Letters to the Editor

Olanzapine-Induced Tardive Dystonia

TO THE EDITOR: Olanzapine is an atypical antipsychotic associated with a low risk of extrapyramidal side effects in schizophrenia (1). It has also been reported to improve the symptoms of tardive dyskinesia (2). However, olanzapine's effects on drug-induced movement disorders in affective illness are less clear. We report on a patient with bipolar disorder who experienced an exacerbation of tardive dyskinesia and developed tardive dystonia while receiving olanzapine.

Ms. A was a 40-year-old African American woman with psychosis. She was diagnosed with schizophrenia and treated with loxapine, 35 mg/day, and her symptoms completely remitted. Her dyskinesia was first noted as bruxism. Ms. A's loxapine dose was decreased to 5 mg/day, yet the bruxism persisted. Then blepharospasm developed, prompting the diagnosis of tardive dyskinesia. Her loxapine treatment was discontinued, and trazodone treatment, 50 mg at bedtime, was initiated. Over the following months, Ms. A experienced increased blepharospasm, lip tremor, difficulty swallowing, and aphonia. Treatment with reserpine, haloperidol, and botulinum toxin was unsuccessful. Eventually, her condition was successfully treated with vitamin E, 1600 IU/day. Ms. A then experienced a manic episode with psychosis and was rediagnosed with bipolar disorder. She received divalproex, 1250 mg at bedtime, and partially responded. Later, olanzapine, 10 mg at bedtime, was added, and her symptoms fully remitted. Seven months later, Ms. A's neck started intermittently turning to the right. Soon thereafter, she was in marked distress, with severe, frequent torticollis. She also displayed severe dysphonia, blepharospasm, and grimacing; moderate lateral and opening jaw movements; and mild upper extremity choreiform movements, lip pouting, back arching, and head bobbing. Her total Abnormal Involuntary Movement Scale (AIMS) score was 15. Clozapine treatment was initiated, and her dose was titrated to 200 mg/day, while her olanzapine treatment was discontinued. At her 4-month follow-up examination, her dystonia had decreased by 50%, and she felt significantly less distressed. Her AIMS total score was 8; she had minimal blepharospasm and grimacing, mild lip and jaw movements, and moderate torticollis.

This case suggests that some patients may develop tardive movement disorders while taking olanzapine. The diagnosis of affective disorder (3) and preexisting tardive dyskinesia may have placed Ms. A at risk of worsening movement disorder with continuing antipsychotic treatment. In addition, signal hyperintensities are common in bipolar disorder and may be associated with extrapyramidal side effects (4), but no magnetic resonance imaging studies were available for Ms. A.

Olanzapine appears to have a higher D_2-receptor occupancy at therapeutic doses than clozapine (5), which may have accounted for Ms. A's worsening tardive dyskinesia and development of tardive dystonia. Until further data are available, careful assessments are warranted for movement disorders in patients with affective psychoses who are taking olanzapine.

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Pain: Cause of Agitation in Elderly Individuals With Dementia

TO THE EDITOR: Agitation is a multifactorial problem that is frequently found in people with dementia. Consequences of this result in institutionalization in nursing homes, hospitals, or other structured living arrangements, which are very costly. For instance, the annual cost of nursing home care in the United States is over $50 billion; by 2030, it is projected to be over $700 billion (1).

Agitation in individuals with dementia occurs for different reasons. Medical causes most commonly include pneumonia, urinary tract infections, or other infectious diseases. Psychiatric causes include delirium, anxiety, depression, and psychosis. Pain is infrequently appreciated as an independent source of agitation in people with dementia. Unfortunately, unnecessary medical evaluations and even treatments may occur when pain is not appreciated as the source of the agitation; this may further aggravate the problem.

When pain is the source of agitation, treatment needs may be quite different. If they are not addressed, further suffering occurs, which further escalates the agitation. The following case illustrates this phenomenon, highlighting the need for early recognition and intervention.

