results and those from Roth and coworkers.[7] They evaluated insomniac individuals only (none with dementia) and observed significant impairment of short-term memory and verbal learning by assessments done in days 1 and 7 after onset of drug administration. It should be considered that short-term studies may not result in full development of the therapeutic effects of a drug with the inconvenience of revealing first-hand, usually transient adverse effects.[15] Also, cognitive and psychomotor benefits experienced by healthy and demented volunteers can vary in magnitude and relevance in perspective of the nature and intensity of the impairment, ranging from substantial gains in performance (sustained or not) to a flatter slope of decline.[17] In our scenario of a minimum follow-up of 3 weeks (including 2 weeks under intervention), witnessing no worsened performance at all among trazodone-treated compared to placebo-treated counterparts may contribute to assume trazodone as a safe drug in the context of cognitively compromised patient. To witness no change in cognitive functions regardless of the benefits of trazodone use in sleep parameters suggests that gains in the primary target do not necessarily translate into improvement of secondary goals, at least not in the settings described. Moreover, instructions regarding attainment to important sleep hygiene principles were discussed by the medical physician with all volunteers' caregivers upon start of the interventional stage of the study. But our data reveal no change in the cognitive measures as result of these recommendations (placebo group), prompting for the discussion of whether behavioral strategies alone are to be considered before pharmacological intervention.

To our knowledge, no study has examined the effect of trazodone on cognition in patients with AD. Among the limitations of our study, one may include that participation was restricted to AD patients with SD, being therefore difficult to extrapolate our findings to other dementia. Also, despite the rigorous execution of the study, assessing all possible cognitive dimensions was beyond the scope of our design, and make room for subsequent analyses. At last, the rather small and nonprobabilistic sample size could be viewed as a limitation.

In conclusion, this study provides evidence that usage of trazodone 50 mg does not impact cognition after a 2-week treatment period of AD patients. More studies are needed to measure effects of trazodone on other cognitive dimension in older adults.

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Catatonia in Outpatients
Analysis of a Single Clinical Practice

To the Editors:

Our literature about catatonia, since the clinical designation of the condition in 1874, is drawn, with rare exception,[1–4] from the scrutiny of hospitalized patients. Whether conclusions reached thereby represent catatonia in the world outside the hospital is almost untested, especially in adults. The current report is presented as a query of such conclusions in another population entirely, one of outpatients in a practice of general psychiatry.

The record of every patient in the practice was surveyed; all evaluations were carried out by the author alone. Information about the means for diagnosis, the practice, and the community in which the latter is embedded is given in Supplemental Digital Content 1 (http://links.lww.com/JCP/A251).

The total number of outpatients examined and observed during the study

period is 298 (age, 16–89 y; mean and median, 46 y; 153 females and 145 males). *Catatonia* has been diagnosed at some time during the course of 8 (2.7%) of these: 4 men and 4 women aged 24 to 67 years at the time of diagnosis. In 2 instances, the diagnosis was made or suspected during the intake evaluation. In no patient had it been made earlier by another provider. In retrospect, it might have been made at intake in 2 of the patients but was not. In Table 1, the characteristic features of catatonia are presented in 5 categories. The features were required to have had an onset, at that time to be *new* or *considerably accentuated*, to be considered catatonic. The diagnosis was difficult in only one instance, once it was considered, and here, a clear response to benzodiazepine was confirmatory.

Note the frequency with which catatonia is chronic (if the disorder is untreated or if benzodiazepine is not given coincidentally for another reason). In no patient for whom benzodiazepine was administered for catatonia was there a failure to respond, even if the features previously had waxed and waned chronically (benzodiazepine has been continued indefinitely in these patients, and a relapse that could not be relieved through dose adjustment did not occur in any of them); sometimes,

considerable dose adjustment has been required (Case Report, Supplemental Digital Content 1 (http://links.lww.com/JCP/A251), and in 2 cases, the dose needed proved to be relatively high (lorazepam, total of 10 mg daily in 1 instance; diazepam, total of 30 mg daily in another). In the 2 patients with autism spectrum disorder (ASD) whose catatonia was of an acute pattern for whom benzodiazepine was tried and effective, the drug could be successfully tapered off some months later in 1 patient (severe environmental stress had been the triggering event for catatonia) and the other patient was lost to follow-up.

