective patients with partial remission had median scores of 0.73 and 0.70, respectively; and patients with no response to therapy 0.72 ($P<0.05$) and 0.70 ($P<0.05$).

Our study provides additional evidence that social competence is an essential aspect of the development of schizophrenia.

Poor premorbid social adjustment may be a prodromal sign of schizophrenia and prodromal signs may interfere with social adjustment during early adolescence. However, social competence in childhood is a feature of normal child development, before any prodromal signs may occur. Social competence in childhood is of prognostic relevance for the therapeutic outcome of psychosis in youth.

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ICD–10: a neuropsychiatrist's nightmare?

SIR: Lewis (*BJP*, February 1994, **164,** 157–158) claims that five problems have been introduced with the term 'organic' in ICD–10 (World Health Organization, 1992). The author sees this as causing "a neuropsychiatrist's nightmare".

As individuals who took part in the drafting of Section F00-F09, "Organic, including symptomatic, mental disorders", we recognise the value of Lewis' critique and offer readers our comments on the five problems he presents.

(1) Lewis qualifies the ICD–10 explanatory note on the term 'organic' as confusing. We find the way he chooses to quote half a sentence out of context indeed confusing, and fail to see the alleged paradox if the two relevant sentences are read in full (we use italics for the parts of the paragraph omitted):

> "Use of the term 'organic' does not imply that conditions elsewhere in this classification are 'nonorganic' *in the sense of having no cerebral substrate. In the present context, the term 'organic' means simply that the syndrome so classified can be attributed to an independently diagnosable cerebral or systemic disease or disorder.*"

(2) He finds difficulty with the use of the terms 'symptomatic' and 'secondary' in ICD–10, saying that these are often tautologous and inconsistent. In our view, and that of many colleagues whom we consulted around the world, the introduction to the ICD–10 section on organic mental disorders (pp. 45–46) gives a particularly clear statement. It proposes that the organic mental disorders can be

grouped together "on the basis of their common, demonstrable etiology in cerebral disease, brain injury, or other insult leading to cerebral dysfunction". The text then proposes that this dysfunction may be *primary*, as when the brain is directly affected by disease, injury or insult, or *secondary*, "as in systemic diseases and disorders that attack the brain only as one of the multiple organs or systems of the body involved". It goes on to say that "the term 'symptomatic' is used for those organic mental disorders in which cerebral involvement is secondary to a systemic extracerebral disease or disorder" (p. 45). Lewis expresses dissatisfaction with the adjectives secondary, symptomatic and systemic, implying that the ICD–10 text lacks consistency: "So, which is it–'secondary' or 'symptomatic'?" It seems that Lewis has disregarded the heading in bold type on page 42: "Organic, including symptomatic, mental disorders". We cannot understand his dissatisfaction, nor his allegation that the section has a "general sense of wooliness", since the terms in question have been in common usage in European psychiatry for almost a century. We agree, however, with his criticism of the "dangling subclause" because this is an error in sentence construction that is misleading and should have been corrected.

(3) Lewis is right in pointing to the variable temporal relationship between an underlying physical disease and the onset of an organic mental disorder (p. 60). The "weeks or a few months" criterion would be adequate with regard to most of the conditions listed on pp. 60–61, but not in the instance of epilepsy, head trauma, degenerative brain disease, or trypanosomiasis.

(4) As regards the "illogical" and "disingenuous" guidelines for identifying disorders classified in the rubric F06, Lewis' imputation that "the onus is now on the poor diagnostician to investigate fully all cases of schizophrenia or depression or anxiety to exclude the possibility of organic disease" is misplaced. While it is unrealistic to expect that brain imaging technology would be part of the routine investigation of schizophrenic symptoms, it is unwise not to make a provision for recording such information when and where it is available. Ignoring the evidence that many diagnosable cerebral or systemic diseases cause 'functional' psychiatric syndromes (Davison & Bagley, 1969; Propping, 1983) is not the best policy for a psychiatric classification in the 1990s.

(5) Lewis' fifth criticism, which he considers as possibly the most serious, is that the kappa coefficients in the reliability studies were "much lower" for the F06 organic disorders than for their "non-organic counterparts". Here Lewis again quotes selectively from Sartorius *et al* (1993). The kappa for the *entire* F06 group was 0.50, compared, for example, to 0.13–0.48 for 'counterparts' such as schizoaffective disorder, schizotypal states, and other non-organic psychotic disorders. While it is true that the diagnosis of organic depressive state (F06.3) had a low kappa, of 0.27, this was not too different from the kappa for dysthymia (0.35) or for other affective episodes (0.30). The other low kappa in the F06 group, that for organic personality disorder (0.37), was actually higher than the kappas for six out of the nine personality disorders in the F6 category. We wish to point out that the overall kappa for the ICD–10 Organic Mental Disorders section was 0.78, one of the three highest in the entire classification, and notably higher than the kappas for mood (affective) disorders, neurotic and somatoform disorders, disorders of adult personality, mental retardation, and disorders of childhood onset. We fail to see in these data the evidence on which Lewis' "most serious criticism" is based.

