

**Not 'antipsychiatry'**

Dr. Nasrallah misrepresents Drs. Jacques Lacan, Erich Fromm, Theodore Lidz, and others in his December 2011 editorial (“The antipsychiatry movement: Who and why,” From the Editor, CURRENT PSYCHIATRY, December 2011, p. 4-6, 53). They were not antipsychiatry, nor am I.

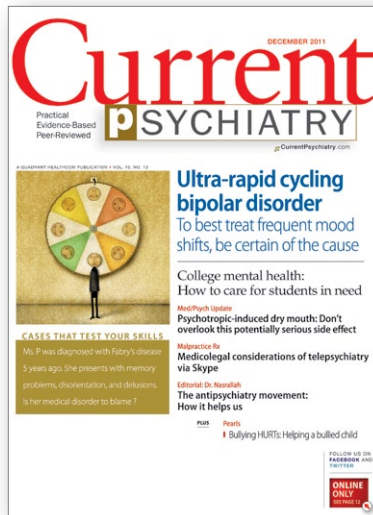
I am “anti” the sort of concrete biological reductionism espoused by Dr. Nasrallah. The psychiatrist nattily dressed in a white jacket and a neat suit and tie looks like a real doctor, but the so-called rigorous objective medical practice he does ignores a huge domain that psychiatry used to be concerned about. In effect, under the leadership of those such as Dr. Nasrallah, psychiatry has “lost its mind.” The disorders we treat are real disorders, but getting rid of the mental dimension and reducing all to “brain disorders” reduces our effectiveness in helping patients, is not humane, and flees from the truth. Psychiatry needs to re-find its mind.

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**Defending Dr. Szasz**

Dr. Nasrallah’s editorial (“The antipsychiatry movement: Who and why,” From the Editor, CURRENT PSYCHIATRY, December 2011, p. 4-6, 53) on the antipsychiatry movement was an excellent historical overview. It also was right on target in its conclusion, namely, that antipsychiatry can help keep psychiatry honest and rigorous.

However, the portrayal of Dr. Thomas Szasz as an antipsychiatrist



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is not wholly correct. For decades, Dr. Szasz has severely criticized both psychiatry and the antipsychiatry movement. Like antipsychiatrists, he is critical of psychiatry, but unlike antipsychiatrists, Dr. Szasz steadfastly has defended the right of consenting individuals to engage in treatment, as long as their participation is voluntary. It is this libertarian streak, the idea that people are free to choose any treatment arrangement they desire—including psychiatric—that distinguishes Dr. Szasz from antipsychiatrists such as Drs. Theodore Lidz, R.D. Laing, and others.

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**Perpetrators of abuse**

Dr. Nasrallah appears to be deliberately avoiding some of the real abuses perpetrated by modern-day psychiatry (“The antipsychiatry movement: Who and why,” From the Editor, CURRENT PSYCHIATRY, December 2011, p. 4-6, 53), including deliberately misdiagnosing

alcoholics and drug abusers as bipolar in order to give them mood stabilizers, often while they are actively abusing substances.

It seems unfathomable for a physician to document a history of depressive episodes, let alone manic episodes, in someone who has been either intoxicated or in early withdrawal constantly over the years, yet this is done routinely. The abuser is happy to play along, as long as the psychiatrist prescribes benzodiazepines along with valproic acid, lithium, or lamotrigine for the patient’s persistent panic attacks and chronic insomnia.

This psychiatric version of “when you have a hammer, everything is a nail” extends to the treatment of uncomplicated grief with antidepressants, additionally labeling oppositional defiant adolescents as bipolar to give them mood stabilizers, and, of course, treating large-portion junk food eaters for “bulimia,” placing them into eating disorder programs, and prescribing psychotropics.

Dr. Nasrallah’s examples of old abuses are a “straw man” argument and unfortunately divert attention from the legitimate concerns about “scientific” modern psychiatry.

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**Dr. Nasrallah responds**

*I appreciate the letters from Drs. Abramson, Rosko, and Cann. I particularly liked their “healthy skepticism” about parts of my editorial about antipsychiatry.*

*My clinical training was heavily “mind-oriented” with intensive psychodynamic as well as behavioral psychotherapy (not cognitive-behavioral therapy), and my*



National Institute of Mental Health research training was heavily “brain oriented” with a neuroscience focus. I integrated both brain (hardware) and mind (software) in my work with each patient and it worked spectacularly well for both of us! George Engel, the father of the biopsychosocial model, was one of my residency supervisors at the University of Rochester, so I was “inoculated” by his mentorship against the hazards of biological reductionism, to which Dr. Abramson assumes I subscribe.

