Catatonia 20 Years Later

TO THE EDITOR: Gjessing and Gjessing (1) described periodic catatonia as a syndrome of reappearing excitement or stupor. Gjessing (2) and Annel (3) reported that lithium carbonate was successful in extending the period between episodes of periodic catatonia. Two reports have described cases of periodic catatonia that responded to lithium carbonate (4, 5). We present the case of a patient with long-standing periodic catatonia who was successfully treated with lithium carbonate.

Ms. A was a 55-year-old woman who upon admission reported having experienced bizarre and paranoid behavior, disorientation, amnesia, and reduced responsiveness over the previous month. She was in a deeply stuporous state and was diagnosed with schizophrenia. Her course in the hospital was characterized by episodes of greater motor activity, which would alternate with stuporous periods that would last several days. Results of a physical examination, computerized tomography scan, and an EEG revealed no abnormality. Her symptoms resolved after six ECT treatments.

Ms. A had had 14 admissions over 13 years that had been similar in presentation. Results of physical examinations and investigations that included thyroid function tests repeatedly showed no abnormality. A regimen of sodium amytal, 450 mg i.v., resulted in temporary symptomatic improvement but did not elicit additional information. She continued to respond to ECT but did not respond to maintenance treatments of haloperidol, 4 mg t.i.d.; pimozide, 2 mg t.i.d.; loxapine, 37.5 mg/day; amitriptyline, 100 mg h.s.; tranylcypromine, 10 mg t.i.d.; or various benzodiazepines. All medication trials had been longer than 4 weeks' duration. She had developed cholestatic jaundice while on a regimen of thioridazine. Ms. A was treated with lithium carbonate, 300 mg t.i.d. Her catatonia resolved and has not recurred in 7 years.

The possibility that remission of symptoms was coincident with the natural resolution of the illness is unlikely, considering the protracted course, the remarkable response, and the subsequent prolonged remission. In retrospect, a trial of maintenance ECT could have been attempted, given the poor pharmacological responses.

Abrams and Taylor (6) demonstrated that catatonia exists in patients with schizophrenia, mood disorders, and organic disorders. A study by Benegal et al. (7) suggests that catatonia can exist independent of any known disorder. Fink (8) has argued for a separate diagnostic classification for catatonia.

The clinical presentation was not consistent with a typical mood disorder, and symptoms were resistant to neuroleptic treatment. Although this does not preclude catatonia due to schizophrenia, the cyclical nature of symptoms and response to lithium carbonate suggest catatonia due to an atypical mood disorder, or a separate idiopathic etiology.

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Vitamin C for Paroxetine- and Fluvoxamine-Associated Bleeding

TO THE EDITOR: Paroxetine is a selective serotonin reuptake inhibitor (SSRI) that is widely used for treatment of depression, obsessive-compulsive disorder, and panic disorder. There have been several reports of bleeding abnormalities connected with the use of SSRIs (1–4). I report here the case of a patient with panic disorder who developed bleeding related to the use of paroxetine and fluvoxamine.

Ms. A was a 33-year-old woman who had been suffering from panic disorder with agoraphobia for 2 years. Panic symptoms had begun after her father died. At that time she was also under considerable stress because of marital problems. At first she was successfully treated with a regimen of clomipramine, 100 mg/day. Because of persistent side effects, her medication regimen was changed to paroxetine, 40 mg/day. She was also being treated as necessary with lorazepam (1 mg) and temazepam (10 mg). The medication was well tolerated, and within months the symptoms had subsided. However, she complained of subjective agitation, a side effect that is quite commonly seen with paroxetine. Two weeks after she had started paroxetine treatment, she mentioned spontaneous bruising on her arms and legs and excessive menstrual blood loss, but she did not connect these with the medication. She had never had these complaints before. Gynecological examination did not reveal any abnormalities. Prothrombin time was 18 seconds (reference value: <24), and partial thromboplastin time was 41 seconds (reference value: <40). Results of other hematological laboratory tests were normal, and she was in good health. It did not seem that the marginal increase in the prothrombin time was a satisfactory explanation for her tendency to bleed, especially since there have been no reports in the literature about a relation between SSRI-associated bleeding and abnormalities of blood coagulation. Ms. A was given 500 mg/day of vitamin C; this caused the bleeding to stop within 3 weeks. Subsequently, she was asked to stop the vitamin C, which led to a gradual recurrence of ecchymosis and excessive menstrual blood loss. Shortly afterward the paroxetine regimen had to be discontinued because of her subjective feeling of agitation; she was then given fluvoxamine, 150 mg/day. The agitation ceased, as did the bleeding. However, the bleeding soon began again, and Ms. A was advised to resume taking vitamin C; once again the bleeding stopped.

The idea for using vitamin C was based on the knowledge that long ago citrus fruit was used in the Dutch navy to reduce the bleeding in sailors who had scurvy. There is one case report in the Dutch medical literature in which the use of vitamin C stopped bleeding related to SSRI medication (5). The main