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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-

tryptophan shortens REM latency (6) which is the opposite of the effect on REM latency observed in our diamorphine addicts. If, as in rats and mice, morphine tolerance and dependence in humans is accompanied by an increased rate of brain serotonin synthesis our observations suggest that this excess serotonin is only partially available for normal cerebral synaptic activity. Methadone has similar effects on brain serotonin (7).

We postulated that our findings support the hypothesis of Collier (8) that physical dependence and tolerance to morphine and related substances are mediated via a blocking action of the receptor mechanism for serotonin at brain synapses. If this be correct it would account for the paradoxical findings of Chernik *et al.*, for as PCPA reduced the available serotonin the parallel reduction in methadone intake described in their report would progressively remove the blockage of serotonin receptors, so that the nett effects could well be the maintenance of equilibrium in the serotonin actually available for synaptic transmission. This would be reflected in the relative stability of the EEG sleep pattern. It would be interesting to know whether the patient's clinical condition also remained stable.

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References

1. Davison, K., and Osselton, J. W. (1972). 'REM sleep in heroin addicts.' Paper presented at First European Congress of Sleep Research, Basle, October 1972. (Proceedings to be published shortly.)
2. Way, E. L. (1972). 'Role of serotonin in morphine effects.' *Fed. Proc.* **31**, 113–120.
3. Wyatt, R. J., Zarcone, V., Engelman, K., Dement, W. C., Snyder, F., and Sjoerdsma, A. (1971). 'Effects of 5-hydroxy-tryptophan on the sleep of normal human subjects.' *Electroenceph. clin. Neurophysiol.*, **30**, 505–9.
4. —— Engelman, K., Kupper, D. J., Scott, J., Sjoerdsma, A., and Snyder, F. (1969). 'Effect of parachlorophenylalanine on sleep in man.' *Electroenceph. clin. Neurophysiol.*, **27**, 529–32.
5. Shen, F. H., Loh, H. H., and Way, E. L. (1970). 'Brain serotonin turnover in morphine tolerant and dependent mice.' *J. Pharmacol. exp. Ther.*, **175**, 427–34.
6. Oswald, I., Ashcroft, G. W., Berger, R. J., Eccleston, D., Evans, J. I., and Thacore, V. R. (1966). 'Some experiments in the chemistry of normal sleep.' *Brit. J. Psychiat.*, **112**, 391–9.
7. Ward, D. F., and Russell, R. L. (1971). 'The effects of chronic methadone administration on brain serotonin synthesis in tolerant rats.' *Fed. Proc.*, **30**, 279.
8. Collier, H. O. J. (1966). 'Tolerance, physical dependence and receptors.' *Adv.Drug Res.*, **3**, 171–88.

CAPACITY TO MANAGE AFFAIRS

Dear Sir,

When medically assessing either a person's testamentary capacity or his ability to manage possessions, it has been customary to do so in relation to the nature of the actual affairs to be handed down or managed. For example, questions such as 'has the patient a reasonable knowledge of his estate' and 'has he the capacity to appreciate who might be entitled to his bounty' can be asked in both instances. Inquiry couched in these terms, however, seems to overlook that affairs themselves can sometimes become too complex for normal people.

Complexity of affairs increases beyond the capacity of some normal people to manage them properly, when such increases do not depend on the skill and effort of the person concerned, e.g. from a chance large win in a lottery or a sizeable unexpected legacy. When this happens to someone who is already mentally ill, but whose capacity to manage has not until then required questioning, medical assessment needs special care. Any pre-morbid (and thus still normal) relative inability should be discounted, only incapacity due to ill health being relevant.

Thus a healthy only child of low normal intelligence, perseverance or emotional control, may become mentally ill but continue to live with and be informally supervised by wealthy parents until inheriting (without restriction) on their deaths. Although implicit in medical assessment, it then helps to keep issues clear if specific reference to illness is made in the questions asked, e.g. 'has the patient's knowledge of his estate been significantly influenced *by mental disorder*' and 'has review of possible beneficiaries suffered *because of mental illness*'?

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A COLLUSION WITH SANITY: A CLINICAL EXTRACT

Dear Sir,

It is an observable fact—which has become exaggerated, unfortunately, into a fashion and a political expedient—that certain kinds of unusual experience and behaviour are conveniently labelled 'mad' in order to legalize the removal of a person from his