

Correspondence

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

FAILURE OF BROMIDE PSYCHOSIS TO RESPOND TO ECT

DEAR SIR,

Several investigators (Arneson and Ourso, 1965; Mayer-Gross *et al.*, 1969; Carney, 1971) have reported electroconvulsive therapy (ECT) to be effective in treating patients with bromide psychosis. We recently had the opportunity to treat one such patient and found that ECT had no therapeutic effect.

Case Report:

A 35-year-old housewife, who had had a depressive illness treated with ECT nine years ago, was brought by her husband who stated that she had not been herself for the past month: she appeared glassy-eyed and would frequently mistake the day of the week, she had to hold on to the wall while walking in order to keep from falling, she claimed she saw people marching outside the house, and heard intruders. She had difficulty staying awake, and had anorexia and weight-loss. On the day of admission, he found at home empty bottles of 'Sleep-Eze' (each tablet contains 25 mg methapyrilene hydrochloride and 0.125 mg scopolamine hydrobromide) and Miles' 'Nervine' (each tablet containing 292 mg sodium bromide, 292 mg potassium bromide, 32 mg ammonium bromide, 7 mg niacinamide, 1 mg thiamine, 1346 mg citric acid, and 1747 mg sodium bicarbonate). He estimated his wife had taken a combination of about 600 pills during the month prior to admission. There was no evidence that she had recently abused alcohol.

On admission, mood was labile, varying between irritability/perplexity and joviality, her speech was slurred and rambling, and she described visual hallucinations and said the hospital staff was involved in orgies on the ward. She was disoriented. Concentration and memory functions were not testable. Physical examination was normal, except for a facial acneiform rash. There was no evidence for anticholinergic toxicity (mydriasis, tachycardia, hot/dry/flushed skin) or focal neurological dysfunction.

The initial serum bromide level was over 400 mg per cent; all other laboratory values were normal.

A therapeutic trial of physostigmine did not alter her symptoms, and she was given a course of six bifronto-temporal ECTs over two weeks. She remained disoriented, with slurred speech and rambling thought processes, visual hallucinations, and delusional ideas. Serum bromide levels remained above 250 mg per cent. ECT was discontinued and a vigorous medical regimen begun with 3,000 ml intravenous sodium chloride and 6 grams ammonium chloride per day in divided doses, and 80 mg per day of furosemide. Within several days a marked improvement was noted. After ten days her psychosis had cleared and bromide could not be detected in her serum. After recovery from her toxic state, she continued to have depressive symptoms with vegetative phenomena; she responded to three additional ECTs and was discharged in a fully recovered state.

The failure of our patient with bromide psychosis to respond to ECT may be related to her very high (> 400 mg per cent) serum bromide level—higher than levels previously reported in patients who responded to ECT. We did confirm Carney's (1971) finding that when the delirium clears it may reveal another psychiatric syndrome requiring treatment.

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