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Correspondence

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The Editor, British Journal of Psychiatry, 17 Belgrave Square, London SW1X 8PG

NAXOLONE IN AMYLOBARBITONE-RESPONSIVE CATATONIA

DEAR SIR,

Classical stuporose catatonia is a rare but dramatic form of schizophrenia characterized by extreme immobility, mutism, negativism and waxy flexibility. Several placebo-controlled studies (Elkes, 1957; Dysken, 1978) demonstrate that some catatonic patients experience a lucid interval following the intravenous administration of sodium amylobarbitone. In contrast, patients with non-catatonic forms of schizophrenia do not show such lucid intervals (Dysken, 1978) nor is there any evidence that oral barbiturates are effective in the treatment of these schizophrenic disorders (Casey *et al*, 1960). This difference in drug response suggests that there may be a partial but specific psychopharmacological difference between catatonic and non-catatonic schizophrenics. Animal studies have demonstrated that catatonic-like states can be induced by the administration of antipsychotic, narcotic, and cholinergic agents (Costall and Naylor, 1973). Recently it has been found that intra-cerebrospinal-fluid injection of β-endorphin in rats produces marked and prolonged muscular rigidity and immobility which is similar to catatonia and is reversed by the opiate antagonist naxolone (Bloom *et al*, 1976). If stuporose catatonia in humans does in fact result from increased functional activity of β-endorphin, then naxolone administration should reverse the catatonic state.

We had the opportunity to conduct a single-blind study of naxolone administration in a 25-year-old man who had catatonia characterized by the above symptoms. He had a history of multiple admission to hospital for chronic schizophrenia, and had developed intermittent catatonic episodes during the past two years. The patient was told that intravenous medication would be given in an effort to help him talk with his doctors. First saline was administered at a rate of 0.5 ml/min; the patient showed no response after 5.0 ml of saline. Next, 20 mg of naxolone was given intravenously over 10 minutes without eliciting any behavioural change. Approximately 10 minutes after naxolone administration ceased, intravenous sodium amylobarbitone was begun at a rate of 25 mg/min. At 250 mg the patient responded dramatically by initiating conversation with his doctors. He appeared animated, answered numerous questions in detail, and stated that he had remained immobile for fear that he might fall and injure himself. Negativism and waxy flexibility were no longer present. This lucid interval lasted approximately two hours, after which the patient lapsed back into the catatonic state.

Our case study suggests that in barbiturate-response stuporose catatonia the catatonic state is not caused by increased amounts of central β-endorphin. If this were the case not only would sodium amylobarbitone reverse the catatonia but naxolone should as well. We recognize that these results are subject to the limitation of a single case; but since cases of catatonia are rare and may involve a specific neuropharmacological mechanism, our failure to reverse the catatonia with naxolone is theoretically significant.

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MAURICE W. DYSKEN
JOHN M. DAVIS

Department of Psychiatry,
University of Chicago,
Chicago, Illinois 60637, U.S.A.

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