adjusted maternal dose. At maximum, the infant would ingest 0.43% of the weight-adjusted maternal dose.

Upon receiving the results of levels in the breast milk, the woman began breast-feeding exclusively at 8 weeks after delivery. Follow-up of the infant at 4.5 months indicated that the infant was developing well, and no adverse effects of quetiapine were reported.

Although more studies are required to confirm our findings, the level of infant exposure to quetiapine in breast milk appears to be too small for significant pharmacological effects.

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Neuroacanthocytosis

To the Editor: Neuroacanthocytosis, also known as choreoacanthocytosis, denotes a heterogeneous group of diseases that are characterized by CNS abnormalities in association with blood dyscrasia (1, 2).

Neuroacanthocytosis is a rare movement disorder marked by progressive muscle weakness and atrophy, progressive cognitive loss, chorea, and acanthocytosis (spiked RBCs). Other symptoms may include facial and vocal tics, uncontrolled muscle movement, progressive gait instability, seizures, self-injury of the tongue and lips, and changes in personality (3). It is associated with atrophy and neuronal loss within substructures of the basal ganglia, particularly within the caudate nuclei, the putamen, and the globus pallidus (4, 5).

The disorder may be confirmed by tests demonstrating over 15% of RBCs with acanthocytes or abnormal circulating RBCs that have thorny projections. In addition to RBC acanthocytes, creatine phosphokinase and serum transaminases can be markedly elevated (6).

This disease has been reported in several ethnic groups, but epidemiological data are insufficient to report prevalences. This neurodegenerative disorder is usually inherited as an autosomal recessive trait linked to chromosome 9q21 (7). Symptoms typically become apparent between the ages of 25 to 45 years. Disease progression is poorly understood, and no cure exists. Reported causes of death include the following: emaciation due to progressive weakness, dysphagia, and tracheobronchial aspiration (8).

Ms. A was a 33-year-old woman who was admitted to the general medical hospital for rhabdomyolysis. She had been diagnosed with neuroacanthocytosis 4 years earlier in a university setting. She had continuous, uncontrolled, and rapid involuntary movements; a heart rate of 132 bpm; a WBC count of 13.1/mm^3; and a creatine phosphokinase level of 35673 U/liter. She arrived at the hospital taking 5 mg t.i.d. of diazepam, 0.5 mg t.i.d. of benztropine mesylate, and 0.5 mg of haloperidol, as needed for agitation.

We recommended that she be placed into an intensive care unit and intubated. Molindone hydrochloride, 50 mg t.i.d., was introduced; diazepam, benztropine mesylate, and haloperidol were discontinued. After 5 days of taking propofol and with ventilator support, Ms. A's creatine phosphokinase level had fallen to 911 U/liter. Ms. A was extubated on day 8 when her creatine phosphokinase level was 876 U/liter. On day 9, the molindone hydrochloride was titrated to 100 mg t.i.d. On day 11, divalproex sodium, 250 mg t.i.d., was introduced. Upon discharge on day 14, Ms. A's creatine phosphokinase level was 794 U/liter.

The combination of molindone and divalproex was effective in reducing her extreme involuntary movements. Ms. A was calm, alert, aware, conversant, and oriented to the clinical setting, her age, the month, and the year. She regained some level of independent function in her upper extremities and was able to ambulate on a treadmill for brief periods. Upon discharge, her parents took her to their home.

Treatment for this disorder is symptomatic and supportive. Maintenance of proper nutrition is a challenge. A feeding tube may be needed for some patients as the disorder progresses. Antipsychotic drugs can provide stage-dependent relief from chorea and tics. Benzodiazepines may be used to reduce anxiety and diminish the intensity of movement disorders.

Neuroacanthocytosis is a progressive disease. It is usually fatal, the result of symptoms that contribute to pneumonia, cardiomyopathy, and nutritional deficiencies. Life expectancy following the onset of moderate symptoms is typically 5–10 years. However, the life span may be near normal for patients with no prominent neurological or cardiac complications (9).

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Modafinil Augmentation of Phenelzine for Residual Fatigue in Dysthymia

To the Editor: Monoamine oxidase inhibitors (MAOIs) are sometimes required to treat refractory depressive disorders (1). Although effective, they require careful attention to concomitant medicines and foods to avoid a hypertensive crisis or other severe reactions (2, pp. 2557–2559). Modafinil is an agent used to promote wakefulness in patients suffering from excessive daytime somnolence (2, pp. 1160–1162). Most patients with depression complain of fatigue even after antide-

pressant treatment (3). Modafinil augmentation has been used to enhance antidepressant response (4). We know of no interaction studies that have been performed to evaluate the safety or efficacy of combining modafinil with an MAOI. I report what I believe to be the first published case of the use of modafinil to combat excessive daytime somnolence in a patient successfully treated for dysthymia with phenelzine and lamotrigine.

