Brit. J. Psychiat. (1973), 122, 493–6

Correspondence

Letters for publication in the Correspondence columns should be addressed to:
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LAING AND ANTI-PSYCHIATRY

DEAR SIR,

In the *Journal* for November 1972 (121, 563–4), Professor H. J. Walton reviews *Laing and Anti-psychiatry*. He is kind enough to mention that the article which appeared in the *Journal* in 1969 (115, 947–58) by Siegler, Osmond and Mann, discussed Laing's book *The Politics of Experience*.

Unfortunately Professor Walton does not make clear in his review that this article, which, so far as I know, was the first systematic analysis of Laing's position, ends up by rejecting it unequivocally. My colleagues and I would like to underscore the position which we took in 1968, when the article was written.

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THE DYSKINETIC SYNDROME

DEAR SIR,

The paper on 'Tardive dyskinesia' by Turek, Kurland, Hanlon and Bohm (*Brit. J. Psychiat.*, 1972, 121, 605–12) has prompted me to write this letter, as I have an isolated observation that meets with the findings of that paper. However, in this case the dyskinetic syndrome showed itself shortly after the start of neuroleptic medication.

It was a case of a woman, 46 years old, who became an in-patient at the Hospital Psiquiátrico, Oviedo, in November of 1970; the clinical picture consisting of agitation, vivid auditory hallucinations and delusions. She was diagnosed as paranoid schizophrenia and neuroleptic administration was started with chlorpromazine in a maximum dose of 100 mg. t.i.d. The mental state improved after a few weeks and she was discharged with out-patient follow-up. In January 1971, several neurological symptoms manifested, consisting of abnormal orofaciolingual movements of choreic type with rhythmical displacements of the tongue, lip smacking and chewing movements; occasionally the trunk muscles were also involved, with backward and forward movements. The picture resembled closely irreversible dyskinesia; several attempts were made to reduce it; medication withdrawal did not affect it and if anything made it worse, antiparkinsonian drugs were of no use, change to thioridazine, 100 t.i.d. was also of no effect. Finally a combination of haloperidol 2 mg. a day and diazepam 15 mg. a day reduced somewhat the abnormal movements, but they are still present.

I was very much impressed by the devastating effect of such a small total amount of chlorpromazine given only for two or three months, that no doubt produced an irreversible damage in the form of a dyskinetic syndrome. I would appreciate any comments on similar observations.

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PARACHLOROPHENYLALANINE, SEROTONIN AND SLEEP

DEAR SIR,

The paper by Chernik, Ramsey and Mendels, 'The effect of parachlorophenylalanine on the sleep of a methadone addict' (*Brit. J. Psychiat.* (1973), 122, 191–7) is of considerable interest. The authors report their failure to observe the suppressive effects of PCPA on REM sleep previously reported by other workers. In a study of the EEG sleep pattern of six diamorphine addicts (1) we observed a normal REM proportion but an increased REM latency which was positively correlated with the daily dose of diamorphine ($r = \cdot 86$; $p < \cdot 05$).

There is much evidence, recently reviewed by Way (2), that morphine dependence and tolerance in rats and mice is accompanied by increased synthesis and turnover of brain serotonin. There is also evidence, reviewed by Wyatt *et al.* (3), linking serotonin with the genesis of REM sleep. The inhibitor of serotonin synthesis, PCPA, not only selectively decreases REM sleep in humans (4) but also prevents the development of physical dependence to morphine and rats and mice (5). In humans, the ingestion of the serotonin precursors, L-tryptophan and 5-hydroxy-