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Dystonia – a potential psychiatric pitfall

SIR: We welcome the article by D. G. Cunningham Owens (*Journal*, May 1990, **156**, 620–634) since it is most important for the psychiatric community to be familiar with the recent developments surrounding dystonia (Fahn *et al*, 1987; Marsden & Quinn, 1990). Recently, a 42-year-old woman admitted to our psychiatric unit with the diagnosis of conversion hysteria was, after a comprehensive evaluation (including computerised tomography, magnetic resonance imaging, and cerebral angiography), given the diagnosis of dystonia, secondary to an arteriovenous malformation occupying the right basal ganglia. The patient presented with flexion of the fourth and fifth fingers and sustained contraction of the left hand and forearm. She also complained of pain and stiffness in the affected area. Her condition had started five years before admission, while she was going through significant life stress, and during that period she sought the help of several neurologists, neurosurgeons, psychiatrists and orthopaedists. However, she was first seen by a neurologist who, considering her disorder primarily psychogenic, referred her to a psychiatrist.

We agree with the author's opinion that psychiatrists should be cautious in attributing any dystonic abnormality to a purely psychogenic causation. As our case shows, however, we would like to extend this advice to other clinicians, in particular neurologists.

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Primitive reflexes in Alzheimer's disease

SIR: Girling & Berrios (*Journal*, December 1990, **157**, 888–893) report on an uncontrolled study of 146 elderly patients with clinical evidence of Alzheimer's disease and find a correlation between cognitive impairment, frontal lobe signs, including primitive reflexes, and extrapyramidal signs.

We have carried out a pilot study of 133 female patients above the age of 55 in a large psychiatric hospital in order to see if primitive reflexes were associated with cognitive impairment.

The mean age of our population was 78.6 years, similar to the psychiatric ward population of Girling & Berrios, which had a mean age of 80.0 years. We found the following frequencies of primitive reflexes: glabellar tap 81.2%, grasp reflex 52.6%, sucking reflex 30.8%, forced grasping 33%, palmomental reflex 23.3%, snout reflex 26.3%. These reflexes were found more frequently in patients with severe global dementia. When reflex frequency was plotted against age in the severely demented group, there appeared to be a bimodal distribution with a dip in frequency in the 75–79 age range.

These preliminary findings are compatible with those of Girling & Berrios and with the existence of two subtypes of Alzheimer's disease, as postulated by several authors.

However, the experiment should be replicated in a controlled study of a much larger population of patients with clinical evidence of Alzheimer's disease to minimise the possibility of an age effect.

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Panic attacks in schizophrenia

SIR: Argyle (*Journal*, September 1990, **157**, 430–433) studies the occurrence of regular panic attacks in 20 chronic schizophrenic patients, and finds seven cases, a far from negligible amount. The author also reports that neuroleptics may increase panic attacks in some psychotic patients.

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.

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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