Ms. A, an 86-year-old resident of a nursing home with a 4-year history of dementia of the Alzheimer's type, was admitted to our hospital for further evaluation and treatment of progressive agitation. She had no psychiatric history before the diagnosis of Alzheimer's disease. For the first 3 years after her diagnosis, she remained independent. In the

fourth year, she had to be placed in a nursing home because of her inability to care for herself and the development of anxiety, dysphoria, and perceptual distortions, which resulted in her being placed on a regimen of paroxetine and haloperidol.

Ms. A had a long history of osteoarthritis, osteoporosis, and scoliosis, which affected her walking. Within 6 months of being in a nursing home, she fell and sustained a left intertrochanteric fracture, which required surgery. After surgery, she was only briefly treated with oxycodone. Shortly thereafter, she began screaming and became increasingly agitated, which escalated to aggressive behavior. At this time, she was transferred to our hospital for presumed worsening of her agitated depressive symptoms.

On admission, Ms. A was alert but oriented to person only. Although she was unable to give a detailed medical history, she consistently acknowledged significant discomfort, especially in her back and left leg. Her medications at admission were paroxetine and haloperidol, the doses of which had been recently increased in an attempt to control her agitation.

A medical assessment revealed no acute medical causes for her distress (including infection or cardiovascular or pulmonary changes). Untreated back and left leg pain was felt to be the most likely cause of her agitation; hence, she was treated with scheduled analgesics, including acetaminophen and ibuprofen. Within 2 days, her agitation markedly decreased, and her behavior was more appropriate. She was able to return to the nursing home; continued improvement was noted at her 1-week follow-up evaluation.

In nursing home residents, agitation is very common. Determining whether or not pain is the primary source of agitation is complicated by a cognitively impaired person's difficulty in communicating his or her symptoms (2). The presence of dementia should not be an excuse for not doing a proper evaluation to identify and treat pain in elderly patients.

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Clozapine-Induced Stuttering: Epileptic Brain Activity?

TO THE EDITOR: Thomas et al. (1) reported stuttering in a patient with schizophrenia who was taking clozapine, 400 mg/day. The patient had also developed pharyngeal dystonia, together with buccolingual and facial dyskinesia. The authors ascribed the phenomenon of stuttering to the dystonic syndrome. It was associated with paroxysmal activity in EEG recordings. A second case of stuttering associated with 400-mg/day clozapine treatment was reported by Ebeling et al. (2). We report the case of a 49-year-old woman treated with clozapine who suffered prominent stuttering preceding a generalized epileptic seizure and who recovered after antiepileptic treatment.

Ms. A was first hospitalized at 24 years of age with paranoid ideation and ideas of persecution. Her formal thinking was characterized by a looseness of association. Hallucinations were never documented, and results of her physical examination, laboratory tests, and brain imaging studies were normal. Her family history included mental illness; her mother and a brother had both been hospitalized for paranoid psychoses. Neuroleptic treatment with several typical neuroleptic drugs was associated with acute dystonia (retrocollis and oculogyric crisis), requiring cholinergic medication. Clozapine treatment, 450 mg/day, was initiated. Her EEG recordings showed diffuse slowing but no epileptic activity at this dose. A recurrence of psychotic episodes required several hospital readmissions and temporary increases of her clozapine dose. EEG abnormalities with triphasic sharp waves were first reported at doses of 650 mg/day but remitted when the dose was decreased below 600 mg/day.

Ms. A reported stuttering for the first time when her dose of clozapine was increased to 700 mg/day during a psychotic episode. The stuttering did not completely remit, but the intensity fluctuated. Intermittent sharp waves were documented in her EEG recordings; they disappeared after a dose reduction to 650 mg/day. Ms. A increased her clozapine dose because psychotic symptoms occurred; she ignored the greater intensity of her stuttering. At a clozapine dose of 750 mg/day, she developed a generalized epileptic seizure, followed by myoclonic jerks of her arms, which persisted for approximately 2 hours after her admittance to a neurological intensive care unit. EEG recordings showed generalized polyspike wave activity, so antiepileptic treatment was started with intravenous phenytoin; her dose of clozapine was reduced to 600 mg/day. Her stuttering disappeared along with the addition of the anticonvulsive medication and the normalization of her EEG recordings. Phenytoin treatment was replaced with valproate treatment. After a follow-up examination at 6 months, her stuttering had not reoccurred on a regimen of clozapine, 600 mg/day, and valproate, 900 mg/day.

