gether with valproate because the latter decreases the clearance of lamotrigine.

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Electric Sensations: Neglected Symptom of Escitalopram Discontinuation

TO THE EDITOR: We make reference here to a patient who experienced electric sensations after discontinuing escitalopram treatment.

Ms. A was first referred to our clinic by her family doctor for depressive symptoms (asthenia, insomnia) in the context of a psychosocial stress situation. Treatment with citalopram, 20 mg/day, was initiated, which was soon replaced by escitalopram, 10 mg/day, the enantiomer of citalopram. Because she was responding well, Ms. A reduced her dose to 5 mg/day and, 3 weeks later, stopped treatment altogether. About a week later, she began to experience electric shock-like sensations or visual flashes lasting for about 1 second each. This was followed by a phase of spatial disorientation that lasted for about 30 seconds and was experienced as highly unpleasant and frightening. The sensations were only felt when Ms. A was in an upright position; she had no history of loss of tonicity. These episodes occurred up to three times a day over a period of 2 weeks. Prodromal symptoms or specific triggers were not reported. While Ms. A was taking citalopram/escitalopram, no side effects were observed. Interactions with other substances could be eliminated because this was her only medication.

Ms. A's depressive symptoms soon returned, resulting in more therapy with escitalopram (10 mg/day). She began to feel better and thus decided to reduce her medication, although it was well tolerated, to 5 mg/day and, after 4 weeks, again ceased taking it. One week later, the electric shocks reappeared, although they were now less intense. Ms. A's family doctor continued to urge pharmacological intervention to treat her depression. However, she refused to cooperate out of fear of becoming addicted to antidepressants because of the sensations that she interpreted as withdrawal symptoms. An EEG as well as a detailed neurological examination revealed no abnormalities.

There is not much relevant literature on the subject of electric sensations as a discontinuation symptom of selective serotonin reuptake inhibitors and nothing at all in connection with citalopram or escitalopram, although comparable symptoms have been described as occurring after treatment with paroxetine and sertraline (1). The pathophysiology is not clear; the down-regulation of serotonin 5-HT_2 receptors and the desensitization of both the 5-HT_2 receptor transmembrane signaling system and the 5-HT autoreceptors seem to be of particular concern (2). There are two therapeutic options: resuming the medication and tapering it off even more slowly (2) or allowing the syndrome to run its course. However, the patient would need to be reassured that the symptoms are likely to disappear within 2–8 weeks without serious consequences. None has been reported so far (3).

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Aripiprazole-Induced Movement Disorder

TO THE EDITOR: Aripiprazole is a dopamine D_2 partial receptor agonist (1). Trials have shown rates of extrapyramidal side effects similar to those of placebo administration, as seen with other atypical antipsychotic agents (1). Some case reports have found that atypical antipsychotics may even be helpful in treating tardive dyskinesia (2, 3). Although the occurrence of extrapyramidal symptoms seems to be less frequent with atypical antipsychotics, it is important to note that these effects can still occur. To our knowledge, only one case of aripiprazole-associated dyskinesia has been reported (4). We report a case of pseudoparkinsonism and rabbit syndrome that occurred in an antipsychotic-naive patient during treatment with aripiprazole.

Ms. A was a 27-year-old woman diagnosed with bipolar I disorder. Before she received care through our clinic, her condition was maintained with 150 mg/day of extended-release bupropion, 100 mg/day of lamotrigine, and 75 mg/day of extended-release venlafaxine. She had an emergence of manic symptoms and was administered aripiprazole, 10 mg/day. Ms. A had never been treated with an antipsychotic in the past. One month later, her dose of aripiprazole was increased to 20 mg/day. During the course of the next month, she was tapered from lamotrigine and venlafaxine to consolidate her drug regimen. She was seen for follow-up, where she complained of muscle stiffness and tongue movements that had grown increasingly worse over the last 2 months. She was observed by her outpatient psychiatrist to have muscle rigidity, a masked face, a shuffling gait, and orofacial movements consistent with rabbit syndrome.