Dr. Thomas Szasz certainly was more of a libertarian than an antipsychiatrist and did a great injustice to patients with severe brain disorders, such as schizophrenia, by asserting that they are competent enough to choose or deny treatment, possibly because of the early state of neurobiology research 50 years ago when the neurotoxic effects of psychosis were still undiscovered. Unlike persons with healthy brains and prefrontal executive functions that enable sound decision-making, schizophrenia patients have anosognosia—the neurologic term for lack of insight and self-monitoring—severe cognitive deficits in processing information and decision-making, and reality distortion, and they lack the capacity to determine that they urgently need treatment. Witness the death of thousands of schizophrenia patients who were abruptly released from Italy’s asylums in the 1980s because they lacked the basic brain functions needed to survive. It was a tragic mistake to leave them to their own devices in the name of freedom, conceptualized by lay legislators who had no idea how impaired the brain is in many schizophrenia patients.

Finally, Dr. Szasz practiced long before research demonstrated that the longer psychosis went untreated, the worse the deterioration and functional outcome. Thus, his stance to let patients with psychosis refuse medications significantly harmed those patients, worsened their symptoms, and reduced their chance for remission.

*Dr. Cann’s allegations of the “real abuses” of modern day psychiatry are to the best of my knowledge just that—allegations. I have never seen valid documentation of the large-scale abuses he cites, although an occasional deviation occurs in any profession. The practice guidelines for various psychiatric disorders never recommend what Dr. Cann claims is happening with diagnostic distortions and ulterior motives.*

*Psychiatry still is evolving as a medical discipline and there are comorbidities that confound the primary diagnosis—such as anxiety or heavy drinking in bipolar disorder—but research is actively seeking a biopsychosocial explanation. The Epidemiological Catchment Area study,<sup>1</sup> published 20 years ago before any of the current medications were introduced, is upheld as the best estimate of the prevalence of psychiatric disorders in the United States—approximately 25% lifetime risk, which means approximately 75 million children, adolescents, and adults have a diagnosable psychiatric disorder. Some of them receive good evidence-based treatments and some do not, but many more never receive any treatment and suffer in quiet desperation.*

**Henry A. Nasrallah, MD**  
Editor-in-Chief

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## Striving for greatness

As an advanced practice nurse in psychiatry who is passionate about my work, I was re-motivated by Dr. Nasrallah’s editorial on the model psychiatrist (“The model psychiatrist: 7 domains of excellence,” From the Editor, *CURRENT PSYCHIATRY*, November 2011, p. 5-6). It was great to see that these traits are being encouraged and practiced. I appreciated the piece, as well as the support that it gives to all types of

mental health practitioners, telling us that there are still psychiatrists striving for “greatness.”

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## Reducing hypersalivation

We would like to counter the suggestion to use benztropine or clonidine as a means to control clozapine-induced hypersalivation as suggested in “Reducing clozapine-induced hypersalivation” (Pearls, *CURRENT PSYCHIATRY*, October 2011, p. 77-78).

As stated in the article, clozapine is an antagonist for all known muscarinic receptors except M4—where it is an agonist—making it a potent anticholinergic medication with the potential to cause excessive saliva production.<sup>1,2</sup> Another proposed mechanism for clozapine-induced hypersalivation is its antagonist activity at  $\alpha$ -1 receptors, thus the suggestion to use clonidine to combat this side effect. The use of benztropine, another medication with known anticholinergic activity, or clonidine, a medication that may cause additional hypotension, to treat clozapine-induced hypersalivation may cause further unwanted complications and may not be the best option to treat this troubling side effect.

We have had great success locally using medications such as atropine or ipratropium as first-line treatments for clozapine-induced hypersalivation in an effort to minimize additional systemic side effects, such as those seen with benztropine and clonidine. Atropine eye drops administered orally, starting with 1 drop and titrated up to 2 drops twice daily to adequate response, has been shown to be successful according to patient opinion in several case reports.<sup>3</sup> Alternatively, intranasal

ipratropium orally administered 1 to 2 sprays, up to 3 times daily has shown improvement in clozapine-induced hypersalivation according to patient report.<sup>3</sup> Although controlled trials to support the use of topical treatment for clozapine-induced hypersalivation are necessary, attempting to minimize additional adverse effects warrants a trial of atropine or ipratropium before using systemically acting medications such as benztropine or clonidine.

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**The authors respond**

*We thank Drs. Hutfilz, Garris, and Kennedy for their response and comments. We acknowledge that the medications we suggested have potentially harmful adverse effects; however, they*

*are evidence-based. Atropine eye drops and ipratropium are interesting suggestions; if their use is based on evidence, they should have been included in the article.*

*We agree that further studies may show these topical approaches to be superior, but our recommendation, based on the best evidence available from the clinical studies, would be to use medications that have been studied while advocating for further study of medications with a theoretical superiority that is not yet proven.*

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