Five of the 8 patients with catatonia are high school graduates (3 patients have an ASD). In 2 patients, the terminal degree is a bachelor's degree (both patients have an ASD), and in 1 patient, it is a doctorate degree (the patient has an ASD); 1 is a graduate of a 2-year course in a community college. Four patients are employed; the fifth patient is a competent homemaker. The other 3 patients have mild or pronounced intellectual impairment (all patients have an ASD); none of the patients is employed gainfully enough for independent living.

The most surprising finding within this report is the prevalence of ASD, or likely ASD, as the underlying or predisposing condition for catatonia: 6 of the

8 (Table 1). In none of the patients with ASD did catatonia emerge as a complication of depression. *Autism spectrum disorder* has been diagnosed or considered likely in 26 (9%) of the 298 patients in the practice, a diagnosis suspected before the patient entered the practice in only 3 patients. Catatonia has therefore complicated the course of 23% of these patients with ASD. A case report for 1 of the 8 patients, who has experienced both ASD and catatonia, is included in the Supplemental Digital Content 1 (http://links.lww.com/JCP/A251); it illustrates several of the observations previously described.

DISCUSSION

Several current authorities on catatonia suggest that a disorder of mood, especially mania, is the most prevalent comorbid condition.[1–4] Yet, ASD, which is not a disorder of mood, is the most common precondition for catatonia in adults if the practice described here typifies the nonhospitalized world. Of course, it may not so typify that, but at the least, we may say that it is not true everywhere that most catatonic adults have a mood disorder: ASD is very possibly much more common in such patients than the study of hospital units has revealed.

TABLE 1. Clinical Features

Case	Mutism*	NEG[†]	VI[‡]	PERSEV[§]	EXCIT[‖]	BZD[¶]	BFCRS[#]	Pattern**	DSM-5[††]
1[‡‡]	+	−	+	−	−	+	3	Chronic	ASD
2	+	+	+	+	+	n/a	17	Chronic	ASD
3	+	+	−	+	+	+	8	Acute and recurrent	ASD
4	+	+	+	+	+	+	24	Chronic	ASD
5	+	+	+	+	−	+	15	Acute	ASD
6	+	+	+	+	+	+	23	Chronic	ASD
7	+	+	+	+	−	n/a	22	Acute	Bipolar I
8	+	−	+	+	−	+	8	Acute	Schizophrenia

*Relative to the patient's fluent, usual speech, including uncharacteristic pausing when it derails effective communication.

[†]Negativism. Here defined as an apparently motiveless resistance to interference or suggestion, which may be passive or active (doing the opposite) and may be accompanied by either hostility or indifference; included are refusal to eat or drink, refusal to obey conventional propriety for urination or defecation, and *gegenhalten*.

[‡]Volitional instability. Any of (1 or more) of the following: purposeless posturing, *schnauzkrampf*, immobility, ambitendency, *Sperrung*, automatic obedience, and stupor.

[§]Perseverations. Any (1 or more) of the following: echolalia, echopraxia, stereotypies, grimacing, verbigeration, palilalia, staring, and mannerism.

[‖]Excitements. Any (1 or more) of the following: spells of ceaseless, largely purposeless activity; parakinesia; combativeness; nudism; impulsivity; as well as repetitive and indelicate self-injury (nonsuicidal).

[¶]Responsive to benzodiazepine: lorazepam in 3 patients, clonazepam in 2 patients, and diazepam in 1 patient.

[#]Bush-Francis Catatonia Rating Scale[5] and see Supplemental Digital Content 1, at http://links.lww.com/JCP/A251.

**The course of catatonia. Chronic means at least some features presented, as judged through clinical history taking, in childhood or in adolescence and waxing and waning since.

[††]The principal psychiatric disorder present, other than catatonia. [6] In addition, other psychiatric comorbidities were present in some patients.

