On the other side, Stein *et al* (1990) systematically evaluated 24 Parkinsonian patients for the presence of DSM–III–R (American Psychiatric Association, 1987) axis I syndromes. Nine subjects had clinically significant current anxiety disorders. These data suggest some form of influence of dopaminergic systems in the pathogenesis of anxiety disorders. Roy-Byrne *et al* (1986) found that patients with panic disorder may have abnormal plasma levels of dopamine metabolite, homovanilic acid. These results seem to concur with the prior assumption.

Many basic and clinical observations support the hypothesis that altered function of central noradrenergic neurones is related to the production of anxiety disorders. Cedarbaum & Aghajanian (1977) pointed out that dopamine decreases the firing rate of the locus coeruleus, the major brain norepinephrine-containing nucleus. This inhibitory action of dopamine would be diminished or absent in deficient states of this neurotransmitter, as in Parkinson's disease or in long-term treatment with neuroleptics. This could constitute a first hypothesis to explain the presence of panic attacks both in Parkinson's disease and chronic schizophrenia treated with neuroleptics.

However, Argyle describes the cases of two patients whose panic attacks were clearly related to psychotic symptoms and became less frequent when antipsychotic medication was increased. An explanation of this discordance could be that high levels of norepinephrine have been found in the cerebrospinal fluid of chronic schizophrenic patients with positive psychotic symptoms (Gomes *et al*, 1980), as well as in those who relapsed when haloperidol was discontinued (Van Kammen *et al*, 1989). This could indicate the existence of a relationship between psychotic activity and hyperfunctional noradrenergic state.

In other words, noradrenergic hyperactivity, dopaminergic hypoactivity, or more probably both, could be the possible mechanisms involved in panic attacks observed in schizophrenic patients.

Finally, we believe that the role of noradrenaline–dopamine inter-relationship in pathophysiology of anxiety disorders deserves attention as a direction for future research.

LUIS M. IRUELA
VICENTE IBAÑEZ-ROJO
SERGIO C. OLIVEROS
LUIS CABALLERO

Department of Psychiatry
Universidad Autónoma de Madrid
Clínica Puerta de Hierro
San Martín de Porres, 4
28035 Madrid
Spain

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Bromocriptine in catatonic stupor

SIR: Catatonia, although it has become uncommon in Western countries, is more common in underdeveloped countries. The latest World Health Organization study (Sartorius *et al*, 1986) reported catatonia in 10% of cases diagnosed to have schizophrenia in underdeveloped countries. The fact that bromocriptine has helped in neuroleptic malignant syndrome (Abbot & Loizou, 1986), a condition said to share a common neurophysiological basis with catatonia (Horn *et al*, 1988), prompted its trial in a case of catatonic stupor.

Case report: MR, a 16-year-old, single, female was admitted in a mute and immobile state. Later she also developed waxy flexibility and maintained imposed postures. She had a recent history of treatment with imipramine in a neighbouring Gulf state, without benefit. She was afebrile and was not dehydrated. Laboratory tests, including blood, serum, venereal disease, liver and renal function, electroencephalograph and computerised tomography, were within normal limits.

Trifluoperazine was tried for a week and when there was no improvement, bromocriptine (2.5 mg twice daily) was introduced. Three days later the patient came out of stupor but became emotionally unstable. Bromocriptine was stopped to rule out the possibility of chance association. After three days she had slipped back into stupor which led to reintroduction of bromocriptine and dramatic improvement for the second time. She started talking and described psychotic experiences, for example, that television announcers were talking about her and they could read her thoughts. Flupenthixol (3 mg t.i.d.) was now started and bromocriptine was reduced over the next two weeks. Her mental state improved gradually and she was discharged eight weeks after admission on flupenthixol (3 mg t.i.d.) and benzhexol (2 mg t.i.d.). Six months after discharge the medicines were gradually stopped which led to reappearance of schizophrenic symptoms. These cleared with flupenthixol (3 mg daily).

Use of bromocriptine in this patient allowed clear differentiation of catatonic stupor from depressive

stupor, which would have been difficult if electroconvulsive therapy (ECT) was used. Though it took her longer to get out of the hospital than the conventionally treated cases, the outcome was satisfactory.

One cannot draw firm conclusions from a single success but it would surely be interesting to try bromocriptine in a series of cases.

TARIQ MAHMOOD

Ibn Sina Hospital
PO Box 6952
Ruwi (Muscat)
Sultanate of Oman

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CORRIGENDA

Journal, November 1990, **157**, 777 (Beeley). The author's name should read Linda Beeley and not Linda Bealey.

Journal, December 1990, **157**, 937 (Scull). The author's name should read Andrew Scull and not Andrew Skull.

Journal, December 1990, **157**, 836–837 (Perugi *et al*). The diagrams above the legends for Fig. 1 and Fig. 2 were switched. The diagram accompanying the legend Fig. 1 should have appeared above the legend Fig. 2 and vice versa.

A HUNDRED YEARS AGO

Hysteria and Organic Disease

As our knowledge of organic disease widens and deepens the number of cases relegated to the indefinite if convenient limbo of "hysteria" will no doubt become fewer and fewer. In a recent number of the *Charité Annalen* the details of a very instructive case in this relation are recorded. The patient was a woman of thirty-one, who, after an attack of typhus fever at the age of twenty-one, began to suffer from a gradually increasing anaesthesia, concentric contraction of visual fields, colour blindness, and disturbance of special senses. The patellar reflexes were present. The manner of the patient was marked by apathy, and sleep was induced by merely closing her eyes. There was much emaciation, the apathy became still more marked, and finally before death she was delirious, with hallucinations and delusions. The case was regarded clinically as one of hysteria, with subsequent mental disturbance, but at the necropsy an astonishing condition of things was found. There was tubercle in the lungs, the larynx, and the intestines, degeneration in the posterior columns of the cord, and myelitis in those columns in the cervical region. Such a condition with retained knee-jerk is certainly unusual. But there were also changes in Clarke's column, a congenital fissure in the medulla oblongata, and degeneration in the nuclei of the cranial nerves, the peripheral nerves showing no change. That so many changes in the nervous system should be present without obtruding themselves in such a way as to make possible a diagnosis other than the unsatisfactory one of hysteria is certainly strange. The case is of great importance, as affording a warning that hysteria is not to be diagnosed without the utmost care in excluding every possible form of organic disease.

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Researched by Henry Rollin, Emeritus Consultant Psychiatrist, Horton Hospital, Surrey.