February 2018

The Goldwater Rule and free speech

In his editorial, "The toxic zeitgeist of hyper-partisanship: A psychiatric perspective" (From the Editor, Current Psychiatry, February 2018, p. 17-18), Dr. Nasrallah notes that he "adheres" to the APA's Goldwater Rule. The Goldwater Rule and the reason for its creation and current implementation in the United States cannot be fully understood without appreciating the political circumstances that led to its creation in 1964. The conservative movement had been using the slogan "better dead than red" to criticize Democrats who they felt were soft on communism. Unfortunately, some psychiatrists took these words and the views of Arizona senator Barry Goldwater quite literally. They claimed they understood his psychological structure by listening to his political views, and feared that he would risk starting a nuclear war. Of course, no psychiatrist actually examined senator Goldwater. During the 1964 presidential campaign, a television commercial from President Lyndon B. Johnson's campaign included a mushroom cloud of a nuclear explosion with an implicit reference to senator Goldwater and the "better dead than red" slogan. In the end, psychiatry, and particularly psychoanalysis, as well as President Johnson's campaign, were embarrassed.

One's political views do not inform us of his or her mental health status. This appreciation can be obtained only by a thorough psychological assessment. This is the basis of the Goldwater Rule, coupled with the ethical responsibility not to discuss patients' private communications.

Today, this rule is tested by the behavior and actions of President Donald Trump. Proponents of the Goldwater Rule state that a psychiatrist cannot diagnose someone without performing a face-to-face diagnostic evaluation. This assumes psychiatrists diagnose patients only by interviewing them. However, any psychiatrist who has worked in an emergency room has signed involuntary commitment papers for a patient who refuses to talk to them. This clinical action typically is based on reports of the patient's potential dangerousness from family, friends, or the police.

The diagnostic criteria for some personality disorders are based only on observed or reported behavior. They do not indicate a need for an interview. The diagnosis of a personality disorder cannot be made solely by interviewing an individual without knowledge of his or her behavior. Interviewing Bernie Madoff would not have revealed his sociopathic behavior.

The critical question may not be whether one could ethically make a psychiatric diagnosis of the President (I believe you can), but rather would it indicate or imply that he is dangerous? History informs us that the existence of a psychiatric disorder does not determine a politician's fitness for office or if they are dangerous. Behavioral accounts of President Abraham Lincoln and his self-reports seem to confirm that at times he was depressed, but he clearly served our country with distinction.

Finally, it is not clear whether the Goldwater Rule is legal. It arguably interferes with a psychiatrist's right of free speech without the risk of being accused of unethical behavior. I wonder what would happen if it were tested in court. Does the First Amendment of the U.S. Constitution protect a psychiatrist's right to speak freely?

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The current 'political morass'

Thank you, Dr. Nasrallah, for the wonderful synopsis of the current political morass in your editorial (From the Editor, Current Psychiatry, February 2018, p. 17-18). You followed Descartes' dictum: you thought about matters in a novel fashion. I will assertively share this with others. It is a good piece of teaching.

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The biological etiology of compulsive sexual behavior

Dr. Grant's article, "Compulsive sexual behavior: A nonjudgmental approach" (Evidence-Based Reviews, Current Psychiatry, February 2018, p. 34,38-40, 45-46), puts a well-deserved spotlight on a relatively underrecognized problem that most psychiatrists will encounter at least once during clinical practice. While the article is overall helpful, it completely leaves out any possible biological etiology and underpinnings to the condition that may be important to address while evaluating someone with compulsive sexual behavior. Specifically, are there any endocrine issues that should be considered that may also impact our approach to its treatment?

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The author responds

Dr. Sanghadia highlights the lack of possible biological etiology of compulsive sexual behavior (CSB) in my article. This is a fair comment. The lack of agreed-upon diagnostic criteria, however, has resulted in a vast literature discussing sexual behaviors that may or may not be related to each other, and even suggest that what is currently referred to as CSB may in fact be quite heterogeneous. My article mentions the few neuroimaging and neurocognitive studies that address a more rigorously defined CSB. Other possible etiologies have been suggested for a range of out-of-control sexual behaviors, but have not been studied with a focus on this formal diagnostic category. For example, endocrine issues have been explored to some extent in individuals with paraphilic sexual behaviors (behaviors that appear to many to have no relationship to CSB as discussed in my article), and in those cases of paraphilic sexual behavior, a range of endocrine hormones have been examined—gonadotropin-releasing hormone, follicle-stimulating hormone, luteinizing hormone, testosterone/dihydrotestosterone, and estrogen/progesterone. But these studies have yielded no conclusive outcomes in terms of findings or treatments.

In summary, the biology of CSB lags far behind that of other mental health disorders (and even other psychiatric disorders lack conclusive biological etiologies). Establishing this behavior as a legitimate diagnostic entity with agreed-upon criteria may be the first step in furthering our understanding of its possible biology.

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A different view of patients with schizophrenia

After treating patients with schizophrenia for more than 30 years, I've observed a continuous flood of information about them. This overload has been consistent since my residency back in the 1980s. Theories ranging from the psychoanalytic to the biologic are numerous and valuable additions to our understanding of those who suffer with this malady, yet they provide no summation or overview with which to understand it.

For instance, we know that schizophrenia usually begins in the late teens or early twenties. We know that antidopaminergic medications usually help to varying degrees. Psychosocial interventions may contribute greatly to the ultimate outcome. Substance use invariably makes it worse. Establishing a connection with the patient can often be helpful. Medication compliance is crucial.

It is more or less accepted that there is deterioration of higher brain functions, hypofrontality, as well as so-called dysconnectivity of white matter. There is a genetic vulnerability, and there seems to be an excess of inflammation and changes in mitochondria. Most patients have low functioning, poor compensation, and a lack of social adeptness. However, some patients can recover quite nicely. Although most of us would agree that this is not dementia, we'd also concede that these patients' cognitive functioning is not what it used to be. Electroconvulsive therapy also can sometimes be helpful.

