NEUROPSYCHIATRIC ASPECTS OF BILINGUALISM

DEAR SIR,

I have seen a patient whose case throws further interesting light on G. W. Hughes' review (*Journal*, July 1981, **139**, 25–28) of neuropsychiatric aspects of bilingualism.

He was a Palestinian who left the Middle East at the age of 25, then spent two years in Britain, and then established himself in business in Chile for the rest of his life. He spoke good Spanish, but used Arabic frequently in his family life and business contacts.

During bronchoscopy in Santiago he had cardio-respiratory arrest for at least three minutes, and was then in coma on controlled ventilation for three days. On the fourth day he started muttering unintelligibly. This was regarded as dysphasic or dysarthric speech secondary to brain damage until an Arabic-speaking night nurse realized that he was talking in that language about his youth. He spent about two more days at this stage, but with a further improvement in his level of consciousness he switched from Arabic to a rather faulty English, talking about his time in Britain. This stage lasted for about 24 hours, at the end of which time he was fully conscious and making increasing use of Spanish for communication.

The patient's disability was thus acute cerebral hypoxia and its sequelae, and he was a true bilingual (or trilingual), using at least two languages, and dominant, in the sense that he had great fluency in his native language. In view of the pattern of his recovery, one is tempted to speculate that he was recovering the use of different languages as different areas, or systems, of his brain were recovering from the anoxic injury, rather as memory recovered chronologically.

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NORADRENERGIC OR DOPAMINERGIC ACTIVITY IN CHRONIC SCHIZOPHRENIA

DEAR SIR,

A. A. Schiff and B. C. Shanley (*Journal*, February 1981, **138**, 178) discussed the role of noradrenergic (NA) overactivity in chronic schizophrenia. On the other hand G. W. Ashcroft *et al* and C. D. Marsden (*Journal*, March 1981, **138**, 268–70) discussed the role of dopamine (DA) in schizophrenia. We have recently shown that although the DA-blocking agents (DBA) haloperidol, sulpiride, pimozide and phenothiazine derivatives strongly modified distal colon motility in most non-psychotic subjects (4,100 out of 4,824 = 85 per cent), this effect was registered in only 38 out of 302 (7.9 per cent) of psychotics (Lechin and van der Dijs, 1979a, 1979b, 1981a; Lechin *et al*, 1980a, 1980b).

The 264 noradrenergic-hyperactive psychotic patients fulfilled the Research Diagnostic Criteria of Schizophrenia, whereas the dopaminergic-hyperactive patients were diagnosed as having schizoaffective disorders. Noradrenergic-hyperactive subjects were improved with clonidine, a drug which inhibits release of NA, while dopaminergic-hyperactive subjects were improved with clonazepam, a drug which inhibits release of DA (Lechin *et al*, 1980b; Lechin and van der Dijs, 1981b). The addition of sulpiride, thioproperazine, trifluoperazine, prochlorperazine (DBA), and phentolamine, dihydroergotamine, prazosin (noradrenergic blocking agents) to clonidine and clonazepam, respectively, induced further significant improvements in both types of psychotic patients.

In the light of the physiologic and therapeutic results obtained from our studies, we postulated the existence of two main psychotic mechanisms, one showing hyperactivity of the NA system and the other showing hyperactivity of the DA system. Supersensitivity of DA receptors and of NA receptors, respectively, would accompany the schizophrenic and schizoaffective patients.

With regard to this, it is possible to think that the often postulated overactivity of DA system supposedly present in schizophrenia could be secondary to a lack of DA at the synaptic cleft level (supersensitivity of deafferentation). Similarly, a supersensitivity of NA receptors could be invoked in schizoaffective disorders.

The above facts give rise to the question of the relationship between the nervous system of the gut and the brain. With regard to this, data are accumulating referring to the possibility that the first may be a model for the latter (Fox, 1980; Lechin and van der Dijs, in press).

The fact that nomifensine, a drug lacking peripheral effects, induces distal colon motility changes; whereas domperidone and metoclopramide, drugs lacking central effects, do not induce distal colon motility changes (our unpublished results), reinforces the hypothesis that this intestinal segment tends to behave as a part of the central autonomic nervous system.

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References

Fox, J. (1980) Gut's nervous system: a model for the brain. *Journal of Chemical and Engineering Data*, Dec 1, 32–3.

