

DYSMORPHOPHOBIA

DEAR SIR,

Dr Thomas presented an excellent review of the concept of dysmorphophobia in this *Journal* (May 1984, **144**, 513–516). He also described two cases. The first patient improved dramatically after a surgical procedure on her nose, but the second patient, a 15-year-old male, seemed more refractory to treatment. He clearly met the criteria for dysmorphophobia, and felt that his mouth was deformed, despite the fact that others could not see any abnormality. Dr Thomas mentioned that his current management consisted of encouraging the patient to attend school daily and the therapist making consistent efforts not to become involved in discussions about whether or not his face or mouth was deformed. It appears from this report that these efforts are only partially successful.

We have recently reported two patients with very similar symptoms (Jenike, 1984; Brotman & Jenike, 1984). One responded to the tricyclic antidepressant, doxepin, and the other to the monoamine oxidase inhibitor, tranylcypromine. The first patient was a strikingly attractive 30-year-old married mother of a 3-year-old daughter who was referred for treatment because she had become disabled at work because she believed that she had deep wrinkles on her face. She would spend hours each day examining her face in front of a mirror. The second patient was a 21-year-old single woman who became convinced that her face had swollen to "monstrous proportions." Her symptoms did not respond to imipramine or amoxapine, but remitted within a week after starting tranylcypromine. The first patient continues to do well at 8-month follow-up, and the second patient is asymptomatic and functioning extremely well at almost 1-year follow-up.

In view of these cases, we would recommend beginning patients such as those of Dr Thomas on either a tricyclic antidepressant or a monoamine oxidase inhibitor. If a tricyclic antidepressant trial is unsuccessful, a trial of a monoamine oxidase inhibitor should be attempted. We would be very interested in knowing if Dr Thomas' second patient would respond to such therapy.

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RETROVIRUSES AND SCHIZOPHRENIA

DEAR SIR,

Dr Crow's article (*Journal*, September, 1984, **145**, 243–253) hypothesizing retroviruses as possible aetiological agents in schizophrenia was both cogent and stimulating. In recent months we have tested sera from thirty patients with schizophrenia and schizoaffective disorder (diagnosed by DSM-III criteria) for antibodies to retroviruses HTLV-I, -II, and -III; ten of these patients were from western Ireland and twenty from the Washington, DC area. Sera were initially screened by an ELISA technique using disrupted viral preparations as test antigens. Reactive sera were subsequently assessed for anti-viral specificity by competition or Western blotting assays (Robert-Guroff *et al*, 1982; Sarngadharan *et al*, 1984).

Only one patient had detectable serum antibodies to HTLV-I and one to HTLV-II; none had detectable serum antibodies to HTLV-III. It appears, therefore, that if retroviruses are aetiological agents in schizophrenia then: (1) Their involvement is with a comparatively small subgroup of patients; (2) retroviruses other than the HTLV family are involved; and/or, (3) serum antibodies are not prominent or are non-existent at this stage of the disease, perhaps because the original CNS infection took place *in-utero* before specific humoral immune responses develop (Johnson, 1982). Despite these preliminary negative results, retroviruses are of great theoretical significance as possible agents in schizophrenia and other chronic diseases of unknown etiology that have a familial occurrence.

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