So, how are we to view our patients with schizophrenia in a way that can be illuminating and give us a deeper sense of understanding this quizzical disorder? It has been helpful to me to regard these individuals as a people whose brain function has been usurped by a more primitive organization that is characterized by:

- a reduction in mental development, where patients function in a more childlike way with magical thinking and impaired reality-testing
- atrophy of higher brain structures, leading to hallucinatory experiences
- a hyper-dominergic state
- a usually gradual onset with some evidence of struggle between the old and new brain organizations
- impaired prepulse inhibition that's likely secondary to diffuseness of thought
- eventual demise of higher brain structures with an inability to respond to anti-dopaminergics. (Antipsychotics can push the brain organization closer to the adult structure attained before the onset of the disease, at least initially.)

The list goes on. Thinking about patients with schizophrenia in this way allows me to appreciate what I feel is a more encompassing view of who they are and how they got there. I have some theories about where this more primitive organization may have originated, but whatever its origin, in a small percentage of people it is there, ready to assume control of their thinking just as they are reaching reproductive age. Early intervention and medication compliance may minimize damage.

If a theory helps us gain a greater understanding of our patients, then it's worth considering. This proposition fits much of what we know about schizophrenia. Reading patients' firsthand accounts of the illness helps confirm, in my opinion, this point of view.

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Cognitive impairment in schizophrenia

The authors of "Suspicious, sleepless, and smoking" (Cases That Test Your Skills, Current Psychiatry, September 2017, p. 49-50,52-54) assert that "…the severity of cognitive impairment in schizophrenia has no association with the positive symptoms of schizophrenia" and they add, "Treatment of the cognitive symptoms of schizophrenia with antipsychotics has been largely ineffective." However, in the case they present, Mr. F appears to demonstrate just the opposite: He is given antipsychotics, and over the course of his hospital stay, both his positive symptoms and his cognition improve. His scores on the Montreal Cognitive Assessment increase from 9 (Day 11) to 15 (Day 16) to 21 (Day 24). Thus, in this particular case, treatment with antipsychotics is clearly associated with cognitive improvement.

During the past 15 years, I have routinely measured cognitive functioning in patients with schizophrenia. Some have no impairment, some have severe impairment, and some fall in between these extremes. Most often, impairment occurs in the area of executive function, which can lead to significant disability. Indeed, positive symptoms can clear up completely with treatment, but the deficits in executive functioning can remain.

I think it is fair to say that cognitive impairment is a common, although not nearly universal, feature of schizophrenia that sometimes improves with antipsychotic medication. I look forward to the advent of more clinicians paying attention to the issue of cognition in schizophrenia and, hopefully, better treatments for it.

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The authors respond

We thank Dr. Mahoney for his thoughtful letter and queries into the case of Mr. F.

First, regarding the prevalence of cognitive impairment in schizophrenia, it is our opinion that cognitive impairment is a distinct, core, and nearly universal feature of schizophrenia. This also is the conclusion of many clinicians and researchers based on their significant work in the field; still, just as in our initial case study, we concede that these symptoms are not part of the DSM-5's formal diagnostic criteria.

The core question Dr. Mahoney seems to pose is whether we contradicted ourselves. We assert that cognitive impairment in schizophrenia is not effectively treated with existing medications, and yet we described Mr. F's cognitive improvement after he received risperidone, 2 mg/d, titrated up to 2 mg twice daily. We first pointed out that part of our treatment strategy was to target comorbid depression in this patient; nonetheless, Dr. Mahoney's question remains valid, and we will attempt to answer.

Dr. Mahoney has observed that his patients with schizophrenia variably experience improved cognition, and notes that executive function is a particularly common lingering impairment. On this we wholly agree; this is a helpful point of clarification, and a useful distinction in light of the above question. Improvement in positive and negative symptoms of schizophrenia, as psychosis resolves, is a well-known and studied effect of antipsychotic therapy. As a result, the sensorium becomes more congruent with external reality, and one would expect the patient to display improved orientation. This then might be reasonably expected to produce mental status improvements; however, while some improvement is frequently observed, this is neither consistent nor complete improvement. In the case of Mr. F, we document improvement, but also significant continued impairment. Thus, we maintain that treating the cognitive symptoms of schizophrenia with antipsychotics has been largely ineffective.

We do not see this as a slight distinction or an argument of minutiae. That patients frequently experience some degree of lingering impairment is a salient point. Neurocognitive impairment is a strong contributor to and predictor of disability in schizophrenia, and neurocognitive abilities most strongly predict functional outcomes. From a patient's point of view, these symptoms have real-world consequences. Thus, we believe they should be evaluated and treated as aggressively and consistently as other schizophrenia symptoms.

In our case, we attempted to convey one primary message: Despite the challenges

of treatment, there are viable options that should be pursued in the treatment of schizophrenia-related cognitive impairments.

Nonpharmacologic modalities have shown encouraging results. Cognitive remediation therapy produces durable cognitive improvement—especially when combined with adjunctive therapies, such as small group therapy and vocational rehabilitation, and when comorbid conditions (major depressive disorder in Mr. F's case) are treated.

In summary, we reiterate that cognitive impairments in schizophrenia represent a strong predictor of patient-oriented outcomes; we maintain our assertion regarding their inadequate treatment with existing medications; and we suggest that future trials attempt to find effective alternative strategies. We encourage psychiatric clinicians to approach treatment of this facet of pathology with an open mind, and to utilize alternative multimodal therapies for the benefit of their patients with schizophrenia while waiting for new safe and effective pharmaceutical regimens.

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