The pathogenesis of developmental, as well as acquired or neurogenic, stuttering is unclear. Pharmacological treatment strategies were reviewed by Brady (3). Our observation of clozapine-induced stuttering might indicate that this condition is related to epileptic brain activity rather than to a dystonic syndrome, as suggested by Thomas et al. (1). This view is supported by a complete remission of Ms. A's stuttering after the anticonvulsive treatment with phenytoin or valproate and a normalization of her EEG recordings. Whereas Ms. A experienced acute dystonia while taking classic tricyclic neuroleptics, there was no association of dystonic episodes with stuttering while she was taking clozapine. Higher doses of clozapine (greater than 600 mg/day) are associated with seizures (4). In the case of Ms. A, stuttering with clozapine doses greater than 650 mg/day preceded the seizure, which occurred at a dose of 750 mg/day. We suggest that the onset of stuttering in patients treated with clozapine should prompt EEG recordings for brain epileptic activity.

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Sildenafil for Sexual Dysfunction in Women Taking Antidepressants

TO THE EDITOR: Sexual dysfunction is one of the more common and troublesome side effects associated with serotonergic antidepressants, particularly selective serotonin reuptake inhibitors (SSRIs). It frequently results in switching, discontinuation, or dose reductions to ineffective levels of the prescribed antidepressant. Approximately 50% of patients reportedly experience some degree of sexual dysfunction with SSRIs (1); the most common complaints in women include lessened libido, difficulty with lubrication, dyspareunia, and anorgasmia. Control of iatrogenic sexual dysfunction could improve treatment effectiveness by increasing compliance and decreasing relapse or recurrence.

Sildenafil citrate, a peripherally acting selective inhibitor of type V cyclic guanosine-monophosphate-specific phosphodiesterase, is approved as an effective oral treatment for a diverse etiologic spectrum of male erectile dysfunction (2). It acts at the penis on nitric oxide transmission, stimulating cyclic guanosine monophosphate formation by inhibiting its catabolism. Reports of women taking sildenafil describe increased vaginal blood flow, enhanced clitoral responsiveness, and increased vaginal lubrication (3). We report on the use of sildenafil in an open study of 10 female patients who developed symptoms of sexual dysfunction as a consequence of treatment with antidepressants.

Ten patients were recruited from outpatient psychiatry clinical settings at the University of New Mexico and Texas Tech University Schools of Medicine. Clinicians were asked to identify female patients, age 18 to 60 years, who provided oral informed consent, who were in stable relationships, who had normal premorbid sexual function, and who had developed sexual dysfunction, particularly anorgasmia, with or without other sexual disturbances (i.e., loss of libido, lubrication difficulties, uncomfortable or painful intercourse) while being effectively treated with a thymoleptic. The subjects had to be taking the antidepresssant for at least 6 weeks, currently receiving a stable dose and showing improvement of the presenting condition (usually depression, anxiety, or both), and experiencing sexual side effects continuously for more than 4 weeks. Exclusion criteria were concurrent unstable medical illness, poor overall physical health, history of sexual dysfunction (other than antidepressant-induced), psychiatric disorder not under control, previous or current alcohol or substance abuse or dependence, diabetes mellitus, neurological disorder, or genital anatomical defects. A history of stroke, myocardial infarction, or use or likely use of any nitrate caused a subject to be explicitly excluded. Treatment was provided in the context of the subjects' ongoing clinical care and was approved by the hospital's institutional review board.

Each patient was given three 50-mg tablets of sildenafil and instructed to take one 50-mg tablet no more than 2 hours or less than 1 hour before anticipating sexual intercourse. If sildenafil was unsuccessful in reversing the sexual dysfunction, the patient was instructed to take 100 mg (two tablets) in the same time frame before the next anticipated sexual encounter. Nine of the 10 identified patients took sildenafil and reported a complete or very significant reversal of their sexual dysfunction. This included return of effective duration and intensity of adequate arousal, lubrication, and orgasmic function.

