represents common clinical practice among local psychiatrists. The rapid dose escalation may have contributed to the observed dystonia. Rapidly raising the dose of an antipsychotic is a known risk for acute dystonia (4). This side effect may be exceedingly rare because we noted no prior case reports of such in an adult.

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Memantine and Catatonic Schizophrenia

TO THE EDITOR: The use of typical and atypical antipsychotics has provided marked improvement for many schizophrenic patients. Numerous patients, however, do not achieve full remission of symptoms, often having recurrent episodes. Several adjunctive therapies have been researched targeting the *N*-methyl-D-aspartic acid (NMDA) receptor (1). To our knowledge, this is the only case showing improvement in a patient with schizophrenia possessing catatonic symptoms in which the use of memantine has shown benefit.

Mr. A, a 56-year-old man with schizophrenia, was admitted after police discovered him wandering the streets responding to auditory hallucinations. His symptoms included mutism, staring, posturing, perseveration, stupor, and stereotypy (retrospectively assessed with the Bush Francis Catatonia Rating Scale, score=16) (2). His hospital course was prolonged because of partial response to olanzapine, haloperidol, ziprasidone, and risperidone. There were several psychotic relapses with polydipsia, staring, mutism, immobility, and somatic delusions. He was transferred several times between the acute care and extended care units.

Memantine, 5 mg/day, was started, ziprasidone was discontinued, and clozapine was initiated. The next day, Mr. A's symptoms improved greatly. He spoke more freely, and a feeling of his head being warm had subsided, as well as his preoccupation with drenching himself in baptismal fashion. His memantine and clozapine doses were titrated to 10 mg b.i.d. and 300 mg/day, respectively. His clozapine level was measured at 509 ng/ml.

Uncertainty as to the efficacy of memantine promoted its discontinuation 9 days after initiation. Mr. A's condition subsequently worsened, with a return of staring and soaking himself. Memantine was restarted, and his symptoms again significantly improved. Clozapine and memantine were continued, and his Bush Francis Catatonia Rating Scale score was 3, with automatic obedience.

Memantine is an NMDA receptor antagonist that is approved for use in moderate to severe Alzheimer's disease. Its potential efficacy in schizophrenia may be due to blockade of hyperglutamatergic excitotoxicity in neurons. It is hypothesized that because of a pathological process in the brain, excess glutamate is produced (3). Excess glutamate causes hyperexcitation of glutamate receptors, allowing calcium channels to stay open for prolonged periods. Excessive calcium influx causes free radical damage to the neuron, eventually progressing to neuronal death.

A case report demonstrated the effectiveness of amantadine, an NMDA receptor antagonist with a structure similar to that of memantine, in a patient with schizoaffective psychosis and a Bush Francis Catatonia Rating Scale score of 31 (4). Based upon the effectiveness shown in this patient, memantine may be a useful adjunctive therapy for schizophrenic patients with catatonic symptoms.

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Aripiprazole and Depression in Schizoaffective Disorder

TO THE EDITOR: Aripiprazole, the newest atypical antipsychotic, is reported to have a novel mechanism of action. It is a partial agonist at both the dopamine D_2 and serotonin 5-HT_{1A} receptor and an antagonist at the 5-HT_{2A} receptor (1). Its novel mechanism of action suggests that this drug may have unique therapeutic effects in some patients (2). We encountered a patient with chronic treatment-resistant depression who responded to a trial of aripiprazole.

Mr. A, a 54-year-old married white man, was diagnosed with schizoaffective disorder, bipolar type. He experienced an onset of manic symptoms and psychosis beginning at age 12. His psychotic symptoms persisted during periods of euthymia. His later depressive episodes became more chronic and severe, with significant suicidal ideation. He had had multiple hospitalizations for depression. His most recent manic episode had also required hospitalization. He had suffered from chronic depression that was unresponsive to numerous antidepressants, including adequate trials with venlafaxine, paroxetine, lamotrigine, and sertraline. He had additionally been treated with a series of atypical antipsychotics, including risperidone, ziprasidone, and quetiapine. When we assumed his care, he was receiving 160 mg/day of ziprasidone, 150 mg/day of sertraline, 300 mg/day of lamotrigine, and 2 mg/day of risperidone. Although his psychotic symptoms were controlled, he was depressed and functioning poorly.

