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Correspondence

DICHOTIC PERCEPTION IN AFFECTIVE PSYCHOSIS

DEAR SIR,

Yozawitz *et al* (*Journal*, 1979, **135**, 224–37) interpret findings as supporting the hypothesis of right hemisphere dysfunction in affective disorders. However, I note a few shortcomings in this paper.

Their method of arriving at a diagnosis leaves a lot to be desired. They chose 'selected items' from three different schedules and therefore must be using a new schedule which is probably not validated. Next, two raters (perhaps blind to each other's diagnosis) rated the patients as suffering from either schizophrenia or affective disorder. If the two raters disagreed then a third rater came to a diagnosis. Similar results could be obtained by having each of the raters toss a coin after the initial diagnosis had been made.

Unfortunately the authors do not state if their patients have had ECT, and if they had, as is likely in hospitalized patients with affective disorder, then this might well explain their finding of right hemisphere dysfunction (D'Elia *et al*, 1976; and Squire *et al*, 1978).

In view of the above problems I feel that at most this work lends little support to the hypothesis of right hemisphere dysfunction in affective disorders.

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DEAR SIR,

In reply to Dr Griffin, we take the opportunity to clarify procedural details not fully explained in our paper, which we believe to be central to the issues he has raised.

Dr Griffin's concern about the validity of our interview schedule was doubtless due to our less than complete description of its composition. Our protocol contained all of the items from the PSE (Wing *et al*, 1974) and only added those SADS items (Endicott and Spitzer, 1978) and those US–UK items (Cooper *et al*, 1969) which did not overlap with the PSE or with each other. Certainly, no single interview schedule could claim to demonstrate complete validity. The information elicited by any one of the three interview schedules would have been sufficient for clinical diagnosis. Our use of a combined interview schedule, however, permitted a comprehensive assessment with at least as much validity as any of the individual instruments from which it was constructed.

With respect to our procedure for arriving at diagnoses, we take exception to Dr Griffin's contention that similar results could have been achieved with the toss of a coin. Although one rater did initially screen patients to select those with affective or schizophrenic symptoms, the two other independent raters (project psychiatrists who were blind to each other's diagnoses) were not told to restrict their diagnosis to these two alternatives, and did, in fact, use other categories (e.g. unspecified psychosis and schizoid personality disorder). Accordingly, it is difficult to conceive that random probability could have done as well as these project psychiatrists, who had previously demonstrated satisfactory diagnostic reliability in an extensive study of cross-national psychiatric diagnosis (Cooper *et al*, 1972). [For a lark, we followed Dr Griffin's prescription for tossing a coin. The combined index of ear asymmetry for the resulting groups did not differ significantly on the first day ($P > .05$), while the affective and schizophrenic groups in our study did differ significantly on this measure].

In response to Dr Griffin's comment that our affective patients might have experienced ECT, we assure him that they had not. Although we were explicit in stating that histories of brain damage or epileptic seizure were part of our exclusion criteria, we neglected to report that histories of ECT had

also been used as a reason for excluding patients from our sample. However, we never had to reject a patient because of an ECT history. The reasons for this were twofold. We were testing a relatively young depressive population (between the ages of 18 and 35) who were in hospital for only the first or second time. Most importantly, all of these patients were perceived by the hospital psychiatrists as schizophrenic. [We noted, in our paper, the tendency of hospital psychiatrists in large service-oriented institutions in the United States to underdiagnose depressive disorder]. Thus, none of these patients received antidepressant therapy of any kind.

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RAISED MONOAMINE OXIDASE IN ACUTE PSYCHOSIS?

DEAR SIR,

May I use your columns to report an almost fruitless investigation of platelet MAO in schizoaffective patients? We embarked on this study because we believed that this enzyme activity was reduced in schizophrenic patients, and we hoped that it would help to elucidate the diagnosis of our schizoaffective patients. Since the start of the investigation a great deal of evidence has been published to throw doubt on this premise. However, the results may be of some interest.

Preliminary results have been published in a Ciba Foundation Symposium. (Brockington *et al*, 1976: *Monoamine Oxidase and its Inhibitors*, **39**, 353–69).

We measured platelet MAO with tyramine or tryptamine substrates in 56 schizoaffective patients. The definition of 'schizoaffective psychosis' is given elsewhere (Brockington *et al*, 1978, *Journal*, **133**, 162–8). Since we believed that platelet MAO was a stable characteristic we measured it in patients who had already been discharged (N = 32) as well as those acutely ill in hospital (N = 24). Our findings are shown in Fig 1. As expected, females had a

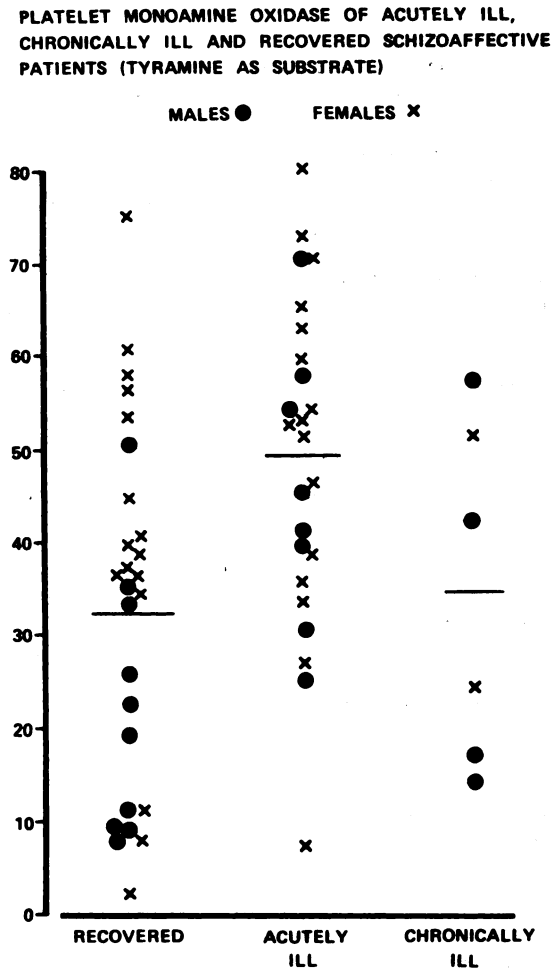

FIG. 1

higher mean MAO (44.3 ± 19.6, N = 34) than males (mean 32.5 ± 18.1, N = 22) (P = <.05). Unexpectedly there was a striking difference between the levels found in acutely ill patients and those in the chronically ill or recovered. The mean for acutely ill patients was 49.1 ± 17.5 (N = 24), while that for the chronically ill was 34.2 ± 18.3 (N = 6) and that for the well patients was 32.2 ± 19.6 (N = 25), one patient being excluded because of uncertain clinical state. The difference between the well and