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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