This report adds to previous findings of the reversal of male sexual dysfunction with sildenafil (2). These women developed sexual dysfunction as a side effect of prescribed antidepressant treatment. In several cases, the initial antidepressant was changed, but sexual dysfunction recurred with the second agent. Sildenafil was consistently effective in improving these iatrogenic adverse effects. In addition, the patients were able to continue the same thymoleptic regimen that improved their current illness. With the exception of occasional mild transient headache or dizziness, no other significant side effects were observed with brief sildenafil treatment.

Sildenafil seems to be an effective intervention for SSRI- and other thymoleptic-induced sexual dysfunction and offers new approaches over generally ineffective strategies with serotonin antagonists (cyproheptadine), cholinergic agonists (bethanechol, neostigmine), α_2-antagonists (yohimbine), herbal treatments (ginkgo biloba), dose reduction, and drug holiday. Effective management of iatrogenic sexual dysfunction can positively affect treatment outcome by improving compliance and reducing the attendant morbidity and mortality of the disorders for which the agents are prescribed but too commonly discontinued. To be determined is whether sildenafil's reversal of sexual side effects caused by antidepressants continues with long-term use for persistent sexual dysfunction. Further study and replication of these findings in randomized placebo comparison trials are necessary and are in process.

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Attention Deficit Hyperactivity Disorder in the Prison Population

TO THE EDITOR: Longitudinal research suggests that 30% to 50% of children with attention deficit hyperactivity disorder (ADHD) still meet the criteria for this diagnosis in young adulthood (1). Longitudinal research also suggests that children with ADHD are at a greater risk than normal subjects of arrest and imprisonment during early adulthood (2). However, to date and to our knowledge, no study has looked at the rates of ADHD in the adult prison population to assess whether this higher risk translates into a higher prevalence of prisoners with ADHD.

We examined a randomly selected group of patients referred to the psychiatry clinic in Mountjoy Prison for adult male offenders over a 10-month period to see how many met the DSM-IV criteria for ADHD. The psychiatry clinic is well placed to examine the rate of ADHD: although many prisoners are referred with psychopathology, 62% of the prisoners referred during the capture phase were sent to the clinic for personal problems or insomnia. Most prisoners in Mountjoy Prison are in their 20s or 30s. Fifty-five prisoners were assessed. Their mean age was 26.2 years. Five (9.1%) met the DSM-IV criteria for current ADHD.

The prevalence of ADHD among school-age children is estimated at 3% to 5% (DSM-IV). Accepting that 50% of these children have an illness that persists into young adulthood suggests that a randomly selected group of young adults should yield a prevalence rate of approximately 2.5%. This cross-sectional study suggests, therefore, that there is a higher-than-expected prevalence of ADHD among young adult prisoners, corroborating the higher risk of imprisonment found in longitudinal follow-up studies of children with ADHD and suggesting that ADHD is a diagnosis that should be borne in mind in adult forensic psychiatry.

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Neuropsychological Functions in Patients With Schizophrenia

TO THE EDITOR: We read with interest the article by Jill M. Goldstein, Ph.D., et al. (1) about sex differences in neuropsychological functions in patients with schizophrenia, but we think their methodology was seriously flawed. Although the authors argued that their study was specifically designed to examine sex differences, they did not match patients and healthy comparison subjects pairwise with regard to relevant demographic characteristics such as gender, age, and education. In a recent study (2), our group also focused on gender differences in neuropsychological functions in schizophrenia by individually matching male and female patients with schizophrenia to healthy comparison subjects with regard to age, gender, and education. We showed that education has by far the highest impact on neuropsychological functioning, especially on verbal intelligence and language, spatial organization, verbal memory and learning, and abstraction-flexibility as measured by the Wisconsin Card Sorting Test. In the study group investigated by Dr. Goldstein et al., male patients with schizophrenia had significantly less education than male comparison subjects (12.9 versus 14.8 years; $t=-2.6$, $df=28$, $p<0.02$), whereas female patients with schizophrenia did not differ from their female comparison subjects with regard to education. Given this difference as well as the differences in reading test scores, spelling test scores, and estimated IQs—all to the disadvantage of male patients with schizophrenia—the most likely explanation of Dr. Goldstein and colleagues' findings is that the poorer performance of men with schizophrenia is due to differences in education and IQ.

