‘Farewell’ to haloperidol?
In response to “Haloperidol clearly is neurotoxic. Should it be banned?” (CURRENT PSYCHIATRY, From the Editor, July 2013, p. 7-8; http://bit.ly/1eMegnr), let me clarify several issues before a consensus is established on whether to discontinue the use of haloperidol.

Remember that since the first use of haloperidol—one of the butyrophenones—more than a half a century ago, practitioners and researchers were aware of its neurotoxicity. Nevertheless, butyrophenones are unique chemicals capable of controlling psychotic symptoms and severe brain dysfunctions, such as extrapyramidal reactions, neuroleptic malignant syndrome, akathisia, tardive dyskinesia, and galactorrhea, among others. Dr. Paul Janssen—founder of the laboratory that first released haloperidol—made a fortune that subsequently prevented him from being awarded the Nobel Prize in Physiology or Medicine.1

A product that was the cornerstone of psychiatric treatment for half a century deserves a better farewell than the one Dr. Nasrallah is offering. Atypical antipsychotics present a number of drawbacks and have dangerous toxicity levels that still need study. I am concerned about metabolic syndrome (diabetes mellitus, hypercholesterolemia, gynecomastia, severe obesity, etc.), which may cost even more to treat than the cost of psychiatric care. In addition to the burden of their high and often unreasonable cost, quetiapine, olanzapine, clozapine, aripiprazole, risperidone, and other atypical antipsychotics have clinical limitations that often restrict their use.

If psychiatry needs a good, immediate fix, it would be in the development and approval of new chemicals that are both better tolerated than the butyrophenones and more affordable than atypical antipsychotics.2

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References

Superior efficacy of atypical antipsychotics
Regarding Dr. Nasrallah’s editorial (July 2013) on the research delineating some of the neurotoxic aspects of first-generation antipsychotics, including haloperidol, he seems to shoot clinical psychiatry in the foot when he describes second-generation agents as having been “much safer for the brain than their older-generation counterparts (although they are not more efficacious).” This closing assertion is not followed by a reference. Indeed, one would anticipate that the newer agents would display greater efficacy given the neurotropic properties of the atypicals described by Dr. Nasrallah in his previous editorial, “Beyond dopamine: The ‘other’ effects of antipsychotics” (CURRENT PSYCHIATRY, June 2013, p. 8-9; http://bit.ly/1A7MzW).

The only real study attempting to clarify this issue has been the Clinical Antipsychotic Trials of Intervention Effectiveness (CATIE) study; patients in that study were chronic and refractory to any intervention. In my practice, I have seen clear and compelling evidence supporting the superiority of atypical antipsychotics—as well as chronicity with multiple relapses and rehospitalizations.

More research into this matter is necessary. In the meantime, we need to be mindful of assertions that might be premature and damaging.

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Dr. Nasrallah responds
I appreciate the comments of Drs. Garza-Trevino and Barris in response to my editorial. Here is my reply to the points they addressed:

The efficacy and neurotoxicity of haloperidol are independent mechanisms. Blocking dopamine receptors controls psychotic symptoms, but neurotoxicity involves triggering apoptosis, increasing free
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