toms on two separate occasions strongly suggests a causal connection between sudden carbamazepine cessation and mania. Of interest, a similar case of rebound mania associated with carbamazepine withdrawal has been reported in a 30-year-old woman being treated for epilepsy (5). As with our patient, this patient also had no previous history of mood disorder. Taken together, these reports should alert clinicians to the possibility of serious mood disturbance in patients who stop taking carbamazepine suddenly, even when they do not have a prior history of mood disorder.

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Vardenafil Reversal of Female Anorgasmia

TO THE EDITOR: Selective serotonin reuptake inhibiting (SSRI) antidepressants commonly produce iatrogenic sexual dysfunction (1, 2). It is uncommon for spontaneous remission of this side effect to occur, even after taking the SSRI for years (3). A variety of augmentation strategies have been proposed to reverse SSRI-induced sexual dysfunction (4). Sildenafil has been used to reverse SSRI-induced anorgasmia in a woman (5). Vardenafil, a phosphodiesterase type-5 inhibitor, is indicated in the treatment of male erectile disorder. I report here a case of SSRI-induced anorgasmia in a woman that was reversed by vardenafil.

Ms. A was a 37-year-old Caucasian woman who was successfully treated for panic disorder without agoraphobia and for generalized anxiety disorder with sertraline, 100 mg/day, for over 2 years after difficulty tolerating trials of alprazolam and clonazepam. Unfortunately, she developed anorgasmia from the sertraline within 3 months of reaching this dose. A dose reduction to 50 mg/day led to relapse, although her anorgasmia improved. Sildenafil augmentation 1 hour before sexual activity reversed her anorgasmia but only at the 100-mg dose; the 50-mg dose was ineffective. The anorgasmia persisted if she forgot to take sildenafil. The cost, however, was prohibitive because the drug was not covered by her health insurance. She was interested in trying vardenafil instead because it cost less than sildenafil. She found that vardenafil in the 10-mg strength was not only effective in reversing anorgasmia but was also more affordable because she could break the 20-mg pills in half. Ms. A could not detect any difference in the onset of action, the duration of effect, or adverse reactions from vardenafil compared to what she felt while taking sildenafil. Vardenafil has continued to be effective when she uses it once or twice a week for 9 months to date without any difficulty tolerating it.

This case report describes a woman with SSRI-induced anorgasmia and no other sexual complaints who had this side effect reversed by vardenafil. It is possible that this response was a placebo effect, although it is unlikely since this side effect persisted with a low dose of sildenafil. Larger-scale placebo-controlled studies would be helpful in determining the effect size and whether other opportunities exist for using vardenafil to reverse SSRI-induced sexual dysfunction.

At this point, vardenafil has been approved by the Food and Drug Administration only for use in men. Further studies in women with primary or secondary sexual dysfunction may reveal other populations and medical conditions that are responsive to vardenafil treatment. This report suggests that augmentation with vardenafil may assist some patients with SSRI-induced sexual dysfunction.

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Catatonia in Juvenile Corrections

TO THE EDITOR: The following are reports on three male youths who were diagnosed with catatonia while residing in an intensive mental health unit within a juvenile correctional facility.

Abe, a 14-year-old African American youth who was diagnosed previously with both schizophrenia and bipolar disorder with psychotic features, was admitted because of paranoid delusions that his food was being poisoned and subsequent inadequate food intake, with a 10-lb weight loss over a 4-week interval. He demonstrated repeated inappropriate touching and maintenance of assumed positions for up to 10 minutes at a time. He was extremely combative at times but immobile at others. He exhibited mutism, stereotypy, mannerisms, negativism, and staring. His Bush-Francis Catatonia Rating Scale score was 26. (1) Abe was treated with lorazepam, 2 mg/day, and his catatonic signs and food intake improved dramatically over the following 5 days. He was treated with quetiapine and lithium for the next 18 months, with partial remission of his mood and psychotic symptoms until he became noncompliant. Abe's catatonic signs reemerged, and he again

responded favorably to lorazepam, 2 mg/day. For the past year, he has been receiving treatment with clozapine and lithium and remains in full remission of catatonic, mood, and psychotic symptoms, with dramatic improvement in social, interpersonal, and educational functioning.