Further comments are related to the data analyses. While the authors tried to control for reading ability, a variable not significantly different between the groups, they did not control for the variables that revealed statistically significant group differences—namely, estimated IQ and spelling test scores. Furthermore, we cannot agree with the conclusions drawn by the authors regarding sex differences based on univariate analyses of variance of the functions. This is because they have not been corrected for multiple testing and especially because the sex-by-group interaction in the multivariate F test across functions is not significant (Wilks's lambda=0.75, F=1.43, df=8, 34).

In summary, we agree with Dr. Goldstein and colleagues' view that matching procedures are essential in comparing neuropsychological performance in subjects with schizophrenia. However, we disagree with the conclusions drawn by the authors, since relevant confounding variables were not adequately controlled in this study.

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Dr. Goldstein and Colleagues Reply

TO THE EDITOR: We appreciate the interest and careful work of Margot Albus, M.D., Ph.D., and Werner Hubmann, M.D., on sex differences in neuropsychological functioning in schizophrenia. We have previously argued, consistent with their thinking, that the size and significance of sex effects in schizophrenia are highly dependent on methodological issues such as sampling, matching, and diagnostic criteria. However, we disagree with Drs. Albus and Hubmann regarding how one's group should be matched to examine sex differences or group differences in neuropsychological or brain abnormalities in schizophrenic and normal subjects. The key issue is separating illness effects from presumed pre-illness characteristics, reflected in whether to match for the patients' IQ or education or both. However, controlling for IQ or education, known to account for substantial variance in other neuropsychological measures, may remove variance directly attributable to the independent variables of interest—i.e., schizophrenia or sex (1–3). This is known as the matching fallacy (3), in which participants are overmatched on a variable that is not independent of the illness per se, such as IQ or education.

Thus, one should match subjects according to socioeconomic or educational status. If, as with schizophrenia, we are studying a neurodevelopmental disorder—i.e., one in which the natural history of the illness has pre- or perinatal origins—then the patients' education and IQ are already affected by the illness. Thus, matching subjects according to patients' education or IQ, pairwise or proportionately by group, would essentially adjust for an illness effect, thus

overmatching—attenuating differences among groups. This may be particularly problematic in studies of sex differences, since we and others have shown sex differences in premorbid histories in schizophrenia—i.e., suggesting early differential illness effects (4). Single-word reading is less affected by the illness than education or IQ (1, 2). Thus, we conducted additional analyses controlling for reading and still found significant sex effects, thus underscoring the validity of the findings in our article.

Finally, regarding our analytic approach, as stated in the article, we did not have the statistical power to adequately test for an overall sex-by-group interaction using a multivariate F test. This does not invalidate our findings, which were reported as effect sizes. The two-to-eightfold effect sizes, in table 3 of our article, clearly showed differences not only within sex (comparing patients and normal subjects) but between sexes (within the group). Furthermore, if our results were solely an education effect rather than a sex effect, we should have reported sex effects for all neuropsychological domains for which Drs. Albus and Hubmann and colleagues report that education had a significant effect (table 4 in their 1997 study). This was not the case.

Although we disagree with the matching procedure used by Drs. Albus and Hubmann, we believe that the topic of sex differences in neuropsychological functioning needs further investigation. It is still unclear which aspects of neuropsychological functions differ in men and women with schizophrenia. Identification of these differences can provide clues to understanding the impact of one's sex on brain abnormalities in schizophrenia.

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Reprints of letters to the Editor are not available.

Correction

In the article "Prediction of Antidepressant Effects of Sleep Deprivation by Metabolic Rates in the Ventral Anterior Cingulate and Medial Prefrontal Cortex" (August 1999, pp. 1149–1158) by Joseph Wu, M.D., et al., the names of two of the authors, James H. Fallon, Ph.D., and David Keator, B.S., were omitted.