[‡‡]The 1 patient who did not meet the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* criteria for catatonia but did qualify for the diagnosis on the basis of the Bush-Francis Catatonia Screening Instrument (2 or more of the first 14 items on the Bush-Francis Catatonia Rating Scale are scored at 1 or higher).

BFCRS indicates Bush-Francis Catatonia Rating Scale; *DSM-5, Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition*; n/a, not tested or not documented.

That catatonia commonly supervenes in ASD is not a new observation and is one that has recently been reviewed in depth.[7] However, how do we account for, if the results of this study are confirmed in studies to come, catatonia in outpatients most often complicating ASD and in inpatients most often complicating a mood disorder? Possibly, catatonia complicates a condition of disordered mood rarely (only once in 6 years in this practice), but when it does, it is abrupt, new and dramatic, and thereby influential in the decision to recommend hospitalization, whereas catatonia in ASD is more often a chronic waxing and waning occurrence, as in the case reported here (Table 1).[8,9] Hence, hospitalization perhaps is prompted much less often.

This last comment points to a diagnostic matter of importance. Some have argued that the features of catatonia overlap with those of autism itself[7]; therefore, in persons with autism, to be considered catatonic, they must constitute a distinct change, as was the policy for the diagnosis in this report. Clinicians, once they consider the diagnosis of catatonia, must be performing well because patients with autism whom they choose to treat for comorbid catatonia are uniformly responding[7,10,11] (Table 1; Case Report, Supplemental Digital Content 1 http://links.lww.com/JCP/A251).

Very possibly, if this article and its case report are representative, in this region, a widespread attention deficit in medical and psychiatric diagnostic practice exists for diagnosing ASD in adults, perhaps because the literature on doing so is still small.[12,13] To what extent such diagnostic oversight takes place generally in the United States and elsewhere is yet unknown. However, another, perhaps even more critical, concern about underdiagnosis is that of catatonia. Two excellent books have been published in the past decade or so, in part motivated by the recognition that catatonia is underappreciated.[3,4] Yet, diagnostic failures and delays continue,[9,10,14,15] including my own (Case Report, Supplemental Digital Content 1 http://links.lww.com/JCP/A251), and this is true, although the diagnosis of catatonia is easy, once considered, and once the diagnosis is made, treatment (benzodiazepine, electroconvulsive therapy, or both) is almost always effective (Table 1).[2] An emphatic suggestion is this: put the Bush-Francis Catatonia Rating Scale to clinical use; it is practical, self-explanatory, and confidence enhancing.[5,16] Because its use in the setting of autism has not been specifically validated, alternatives have been suggested.[7] The experience reported herein, however, suggests that, with the caveat that the catatonic features must at their onset be either new or newly and distinctly exacerbated (see previous), it works well.

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As-Needed Use of Cyproheptadine for Treatment of Selective Serotonin Reuptake Inhibitor–Related Female Anorgasmia

To the Editors:

Sexual adverse effects of antidepressant medications (22%–43%) are highest among selective serotonin reuptake inhibitors (SSRIs) as the mainstream treatment for a wide range of anxiety and mood disorders.[1] Despite the favorable side effect profile of SSRIs, their low price, and well-studied effectiveness, sexual adverse effects may prevent many patients from their use or titration to the optimal dose. Different strategies have been used for treatment of sexual adverse effects of SSRIs including switching to or addition of another antidepressant with less sexual adverse effects (bupropion, trazodone, mirtazapine),[2] addition of non–antidepressant agents (sildenafil, yohimbine),[3] or addition of antihistaminic agents (loratadine and cyproheptadine).[4] Cyproheptadine is an H_1 receptor–blocker antihistamine with 5-hydroxytryptamine 2 serotonin–blocking properties, which has been reported to be effective in the treatment of SSRI-related anorgasmia and delayed ejaculation in males.[5–9] These reports have used cyproheptadine at 2 to 16 mg daily or as-needed doses 1 to 2 hours before sex. One concern about regular daily dose is reversal of the antidepressant effects of