Aripiprazole, 15 mg/day, was initiated and later increased to 30 mg/day. Risperidone and ziprasidone were tapered and discontinued. Mr. A experienced a progres-

sive resolution of his depression. Within 2 weeks of beginning treatment with aripiprazole, he reported improved mood, interest, energy, and concentration. Suicidal and homicidal ideation, hopelessness, and auditory hallucinations abated, and he developed a positive affect. His Global Assessment of Functioning Scale score went from 50 to a present score of 75. His Clinical Global Improvement ratings went from "no change" to "very much improved." Mr. A stated that he was happy and better able to tolerate stress and indicated that his "life [had] improved substantially." Lamotrigine and sertraline were also tapered and discontinued. He was currently taking aripiprazole, 30 mg/day, as monotherapy, with no manic or depressive episodes over the last 12 months.

This is the first case report, to our knowledge, of an antidepressant response to aripiprazole in schizoaffective disorder. Aripiprazole replaced "polypharmacy" in this schizoaffective patient, reducing drug costs, the risk of drug interactions, and potential adverse drug effects (3). Aripiprazole deserves further study for the treatment of depression in schizoaffective disorder and bipolar disorder.

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Benztropine Equivalents for Antimuscarinic Medication

TO THE EDITOR: Michael J. Minzenberg, M.D., et al. (1) are to be congratulated for developing a benztropine equivalent table, as we previously did (2), by reviewing in vitro studies and asking experienced clinicians. However, measuring serum antimuscarinic activity may provide better knowledge (3).

The in vitro literature (1, 2) suggests that haloperidol has negligible antimuscarinic binding activity (10 g of haloperidol equals 1 mg of benztropine) (2). Although our senior pharmacologist also concluded that the clinical antimuscarinic binding activity of haloperidol was negligible (2), the psychiatrists working with Dr. Minzenberg et al. proposed that 13 mg of haloperidol equals 1 mg of benztropine (1). Our recent study (3) also suggested that haloperidol probably has negligible antimuscarinic clinical activity. The mean serum antimuscarinic activity in pmol/ml in 16 patients taking haloperidol was 0.40 (95% confidence interval [CI]=0.30–0.51). This low serum antimuscarinic activity appeared to represent nonspecific antimuscarinic binding (the "noise" of the measuring system) since haloperidol levels (or doses) were not correlated with serum antimuscarinic activity.

Clozapine differs in that its in vitro tables suggest that 1 mg of benztropine equals 15 mg (2) or 8 mg (1) of clozapine; the clinical tables suggest that 1 mg of benztropine equals 375 mg (2) or 85 mg (1) of clozapine. Our recent study agreed that clozapine probably has important antimuscarinic activity (3). The mean serum antimuscarinic activity in 20 patients taking antiparkinsonian medications with mean doses of two benztropine equivalents was approximately 1 pmol/ml (1.05, 95% CI=0.66–1.44) (3). The antimuscarinic activity of 100 mg/day of clozapine in 17 patients was approximately 1 pmol/ml (1.38, 95% CI=0.83–1.93). The mean antimuscarinic activity of 300 mg/day of clozapine in 25 patients was approximately 2 pmol/ml (1.91, 95% CI=1.42–2.40). The mean antimuscarinic activity of 600 mg/day of clozapine in 27 patients was approximately 3 pmol/ml (2.81, 95% CI=2.16–3.46).

To estimate clozapine equivalence, one can approximate that 1 benztropine equivalent equals 0.5 pmol/ml of serum antimuscarinic activity. Using the serum muscarinic activity data of the three clozapine doses, one can estimate that 1 mg of benztropine equals 50 mg of clozapine (obtained from the 100-mg data) to 75 mg of clozapine (obtained from the 300-mg data) to 100 mg of clozapine (obtained from the 600-mg data). Thus, the clozapine equivalent of 1 mg/day of benztropine ranges from 50 to 100 mg/day of clozapine. The misleading fact that clozapine is an agonist for some muscarinic receptors, the M_4, and may cause hypersalivation instead of dry mouth may confound clinicians.

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Cognition, Copyright, and the Classroom

TO THE EDITOR: Many psychiatrists are unaware that the Mini-Mental State Examination (MMSE) is protected by copyright. Its 20 questions were published in 1975 in the *Journal of Psychiatric Research* (1). This journal's copyright notice forbids unauthorized reproduction of the examination. However, clinical experience suggests that unauthorized copies are routinely distributed to trainees and staff. We recently became aware of this while preparing an article for publication.

The notion of granting authors exclusive rights to their work goes back to the 1700s, when the United States and the United Kingdom enacted their first copyright laws, largely covering maps, charts, and books (2). Today, copyright protection is afforded by most countries (3): a Chinese court ordered a Beijing-based school to pay U.S. publishers $1.2 million for copying the Test of English as a Foreign Language and the Graduate Management Admission Test (4).

Much has happened since 1975. The MMSE has become the most widely accepted test of cognitive status, the *Journal*