Mr. B, an 18-year-old Caucasian man with a history of cannabis abuse and declining social, vocational, and interpersonal involvement (but no previous diagnoses of mood or psychotic disorder), was admitted because he was talking to himself, had lack of motivation, and was laughing inappropriately. He exhibited profound mutism, intermittent excitement, posturing, staring, mannerisms, stereotypy, perseveration, autonomic abnormality (elevated blood pressure), automatic obedience, and impulsivity. His Bush-Francis Catatonia Rating Scale score was 24. His catatonic signs responded well to lorazepam, 3 mg/day. Manic symptoms subsequently emerged, and Mr. B was diagnosed and treated for bipolar disorder.

Carl, a 17-year-old Caucasian youth who was previously diagnosed with schizoaffective disorder, was admitted because of declining self-care, response to internal stimuli, and bizarre delusions. He displayed facial grimacing that resolved completely when risperidone, 5 mg/day, was discontinued. His Bush-Francis Catatonia Rating Scale score was 18, and his catatonic signs included immobility, mutism, excitement, posturing, staring, mannerisms, echolalia, stereotypy, negativism, gegenhalten, ambitendency, impulsivity, and combativeness. These signs resolved with lorazepam treatment, 3 mg/day.

All youths had negative serum toxicology screens upon admission to the facility and, in the case of Mr. B, for 6 months before admission, as verified through court-ordered monitoring. The youths received medical and neurological evaluations, including hematological, metabolic, toxicological, and CSF analysis, EEGs, and neuroimaging. All results were normal or lacked positive findings. Prenatal and developmental histories were unremarkable, although Abe and Carl had extensive family histories of mental illness.

There have been several case series and reports of catatonia occurring in the child and adolescent population (2). Although substantial psychiatric morbidity has been identified among youths in the juvenile justice system (3), I am unaware of previous case reports of catatonia occurring among youths detained within the juvenile justice system. Presumably, the etiology and risk factors leading to catatonia in adolescents and young adults in juvenile justice and community settings is similar, regardless of criminal history. However, the identification of this syndrome in male juvenile offenders is especially important, given the paucity of resources for adolescents and the increasing recognition of the prevalence and severity of mental illnesses among juvenile offenders (4). Incarcerated juveniles may exhibit unusual behavioral phenomena, making detection of psychiatric disorders in need of treatment extremely difficult (5).

Considerable functional improvement was evident in all three cases after treatment of catatonia, similar to documented case reports. Also, more youths are entering the criminal justice system than ever before. With more diverse clinical treatment settings and declining resources, greater awareness of the catatonia syndrome, with its well-defined features and response to treatment (6), may aid in its recognition and management.

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Overlap Between Alexithymia and Asperger's Syndrome

TO THE EDITOR: It seems to us that there is a significant overlap between alexithymia and Asperger's syndrome. The term "alexithymia" was coined by Sifneos in 1972. It is derived from the Greek, with *alexi* meaning "no words" and *thymia* meaning "mood or emotion." Patients with alexithymia have great difficulty or are unable to describe their feelings and can have problems making sophisticated differentiation of one feeling from another. Their communicative style shows markedly reduced or absent symbolic thinking (1). As Warnes (2) pointed out, they have "a paucity of fantasies" and "lack the capacity for introspection." They are preoccupied with the "minute detail of external events…[and] are unable to make connections between events, affective arousal and somatic response." Nonverbally, they are "stiff and wooden." They are "mechanical in their object relations." Alexithymic individuals give flat, shallow descriptions of others that lack "psychological counters" (2).

All of these features also fit descriptions of Asperger's syndrome (3), in which the main difficulties are understanding one's own and others' emotions, having problems expressing oneself with nonverbal behavior and in reading that of others, and having a propensity for hypochondriacal features. They also have difficulty with the "theory of mind" and in predicting the cognitions of others. Their imagination is limited. They tend to have a preoccupation with factual information and are strong in areas such as mathematics, engineering, and computers but can have significant problems with interpersonal relationships.

It appears to us that from a clinical perspective a diagnosis of Asperger's syndrome should be considered in patients with alexithymia.

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