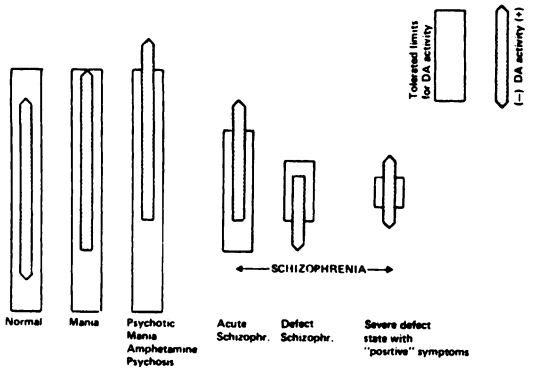

FIG 1.

using the two variables of dopaminergic activity and tolerated limits (Fig 1).

On this model the single abnormality distinguishing the acute schizophrenic syndrome and defect states from other states is a constriction of the tolerated range of dopaminergic activity. Thus situations which increase dopaminergic activity, e.g. stress, may result in psychotic symptoms, acute schizophrenic reaction (reactive schizophrenia, Type 1 schizophrenia).

Further constriction of the range will make attacks of acute symptoms more likely. As the range becomes markedly restricted, dopaminergic activity will fall below the lower limit for normal functioning—this may represent the defect state and in the extreme case there may be no range of dopaminergic activity consistent with normal functioning so that positive and negative symptoms may co-exist at all levels of dopaminergic activity.

Of course this model begs the question as to which neuronal systems are involved in determining the limits of tolerated dopaminergic activity. That these limits exist and show individual variation is demonstrated by the fact that widely different doses of amphetamine are required to precipitate psychotic symptoms in normal individuals.

Constriction of the range might follow selective brain damage as in temporal lobe lesions or Huntington's chorea or some of the cases described by Crow. The possibility, however, exists that there might be two types of constriction—one representing an inherited constriction—the other an acquired constriction.

What practical use is this model? It suggests the possibility that some way might be found to extend the limits of tolerated dopaminergic activity as an alternative to blocking the excess dopaminergic activity in the case of positive symptoms or increasing dopaminergic activity as suggested by McKay in the defect states.

Since we know little of the factors involved in setting these limits, this may seem like looking for a needle in a haystack. However, we do have animal models which may allow us to study the changes of behaviour under dopaminergic stimulation and these may be used to identify means by which the range of dopaminergic stimulation consistent with the preservation of integrated behaviour patterns, may be extended. If the only justification for presenting another model is that it prevents opinions on schizophrenia from being fixed prematurely, then it may be serving a useful purpose.

G. W. ASHCROFT
G. W. BLACKWOOD
J. A. O. BESSON
T. PALOMO
H. L. WARING

University of Aberdeen,
Department of Mental Health,
University Medical Buildings,
Foresterhill, Aberdeen AB9 2ZD

References

JOHNSTONE, E. C., CROW, T. J., FAITH, C. D., STEVENS, M., KREEL, L. & HUSBAND, S. (1978a) The dementia of dementia praecox. *Acta Psychiatrica Scandinavica*, **57**, 305–24.

—— —— —— CARNEY, M. W. P. & PRICE, J. S. (1978b) Mechanism of the antipsychotic effect in the treatment of acute schizophrenia. *Lancet*, *i*, 848–51.

KETY, S. S. (1980) The syndrome of schizophrenia: unresolved questions and opportunities for research. *British Journal of Psychiatry*, **136**, 421–36.

KORNETSKY, C. (1976) Hyporesponsivity of chronic schizophrenic patients to dextroamphetamine. *Archives General Psychiatry*, **33**, 1425–8.

KRAWIECKA, M., GOLDBERG, D. & VAUGHAN, M. (1977) A standardized psychiatric assessment scale for rating chronic psychotic patients. *Acta Psychiatrica Scandinavica*, **55**, 299–308.

