PYRIDOXINE AND SCHIZOPHRENIA

DEAR SIR,

I wish to report the following pilot study which was carried out to see whether pyridoxine would be of any therapeutic value in the treatment of chronic schizophrenic patients who had only partially responded to antipsychotic drugs.

Fifteen chronic schizophrenic patients (8 women and 7 men), ages ranging from 41 to 63 years, with a prevalence of age in the forties, and all with a history of long-lasting schizophrenia for which in the past they had been admitted to hospital more than once, were selected. All were free from primary symptoms, but remained rather apathetic, withdrawn, idle and indifferent, showing no interest in their personal habits or their environment. None of them had been working for years, and every attempt made from time to time in the past to induce them to participate in some kind of occupational or vocational rehabilitation programme had always failed. Their maintenance dose of one of the neuroleptic drugs, continued for more than one year, was renewed monthly when they attended the out-patients clinic.

Without any other change in their therapy, during the month of September 1970 pyridoxine was added to their previous drug-regimen in the dose of 50 mg. t.i.d. As the drug was prescribed, the patients were informed that the new pill was 'a sort of vitamin' and they were asked to return to the clinic every other week, instead of once a month.

After 4 to 6 weeks of this neuroleptic-pyridoxine therapy, 8 out of 15 patients reported a certain degree of subjective improvement, claiming to feel more alert and responsive, more active and less anergic. The improvement was only subjective and it was acknowledged only as such, since no noticeable clinical change could be elicited by the physician. As the therapy continued, however, an improvement of their mental status became slowly but progressively more and more apparent, and 8 to 10 weeks after the beginning of the new drug regimen the patients appeared no longer blunted in their affect, nor indifferent to their personal habits and their environment. While in the past most of them had shown complete lack of interest in becoming involved in conversation, now they were willing to talk about themselves and their illness. At the end of the third month of therapy the lack of drive and motivation and the blunted affect had been replaced in 8 patients by feeling of well-being, and they agreed to be referred either to occupational therapy or to a vocational and rehabilitation programme, and in brief became active participants.

While the number of patients treated is too small to be statistically significant and the lack of a control group of patients may cast some doubts on the validity of the results, nevertheless, considering the theoretical and practical implications, it is the writer's opinion that these results warrant further investigation.

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ANTIDEPRESSANTS IN OBSESSIONAL NEUROSIS

DEAR SIR,

The beneficial effects of tricyclic antidepressant medication in patients with obsessional neurosis have previously been reported from this Department (1).

A blind crossover controlled trial is planned to test the effects of clomipramine in obsessional neurosis and in anxiety states with prominent obsessional features. The crossover will take place at six weeks.

Because of the small numbers of new cases with obsessional neurosis that present in hospital practice, it is difficult to undertake trials at a single centre. We would therefore like to explore the possibility of organizing a multi-centre project.

Communication with this Department from those wishing to take part in the trial will be welcome. We would also appreciate notification of patients with obsessional symptoms so that either these can be rated personally, or assistance in rating can be given.

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REFERENCE

1. FREED, A., KERR, T. A., and ROTH, MARTIN (1972). 'Treatment of obsessional neurosis.' *British Journal of Psychiatry*, **120**, 590–1.

CATATONIA FROM FLUPHENAZINE

DEAR SIR,

Long acting fluphenazine has been successfully used in chronic apathetic schizophrenics to increase their working capacity. But in one such case, reported here, the reaction produced was undoubtedly catatonia, and on removal of the drug and with treatment by antiparkinsonian drugs there was improvement in the catatonic as well as the extrapyramidal symptoms, and the patient reverted to his original clinical state.

Case history

J.A., aged 46, was mute, apathetic with a vacant expressionless facies, and often showed impulsive aggressive outbursts. He was unemployable, ate too much and became obese. The case was diagnosed by several psychiatrists as one of catatonic schizophrenia and no special treatment was tried.