It was decided to taper the aripiprazole and start ziprasidone, 60 mg b.i.d., along with benztropine, 1 mg b.i.d. Ms. A was seen 5 days later with a marked improvement in symptoms. She had no orofacial movements, decreased muscle rigidity, and only a slight tremor. At this visit, aripiprazole was completely stopped, and ziprasidone was increased to 80 mg b.i.d. Two weeks later, Ms. A was seen with no muscle rigidity or orofacial movements. Benztropine was discontinued, and her condition was maintained with ziprasidone without any reemergence of symptoms. When we evaluated these events with the

probability scale of Naranjo et al., the case was ranked as a probable adverse drug reaction (5).

This case illustrates that the risk of developing extrapyramidal symptoms is still present, even with the newest of atypical antipsychotics. Clinicians should vigilantly monitor all patients for the emergence of symptoms, regardless of which antipsychotic a patient is receiving.

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Fatal Agranulocytosis 4 Years After Discontinuation of Clozapine

TO THE EDITOR: Clozapine is currently considered the most efficacious antipsychotic for the treatment of schizophrenic symptoms (1), but its use is limited because of the risk of agranulocytosis. Although the risk of this life-threatening adverse event is highest during the first 4 months of administration, it was recently reported that it can occur even after 11 years of continuous treatment (2). To our knowledge, it has not been reported that agranulocytosis can occur several months or even years after the discontinuation of clozapine. Here is the case of a mentally retarded patient who developed agranulocytosis after 7 years of clozapine treatment and then continued to suffer continuously from severe neutropenia, which developed into fatal agranulocytosis more than 4 years after the discontinuation of clozapine.

Mr. A was a 49-year-old man of Finnish origin who had been treated in a local nursing home because of behavioral problems associated with moderate mental retardation. Severe aggressive behavior was a major problem in his daily life; therefore, clozapine treatment was started. Clozapine, 450 mg/day, resulted in a marked reduction in his aggressive behavior, but it was discontinued because of agranulocytosis. Mr. A started to suffer from severe recurrent infections, and treatment with granulocyte-colony-stimulating factor gave only temporary benefits. His hematologist concluded that his blood dyscrasia was chronic because his total WBC count fluctuated from 0.5 to 1.5 $\times$ 10^9/liter (normal WBC count range=4.0–10.0 $\times$ 10^9/liter). At that time, it was decided to administer palliative treatment in the familiar environment of his nursing home. Mr. A's prognosis did not improve, and he died a few weeks later. A forensic autopsy concluded the cause of death to be clozapine-induced myelodysplasia of the bone narrow. Because of a complaint by a relative, the National Authority for Medico-Legal Affairs asked for an expert's opinion on whether the pharmacological treatment was adequately administered.

To our knowledge, this is the first reported case of clozapine-induced agranulocytosis to have occurred several years after the discontinuation of clozapine treatment. In this case, agranulocytosis was not caused by a reversible, acute toxic or immunological reaction in bone narrow but was a consequence of a permanent change in the maturation of blood cells, leading to myelodysplastic syndrome.

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Review of *Interpersonal Reconstructive Therapy*

TO THE EDITOR: The book review of *Interpersonal Reconstructive Therapy: Promoting Change in Nonresponders* (1) made the point that both the structural analysis of social behavior and my therapy approach—interpersonal reconstructive therapy—have some substance but are too complex to be understandable or useful. The reviewer is not alone in reaching that conclusion. However, the same sentiment about the structural analysis of social behavior was expressed more positively by Jerry Wiggins (2), who wrote that the structural analysis of social behavior "is the most detailed, clinically rich, ambitious, and conceptually demanding of all contemporary models." Of course, reviewers are free to express any opinions they wish, but if their conclusions are to be fair and if readers are to be able to determine whether they agree or not, readers deserve an accurate representation of the material.

Please consider the following two distortions of fact in the review. The first is the following:

> Despite Benjamin's efforts to use the structural analysis of social behavior for research purposes, it has proven too complicated and cumbersome and never gained widespread application. The few research studies using Benjamin's coding system focused primarily on interactions in psychotherapy. I too have felt that the structural analysis of social behavior is an intriguing foreign language, but not terribly practical; and I never did learn the language.

There have been many publications based on the structural analysis of social behavior, and the range of topics is broad. A list of known publications is available from the University of Utah (http://www.psych.utah.edu/benjamin/sasb/index.html) by request. One published review of uses of the structural analysis of social behavior involves a series of articles that appeared in the *Journal of Consulting and Clinical Psychology* in December 1996. Another is a review of articles about the structural analysis of social behavior focused on psychotherapy by Constantino (3). In April 2006, the *Annual*