

Benzodiazepines and Catatonia

To the Editor:

We read with interest the recent overview on "Benzodiazepines and Catatonia" by Menza and Harris (26: 842-846, 1989). The authors noted that benzodiazepines have an anticholinergic effect (Scatton and Bartholini, 1982), which may be relevant to their utility in the treatment of catatonia. In an effort to evaluate this conclusion, we studied the effect of benzotropine in the treatment of catatonic symptoms in an otherwise medication-free patient with a diagnosis of schizoaffective disorder.

The patient was a 55-year-old woman with a 36-year history of episodes of psychosis and depression, separately and in combination. Past treatments included neuroleptic medication and electroconvulsive therapy. She had functioned well for 5 years on a low dose of thiothixene until 8 months prior to admission. Her behavior then became erratic, her speech at times illogical, and her mood labile. After stopping her medication 3 months prior to admission, her functioning declined substantially, and she was fired from three jobs.

When her neighbors noticed all of her furniture on her lawn, the police were called and found the patient sitting on her basement stairs, mute and unresponsive. She was admitted soon after.

On examination, she was immobile except for occasionally repositioning her legs. She intermittently squeezed the examiner's hand, and looked toward the physician on command. Physical and neurological examinations were normal, although she offered occasional resistance. She tightly closed her eyes and clenched her jaws when examination of eyes and mouth was attempted. If so positioned, she would remain seated or standing for several seconds, but would gradually return to supine posture. She focused on her image in a hand-held mirror if held in front of her.

Laboratory work revealed a decreased potassium level (3.2 mEq/liter), but all other studies were unremarkable including complete blood count, urinalysis, drug screen, FTA, VDRL, arterial blood gases, thyroid function tests, sedimentation rate, electrocardiogram, chest radiogram, and computed tomography of the head without contrast.

The initial impression was that she was catatonic.

To assess the effects of an anticholinergic agent, we administered, in an open design, an intramuscular injection of 2 mg of benzotropine. Initially, this was without effect. Two hours later we repeated the same dosage immediately after which she said, "Thank you." She gradually became more alert and communicative from that point, responded to questions appropriately (first with nods of the head, then verbally), and began eating and drinking. She was very suspicious of the staff and not fully cooperative with questioning or further laboratory work, but all this improved over the following days. She admitted she had been mildly depressed recently over the loss of employment, and that not taking her medication had been a mistake. For the remainder of her hospitalization she was remarkably pleasant although somewhat evasive, but fully cooperative. Thiothixene was resumed, and lithium carbonate was added prophylactically. The catatonia did not recur, her suspiciousness resolved for the most part, and she was discharged in good condition 4 weeks after admission.

The rapid improvement in catatonic symptoms observed in our patient following the injection of an anticholinergic agent is consistent with Menza and Harris' conclusion that the anticholinergic activity of benzodiazepines may be relevant to their utility in the treatment of catatonic symptoms. Furthermore, Kanofsky et al. (1987) have suggested that the negative syndrome may represent an attenuated form of the catatonic syndrome. Tandon and Greden (1989) have implicated increased muscarinic activity as a possible mechanism in the production of negative schizophrenic symptoms. Interpretation must be limited because of the open, uncontrolled design, but our finding of a dramatic improvement in catatonic symptoms in a schizoaffective patient following benzotropine would be consistent with this association. The utility of anticholinergic agents in the treatment of catatonic symptoms needs to be systematically evaluated.

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Response

To the Editor:

This case report is interesting from both practical and theoretical perspectives. We are unaware of any previous reports of such a response of catatonic symptoms to an anticholinergic agent and, insofar as her response can be attributed to benztropine, we agree that this may be of some clinical importance and should be studied more carefully.

We speculated in our paper that the usefulness of low-dose benzodiazepines in decreased dopaminergic rigid/catatonic states such as neuroleptic-induced catatonia and neuroleptic malignant syndrome may lie in their anticholinergic effects. The patient described here appears to have a more typical "psychogenic" catatonia and perhaps the anticholinergic effect of low-dose benzodiazepines also operate in some of these cases.

Tandon and Greden (1989) have suggested that the negative symptoms of schizophrenia may result from a cholinergic/dopaminergic imbalance analogous to that seen in parkinsonian movement disorders. If, as Kanofsky et al. (1987) suggest, there is a relationship between negative symptoms and ca-

tatonia, then we might view catatonia more broadly as a dopaminergic/cholinergic imbalance. This case report is supportive of such a model.

However, as pointed out in our paper, Pfeiffer and Jenney (1957) describe schizophrenic patients who had "lucid intervals" which included marked increases in both motor and verbal behavior in response to muscarinic cholinergic agents. Interestingly, not all patients improved; some developed sobbing and apprehension as they did with amobarbital. These responses would not seem to be consistent with a cholinergic hyperactivity model.

Catatonia is etiologically a heterogeneous illness and it is not surprising that different treatments work in different cases. We look forward to further elucidation of the role of anticholinergic agents.

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