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.

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Intranasal Quetiapine Abuse

TO THE EDITOR: We would like to report on the widespread "abuse" of quetiapine among inmates in the Los Angeles County Jail—"the largest mental health institution in the world." Anecdotal reports from clinicians and staff estimate that as many as 30% of the inmates seen in psychiatric services report malingered psychotic symptoms (typically endorsing "hearing voices" or ill-defined "paranoia") in order to specifically obtain quetiapine. A history of substance dependence is common among those engaging in this practice. In addition to oral administration, the drug is also taken intranasally by snorting pulverized tablets. Such abusive self-administration seems to be driven by quetiapine's sedative and anxiolytic effects (to help with sleep or to "calm down") rather than by its antipsychotic properties. Accordingly, the drug has a "street value" (it is sold to other inmates for money) and is sometimes referred to simply as "quell."

Although the prevalence of this behavior beyond this narrow forensic population is unknown, the possibility of such an abuse potential is both curious and clinically pertinent. For example, it suggests that quetiapine is indeed associated with a better subjective response than its conventional antipsychotic counterparts (1). It also appears to give lie to the clinical myth that only psychotic patients will ask for and take antipsychotic medications. In our collective clinical experience, many patients (in particular, those with substance dependence) complain of "hearing voices" in order to procure hospital admission, disability income, or psychotropic medications (2). The "voices" are usually vague, highly suggestive of malingering (3), and occur in the absence of other symptoms (such as clear-cut delusions or thought disorganization) that would warrant a diagnosis of schizophrenia. While antipsychotic medications are not typically recognized as drugs with abuse potential, the use of intranasal quetiapine suggests otherwise and underscores the importance of recognizing malingered psychosis in clinical settings. This phenomenon is reminiscent of the era before the widespread use of atypical antipsychotic compounds, when a select group of patients would inappropriately seek and self-administer not only anticholinergics, such as trihexyphenidyl (4), but also low-potency antipsychotics, such as thioridazine or chlorpromazine. Finally, since the monosymptomatic "voices" endorsed by patients are often assumed to represent psychosis and therefore lead to reflexive prescription of antipsychotic medications, further investigative efforts aimed at distinguishing this clinical presentation from schizophrenia would be useful. If these entities could be reliably disentangled, it would help to reduce the diagnostic heterogeneity of schizophrenia and the unnecessary exposure of patients to the potentially harmful side effects of antipsychotic medications.

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Atomoxetine and Nonresponders to Stimulants

TO THE EDITOR: Atomoxetine has been recently introduced for the management of attention deficit hyperactivity disorder (ADHD) (1), and a vigorous campaign is ongoing to encourage physicians to write prescriptions for this drug. A media blitz is being directed to consumers, encouraging them to seek this medication. Before this expensive norepinephrine enhancer is used as a first-line medication to treat ADHD, its advantages relative to the generically prescribed stimulants need to be established. Ideally, a placebo-controlled blinded study model such as the one previously used by us to study another norepinephrine enhancer, imipramine (2), should be used. Because the costs of administering atomoxetine are about $90 per month and generic stimulants cost, on average, about $25 per month, atomoxetine's role as a first-line therapy should be supported by research.

With this in mind, we evaluated this drug effectiveness in our clinical program by employing measures used routinely to gather data in our program among children who were nonresponders to clinical trials of stimulants.

Seven patients were selected from our clinic (which was previously described [3]). Their average age was 10.5 years, and their IQ was 75.6. Their IQ is deemed average by the New York City Board of Education in its special education program, in which most children have an artificially deflated performance that is most likely consequent to comorbid learning disabilities. All patients were diagnosed with ADHD by using standard DSM-IV criteria. In accordance with the company's recommendations, we used doses of atomoxetine starting with 0.5 mg/kg/day for 3 days and then increased them up to 1.4 mg/kg/day. Parents of the children consented to treatment in accordance with routine hospital procedure.

We measured behavioral changes at baseline (without drug) and at either 1.2 mg/kg/day or when behavioral exacerbation obligated discontinuation by using the 10-item hyperactivity index derived from the Conners Teacher's Rating Scale (4).

In this open-label clinical observation of children taking atomoxetine, no change was seen. Tests performed between