In February 1972 his case was reviewed, his diagnosis confirmed and it was decided to treat him by fluphenazine decanoate 12·5 mg. every fourteen days along with orphenadrine 50 mg. t.d.s. and perphenazine 4 mg. t.d.s. For the next three months he did not show any improvement in his clinical state, when his fluphenazine dose was increased to 25 mg. weekly. But within the next two weeks he developed ataxia, and fluphenazine was stopped. His condition deteriorated further, he refused to move even in bed but obeyed simple commands. He showed flexibilitas cerea and refused food and drink. There was excessive salivation and urinary overflow incontinence. The condition was considered to be one of catatonia induced by phenothiazine therapy.

After about three months from cessation of fluphenazine therapy, the patient gradually showed improvement in his condition and started eating solid food. By the next two months the patient was back to his original clinical state namely spending most of his time sitting in a chair, mute, and with an expressionless face. He refused to work, but would eat and dress himself.

Phenothiazines act on the extrapyramidal centres of the midbrain. Catatonic complications of phenothiazine therapy have been described previously. It has been postulated that phenothiazines block the action of noradrenaline (NA) in the midbrain and that there is excessive rise in other catecholamine namely 5-hydroxy-tryptamine (5-HT). In the midbrain area there is normally a higher level of 5-HT than NA, and the result is the production of extrapyramidal symptoms.

It can be assumed that in catatonic schizophrenia the basic 5-HT level of the brain is raised. Fluphenazine decanoate injection rapidly increased the 5-HT level in the midbrain and thereby aggravated our patient's catatonic symptoms.

It may be concluded that long acting phenothiazines are unsuitable for catatonic schizophrenia.

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PARENT-CHILD RELATIONSHIPS AND HOMOSEXUALITY

Dear Sir,

Your November issue ((1972), **121**, 525–8) has a report by Graham Robertson on 'Parent-child relationships and homosexuality'. In it he declares that in our book, *Homosexuality—A Psychoanalytic Study of Male Homosexuals*, my colleagues and I emphasized the mother-son relationship, but, he implies, we did not stress the disturbed father-son relationship as he says Bene did.

Actually, ours was the first study to emphasize and statistically demonstrate the critical importance of the detached and hostile father in the aetiology of male homosexuality. For example: 'We have come to the conclusion that a constructive, supportive, warmly related father *precludes* the possibility of a homosexual son; he acts as a neutralizing, protective agent should the mother make seductive or close-binding attempts' (p. 311). We devoted an entire chapter to the father-son relationship and I would suggest that Robertson should read it. In all my subsequent writings on the subject, I have underscored the central role a disturbed father-son relationship plays in the genesis of male homosexuality.

Robertson also criticizes our study as being based on 'second hand' information. These purported 'second hand' data were given by a group of 77 highly qualified psychoanalysts who answered hundreds of items tapping parent-child relationships in the cases of 106 homosexuals compared with 100 heterosexuals. Each analyst knew his patient's background and history in fine detail. Our volume was published over ten years ago, and since then I have taken careful histories in my psychiatric examinations of more than 800 male homosexuals; this number has included patients representing all socio-economic strata and the major ethnic groups. I have also examined about 30 parent pairs of male homosexuals as well as children and pre-adolescents who were in a high risk population for homosexuality. The findings described in our study were completely supported by my later information.

Robertson, on the other hand, obtained his material from 'the rather limited information available from the case notes' of a group of out-patients of two hospitals. Because of this limitation the author was able to use only 13 items to tap the complexity of parent-child relationships. Clearly, Robertson's methodology is utterly lacking in reliability and rigour, yet he declares that our most carefully researched findings fit 'rather too neatly into a classical dynamic mould, and one suspects a certain degree of bias in the reporting of the analysts'. Further, Robertson falls back on the skewed sample argument: 'It should be recognized that Bieber *et al.* used an abnormal section of the homosexual population for their study, i.e. they relied on reports about only those men who could afford to undergo lengthy psychoanalysis and were thus using an above-average population with regard to educational background.' In 1960 Gordon Westwood reported a study of 127