LECHIN, F., GOMEZ, F., VAN DER DIJS, B. & LECHIN, E. (1980a) Distal colon motility in schizophrenic patients. *Journal of Clinical Pharmacology*, **20**, 459–64.
—— VAN DER DIJS, B., GOMEZ, F., VALLS, J. M., ACOSTA, E. & AROCHA, L. (1980b) Pharmacomanometric studies of colonic motility as a guide to the chemotherapy of schizophrenia. *The Journal of Clinical Pharmacology*, **20**, 664–71.
—— —— (1979a) Effects of dopaminergic blocking agents on distal colon motility. *Journal of Clinical Pharmacology*, **19**, 617–25.
—— —— (1979b) Dopamine and distal colon motility. *Digestive Diseases and Sciences*, **24**, 86–7.
—— —— (1981a) Colon motility and psychological traits in irritable bowel syndrome. *Digestive Diseases and Sciences*, **26**, 474–5.
—— —— (1981b) Clonidine therapy for psychosis and tardive dyskinesia. *American Journal of Psychiatry*, **138**, 390.
—— —— (in press) Intestinal pharmacomanometry and glucose tolerance: evidences of two antagonistic dopaminergic mechanisms in the human. *Biological Psychiatry*.

DRUG SIDE-EFFECTS AND BRAIN DAMAGE

DEAR SIR,

Neuroleptic drugs induce extrapyramidal disturbances which mimic Parkinson's disease, but this is not taken to mean that they can cause Parkinson's disease: a patient whose Parkinsonism continues long after neuroleptic withdrawal is assumed to have been suffering from incipient Parkinson's disease. Involuntary movement disorders are another group of extrapyramidal disturbances and, *a priori*, it would hardly be surprising if neuroleptics induced conditions mimicking these. Such extrapyramidal disturbances would likewise be expected to be reversible on drug withdrawal; if the disorder persisted, the directly analogous inference to be drawn is that an underlying disease process was responsible.

For several years psychological (Johnstone *et al*, 1981; Owens and Johnstone, 1980) and radiological (Weinberger *et al*, 1979) evidence has been accumulating of organic brain damage in chronic schizophrenics unrelated to drug treatment, and further studies (Kleinman, 1981; Andreasen, 1981; Owens, 1981), described at the recent Annual Meeting of the Royal College of Psychiatrists, indicate an association between brain damage, irreversible movement disorder, and negative symptoms in chronic schizophrenic patients. It is thus becoming increasingly apparent that the involuntary movement disorders seen in neuroleptic-treated schizophrenic patients may be categorized as either drug-induced, if drug withdrawal is followed by their disappearance, or as the result of structural brain disease with or without superimposed drug-induced effects, if the movements persist following drug withdrawal.

The term 'tardive dyskinesia' should be reserved for those cases in whom irreversible movement disorders occur late in the course of the schizophrenic or other brain disease processes.

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References

ANDREASEN, N. (1981) Positive and Negative Symptoms in Relation to CT Scan Findings. Paper given at Annual Meeting of The Royal College of Psychiatrists.
JOHNSTONE, E. C., CROW, T. J., FRITH, C. D., STEVENS, M., KREEL, L. & HUSBAND, J. (1978) The dementia of dementia praecox. *Acta Psychiatrica Scandinavica*, **57**, 305–24.
KLEINMAN, J. (1981) CT Scan Studies at N.I.M.H. Paper given at Annual Meeting of The Royal College of Psychiatrists.
OWENS, D. G. C. (1981) Do Neuroleptic Drugs Cause Tardive Dyskinesia? Paper given at Annual Meeting of The Royal College of Psychiatrists.
—— & JOHNSTONE, E. C. (1980) The disabilities of chronic schizophrenia—their nature and the factors contributing to their development. *British Journal of Psychiatry*, **136**, 384–95.
WEINBERGER, D. R., TORREY, E. F., NEOPHYTIDES, A. N. & WYATT, R. J. (1979) Lateral cerebral ventricular enlargement in chronic schizophrenia. *Archives of General Psychiatry*, **36**, 735–9.

SUICIDE IN FAMILIES: DRAW A LIFE-CHART

DEAR SIR,

The suicidal family described by Dr Khin-Maung-Zaw (*Journal*, July 1981, **139**, 68–69) is of considerable interest as an example of violent suicide and non-fatal deliberate self harm in several members of a family extending over more than one generation and including suicide in identical twins. Whether 'it is at least likely that there was a genetic predisposition to violent suicide in the family' does however still seem to be a matter for conjecture on the basis of the evidence presented in the paper, and the interactional effects of illness events may well have been more important than the author suggests.

To illustrate this, I have tabulated the data along the lines of a Meyer life chart which demonstrates how the suicides seem to have triggered the onset of