

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

PLATELET MONOAMINE OXIDASE OF ACUTELY ILL, CHRONICALLY ILL AND RECOVERED SCHIZOAFFECTIVE PATIENTS (TYRAMINE AS SUBSTRATE)

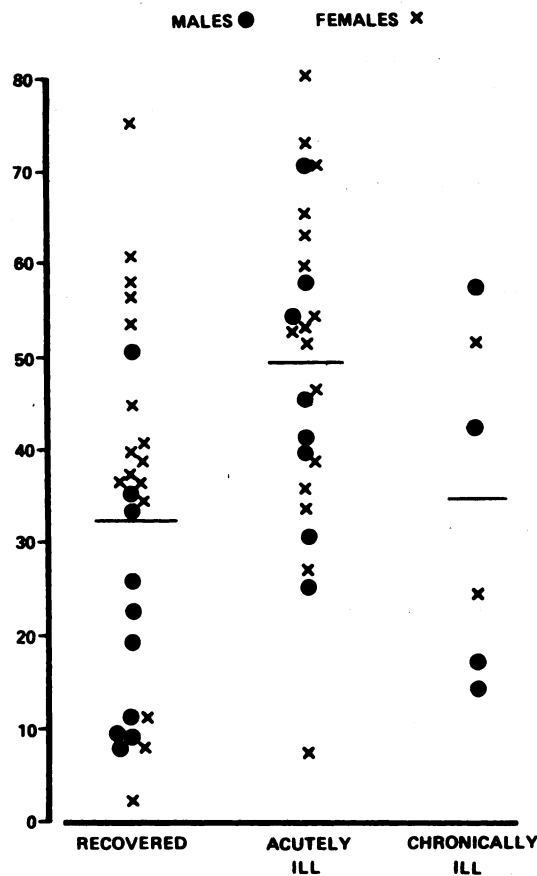


FIG. 1

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

acutely ill patients is highly significant ($P = < .01$).

These differences were not explained by medication. They were present in both 'schizomaniac' and 'schizodepressive' patients and were still evident in all groups after the patients had been given follow-up diagnoses of schizophrenia, bipolar psychosis and unipolar depressive psychosis. They are partly explained by the excess of female patients in the acutely ill group. The mean figure in female acutely ill patients is higher than that in female recovered patients (50.8 ± 18.9 , $N = 16$, compared with 39.3 ± 20.3 , $N = 15$) but not significantly so. In the males the acutely ill patients had significantly higher levels than the recovered patients (45.5 ± 14.8 , $N = 8$ compared with 22.9 ± 14.3 , $N = 10$). A regression analysis of psychopathological variables showed that auditory hallucinosis was significantly related to increased MAO ($P = < .02$), and affective flattening to reduced MAO ($P = < .05$).

Six patients with raised MAO at the time of their illness were studied again after recovery two years later. Five showed substantial falls in MAO from 58.4 to 23.2, 65.7 to 37.0, 54.1 to 36.5, 59.6 to 13.0 and 63.4 to 19.2, while the sixth patient continued to show high levels (70.8, 71.2).

This study, therefore has produced evidence that platelet MAO levels are elevated in acutely ill psychotic patients when compared with similar patients long since recovered. Although this finding was made in a series of schizoaffective patients, we do not think that it can be a characteristic feature of schizoaffective illness itself, because many schizoaffectives behave like typical manic depressives or schizophrenics in their response to treatment and natural history. It could be due to the effects of the psychosis or its treatment in reducing MAO below normal, or it could be due to the elevation of MAO at the time of the illness.

Further longitudinal studies of psychotic patients may prove helpful in resolving the controversy over the activity of this enzyme in psychiatric illness.

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THE CHOICE OF PSYCHOTROPIC DRUGS

DEAR SIR,

I have recently been interested in compiling a list of psychotropic drugs with which to gain detailed experience. In order to do this some guide lines are needed because of the multiplicity of therapeutic

agents now available (e.g. 25 antidepressants).

Consulting standard texts (Alstead and Girdwood, 1978; British National Formulary, 1976-78; Sargant and Slater, 1972; Crammer, Barraclough and Heine, 1978) I have extracted the following general principles.

1. Use drugs with well established and appropriate properties rather than newer drugs.
2. Consider the patient's age and physical state (including pregnancy and breast feeding).
3. Minimize the number of drugs used and beware of drug combinations and interactions.
4. Take into account the patient's life style (including dangerous pursuits such as driving) when considering type, dose and frequency of administration.
5. Consider patient compliance and appropriate route of administration.
6. Avoid drugs to which the patient has had an adverse reaction and prefer those which have previously been of benefit.
7. Where possible, use drugs which are speedy in onset of action and low in side-effects (including addictive potential).
8. Do not neglect the patient's views of medication.
9. Do not use more than one drug of one class at once, and when a drug change is necessary go to a chemically different group.
10. Maintain a self-critical attitude in drug use and do not forget relative costs.
11. Gain experience with a few drugs thoroughly rather than with many superficially.
12. Careful trial of less well established treatments may be justified in refractory cases.
13. If blood levels can be measured, consider if this is of practical benefit.

I would suggest that for practical purposes the above list could be condensed and the scheme thus derived be applied to each type of psychotropic agent (antidepressants, major tranquillizers, etc). My suggestions are as follows:—

1. Choose one or two long established drugs to use regularly ('category one drugs') and study the standard literature about them. Keep abreast of new reports about these. (For major tranquillizers, examples of category one drugs would be chlorpromazine and trifluoperazine).
2. Select for use as few drugs as possible ('category two drugs') to overcome the major practical deficiencies of category one drugs. (For instance, to overcome the cardiotoxicity of amitriptyline one might choose doxepin; another example would be the use of fluphenazine decanoate for schizophrenic patients who will not reliably take category one drugs).
3. In choosing category two drugs prefer the