Ms. A was a 54-year-old Caucasian woman who was seen for the treatment of dysthymia, which had lasted 6 years. She had not improved after psychotherapy with three different therapists and did not respond to treatment with adequate trials of 80 mg/day of fluoxetine, 300 mg/day of extended-release venlafaxine, 600 mg/day of nefazodone, 62.5 mg of mirtazapine at bedtime, and 100 mg/day of clomipramine. She had a partial response to a trial of tranylcypromine, 30 mg b.i.d., and a more significant response with phenelzine, 30 mg t.i.d. Clinical improvement was further enhanced with lamotrigine augmentation at 200 mg b.i.d., although she had no evidence of bipolar symptoms according to her history. Ms. A essentially described remission of depressive symptoms lasting 1 year with this combination but continued to describe fatigue and hypersomnolence, whereby she could sleep all night and part of the day. These complaints did not appear to be brought on by her medication. Neither she nor her husband described signs of a sleep disorder, such as snoring or restlessness. Modafinil was added to her regimen, and Ms. A described rapid clinical improvement in energy and motivation, taking 100 mg/day to the extent that she felt more productive. She described no side effects or any sign of hypertensive reaction and was stable with this combination for at least 6 months.

This report may be the first to describe a safe and effective combination of modafinil and an MAOI. There are some obvious limitations. The patient did not have a sleep study performed, so perhaps modafinil was treating an underlying sleep disorder, although no change in nighttime sleep was described. A placebo control would have been helpful, although years of taking other agents did not elicit this kind of response. Long-term safety cannot be guaranteed. Perhaps a drug interaction had not had time to develop, although usually this kind of adverse reaction can occur after as little as a single dose, and this individual had already taken hundreds of doses. Further study on the safety and usefulness of modafinil augmentation with MAOIs would be helpful. Despite these drawbacks, this case does suggest that some individuals taking MAOIs may be able to derive clinical benefit to manage the persistent fatigue and hypersomnolence that may occur during treatment of depressive disorders by adding modafinil.

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Practice Guidelines and Combining Atypical Antipsychotics

TO THE EDITOR: The recently published APA Practice Guideline for schizophrenia (1) completes a trilogy of guidelines for schizophrenia; the other two are the Texas Medication Algorithm Project (2) and the Expert Consensus Guideline Series (3). We can use these guidelines as suggestions, or we can use these them as strict rules. In Philadelphia, where I work, the administrators of AmeriChoice of Pennsylvania, a managed care company, insist that we use these guidelines as strict rules.

According to AmeriChoice's interpretation of these guidelines, we "shalt not" combine atypical antipsychotics until we first use clozapine. I do not believe that this commandment is rational or therapeutic for a certain group of patients. These are the patients who have a significant but partial response, with minimal or no side effects, to the maximum dose of an atypical antipsychotic. For example, before medication, these patients might have intrusive auditory hallucinations constantly or almost every day. After taking the maximum dose of an atypical antipsychotic, they might have less intrusive auditory hallucinations that occur only 1 or 2 days a week.

These patients usually want and need a fine-tuning of their medication, not a complete overhaul. Substituting clozapine would require laboratory tests every 2 weeks—possibly for the rest of their lives—and would increase the risks of weight gain, lethargy, seizures, and agranulocytosis. Why would these patients want to switch to clozapine if they are already taking a medication that provides significant although partial relief from their symptoms, that requires relatively infrequent laboratory monitoring, and that has few or minimal side effects?

In my clinical experience, adding an atypical antipsychotic (other than clozapine) to the original atypical antipsychotic might reduce or eliminate symptoms for these patients. The additional atypical antipsychotic could be safely and easily withdrawn if it were ineffective or had adverse side effects. An additional atypical antipsychotic would be preferable to an additional typical antipsychotic because of its lower risks for extrapyramidal symptoms and tardive dyskinesia.

This particular clinical situation that I am describing—of patients having a significant but partial response, with minimal or no side effects, to the maximum dose of an atypical antipsychotic—is frequent among chronically psychotic patients. By refusing to pay for an additional atypical antipsychotic before clozapine is tried, AmeriChoice is encouraging us to choose clozapine, which is often inappropriate and impractical in this situation. The potential benefits of clozapine often do not outweigh the labor and the potential risks necessary for its use in this situation. For all practical purposes, AmeriChoice is using these guidelines to deny patients a medication regimen, combining atypical antipsychotics that might reduce or eliminate symptoms and that is often more appropriate and more practical than substituting clozapine in this situation.