DOPAMINE HYPOTHESIS IN ACUTE PSYCHOSIS

DEAR SIR,

Angus Mackay (1980) and Tim Crow (1980) recently discussed in this journal the role of dopamine in schizophrenia, but drew somewhat different conclusions. Both Mackay and Crow favoured dopaminergic overactivity in some region of the brain (possibly in the mesolimbic or mesocortical areas, but probably not in the neostriatum) as important in causing the positive Schneiderian symptoms of the acute psychotic phase of some schizophrenic illnesses (the Type 1 syndrome of Crow). Mackay, in addition, suggested that the defect state of

chronic schizophrenia (the Type 2 syndrome of Crow, or classical Kraepelinian schizophrenia) may be due to cerebral dopaminergic underactivity. Crow, however, thinks that the Type 2 syndrome is more likely to be due to "cell loss and structural changes in the brain". Whatever the cause of chronic Kraepelinian schizophrenia, both authors supported the concept of a 'dopamine hypothesis' to explain the positive symptoms of acute Schneiderian schizophrenia. I believe that such a view of the role of cerebral dopamine in psychiatric illness is too restricted.

The 'dopamine hypothesis' of schizophrenia obviously was a simplistic notion from its birth. The discovery that the neuroleptic drugs used to control acute positive Schneiderian symptoms of schizophrenia all possessed the ability to antagonize cerebral dopamine receptor action led to the concept that over-activity of cerebral dopamine mechanisms might cause schizophrenia. As both Mackay and Crow pointed out, these drugs only control the positive symptoms such as florid delusions, hallucinations and thought disorder of schizophrenia. However, neuroleptic drugs also control similar symptoms occurring in many other types of illness. Neuroleptic drugs frequently are used to control mania. Casualty officers, general physicians, psychiatrists and neurologists have for many years employed neuroleptic drugs to calm patients with acute toxic confusional states (delirium) due to drugs, fever, metabolic encephalopathy, encephalitis, stroke, brain tumour, and so on. Indeed, neurologists sometimes are forced to employ neuroleptics to control the acute toxic confusional state provoked by levodopa in patients with Parkinson's disease. Levodopa also may provoke florid delusions and visual hallucinations in the setting of clear consciousness and preserved insight in some patients with Parkinson's disease, and such symptoms also respond to the administration of a neuroleptic drug. The other plank of the 'dopamine hypothesis' was the discovery that amphetamine could provoke a syndrome akin to schizophrenia in some individuals, but amphetamines also can cause a typical acute toxic confusional state.

Such clinical observations suggest that the current focus of attention on dopamine in schizophrenia is too narrow a view. I suggest that the 'dopamine hypothesis' for schizophrenia should originally have been constructed in a wider context as a 'dopamine hypothesis' to explain the positive symptoms of acute psychotic illness, whatever its cause.

C. D. MARSDEN

Department of Neurology,
Institute of Psychiatry and
King's College Hospital Medical School,
London SE5

References

MACKAY, A. V. P. (1980) Positive and negative schizophrenic symptoms and the role of dopamine. *British Journal of Psychiatry*, **137**, 379–86.

CROW, T. J. (1980) Positive and negative schizophrenic symptoms and the role of dopamine. *British Journal of Psychiatry*, **137**, 383–6.

ANOREXIA NERVOSA IN A DEVELOPING COUNTRY

DEAR SIR,

Anorexia nervosa is rare in Africans and in those of African extraction. The first case in a black child was described by Warren and Vande Wiele (1973). A further case is now reported.

The patient was a 22-year-old Nigerian girl admitted to the University Teaching Hospital of Lagos. Her father, to whom she was very close, had separated from her mother. She blamed the latter for the separation. She had an elder sister. During adolescence she was teased by her peers on account of her precocious physical development. She had married after becoming pregnant while still at school but had separated before the birth of her child. At the time of admission she was living with her mother.

The history began during her pregnancy, six years prior to admission. She attempted to lose weight by reducing her food intake and using purgatives. For the last five months she had refused all food and had induced vomiting after being forced to eat. Her menstruation returned after parturition but then ceased five months before admission. Her normal weight was 56 kilos but on admission was only 30 kilos.

On examination she was mute and emaciated. Lanugo hair was prominent on her back.

The initial management included intravenous fluid replacement and tube-feeding. This was subsequently changed to a 4,800 calorie diet. She reacted to weight gain by self-induced vomiting. Chlorpromazine and cyproheptidine were prescribed to stimulate her appetite. She developed a severe depressive condition which responded to six applications of electroplexy. After an admission of five months she was discharged but was readmitted one month later with further food refusal. During this second admission of one month hypnotherapy was employed. Her final weight was 56 kilos and her menstruation returned.

ABUA NWAEFUNA

A. M. Dogliotti College of Medicine,
University of Liberia,
P.O. Box 589,
Monrovia, Liberia