Lewis accuses the authors of ICD–10 of seeking too assiduously to separate organic and functional disorders, saying that this constitutes the sin of scholasticism. He fails to appreciate that the collective authors of ICD–10, with their wide international representation (listed on pp. 312–325 of the *Clinical Descriptions and Diagnostic Guidelines*), have fully recognised the illusory notion of such a forced dichotomy by stating in the Introduction that many organic disorders "are symptomatically similar to conditions classified in other blocks" (schizophrenia, affective disorders, and neurotic, stress-related and somatoform disorders). Moreover, the organic/non-organic distinction should not be equated with disorders having or not having a biological basis, as both Lewis and some of the authors of the draft DSM–IV seem to imply.

Finally, we are at a loss to understand the connection made by Lewis between the organic section of ICD–10 and the "embattled image of the World Health Organization itself". ICD–10 is a truly international classification, constructed by obtaining consensus among senior clinicians and research workers worldwide. It was their preference to retain the term 'organic', in no sense implying that all other disorders are less 'biological'. Alternative terms, such as 'cognitive disorders', are not superior, because cognitive impairment is not invariably present in this group of conditions, and there are commonly changes in behaviour as well as in cognition.

While ICD–10 may be a nightmare for one, it provides clinicians and research workers with a nosology based on international consensus and accord.

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A catecholamine model of fatigue

SIR: The chronic fatigue syndrome (CFS) has been proposed as a clinical disorder characterised by prolonged, excessive fatigue and concurrent neuropsychiatric disturbance (Lloyd *et al*, 1988). Phenomenologically, similarities exist with another proposed neuropsychiatric entity, 'atypical depression'. The latter is also characterised by anergia, limb heaviness or weakness and hypersomnia; its clinical validity is argued largely on its preferential response to monoamine oxidase (MAO) inhibitors (Quitkin *et al*, 1988).

To date, no studies have shown that patients with CFS respond to antidepressants. Given the prominence of muscle pain, sleep and mood disturbance in these patients, the proposed role of serotonin in the production of such symptoms (Lopez-Ibor, 1988) and patients' reported sensitivity to the side-effects of tricyclic agents, we chose initially to evaluate fluoxetine. Given the syndromal overlap between CFS and 'atypical depression', we also evaluated the novel reversible inhibitor of MAO-A, moclobemide.

In the first phase of this open evaluation, 15 CFS patients were treated with 20 mg fluoxetine daily for four to six weeks. The dose was increased to 40 mg daily in two patients, as they had reported a partial response. Sixteen other patients were later treated with moclobemide, started at 150–300 mg per day and increased to 450–600 mg. Response was rated 1–5 on a global outcome scale assessing both overall symptom severity and consequent disability. Ratings were made at 4–6 weeks of treatment, or at therapy cessation due to adverse effects.

Of the 15 patients treated with fluoxetine (7 men, 8 women, mean age 40.5 years, range 18–67), 47% (7/15) reported at least some improvement, though only 27% (4/15) showed a significant clinical response (rating $\geqslant 4$). Four patients (27%) stopped the medication because of side-effects (agitation in two). By contrast, 69% (11/16) of patients treated with moclobemide (8 men, 8 women; mean age 34.7 years, range 16–45) experienced at least some improvement, with 56% (9/16) experiencing a significant clinical response. In two patients the rapid development of severe agitation resulted in cessation of therapy.

Treatment trials in patients with CFS have emphasised a significant non-specific treatment effect (Lloyd *et al*, 1993). Caution is therefore required in the interpretation of uncontrolled studies. The trend towards a difference in clinical response rates between the two antidepressants (56% v. 27%; $\chi^2=2.78$, $P=0.095$) cannot be easily explained by non-specific effects. The response rate to fluoxetine would seem to approximate that of placebo in controlled trials. If further studies confirm a reduction in symptoms in response to agents such as moclobemide, which have their principal effects on noradrenaline and/or dopamine levels (as distinct from serotonin) in the central nervous system, then this would support a catecholamine model for fatigue. Further, it would lend support to the hypothesis that CFS differs at a biochemical level from typical mood disorders, which characteristically respond well to selective serotonin reuptake inhibitors such as fluoxetine.

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