



October 2015

## We are not 'psychiatrists'

I found Dr. Nasrallah's editorial regarding the future developments in psychiatry interesting (Do you practice sophisticated psychiatry? 10 Proposed foundations of advanced care, From the Editor, CURRENT PSYCHIATRY, August 2015 p. 12-13). As a young psychiatrist in private practice, I understand why the title "psychiatrist" was initially adopted. I am sure that many of my colleagues agree that the word "psyche" is an abstract, confusing concept: How can we claim to treat something that is not part of known human anatomy?

Nevertheless, we need to clarify the specific nature of our work, namely: the diagnosis and treatment of diseases of the brain, considering other medical causes that can present or exacerbate brain nosology, while providing guidance to modify behavior, thus improving the functional, social, and overall lifestyle of our patients.

We need to change our title to what we really are—encephalopathologists, not psychiatrists!

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## 'The beauty of the asylum'

I appreciate Dr. Nasrallah's metaphor of closing asylums to psychosocial *abruptio placentae* (Needed: A biopsychosocial 'therapeutic placenta' for people with schizophrenia, CURRENT PSYCHIATRY, October 2015 pp. 16,19-20). His proposed components of a therapeutic placenta are supported by evidence-based practice and compassion. I wrote a poem about my feelings about this editorial.

### Asylum

*I inherited an asylum by profession  
where past lives listen  
when I console a grief stricken heart  
watch when medicines are given.  
There are names, dates, and why  
scribbled on walls begging for closures.  
Around me are kindling, plastic  
wasting brains waiting for answers.  
Where are the lives that belong to them?  
Some were sent home alone  
others with loved ones, to foster homes.  
They had twins, farmed corn, caught  
catfish, carved decoys, built roads,  
stargazed away from here.  
I cried, stumbled when they slept  
under bridges, get mugged, homeless  
called from morgues, in jail, sent here.  
Like a pendulum of serenity, despair  
I vacillated from talking to silence  
writing then putting away my  
prescriptions.  
Exhausted I remember past lives  
that chattered once with joy and grief.  
That is the beauty of the asylum  
I inherited this chain of custody  
today, I am one among them.*

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## Challenges with false-positive urine drug screens

Drs. Jeffrey Pawlowski and Vicki L. Ellingrod's article, "Urine drug screens: When might a test be false-positive?" (Savvy Psychopharmacology, CURRENT PSYCHIATRY, October 2015 p. 17, 22-24), not only was of high clinical relevance, but it also hinted at another issue of crucial importance: namely, not prematurely dismissing a patient's reports that he (she) has been abstaining from a drug. It is easy for providers to become jaded and assume that patients, particularly those with a history of substance use, are not being truthful when their self-reported abstinence contradicts laboratory results.

I hope that this article encourages us to become intimately familiar with the specifics of the urine drug screens we employ in practice. We owe it to our patients to do so.

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In the article, "Urine drug screens: When might a test be false-positive?", it was noted that false positives in immunoassays are rare, but that those

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involving opiates and amphetamines were more common than cocaine-metabolite and cannabinoid false positives. In the Table, the authors noted that dextromethorphan, diphenhydramine, fluoroquinolones, poppy seeds and oil, and rifampin can trigger a false-positive result for opiates.

The importance of false-positive opiate screens cannot be overemphasized, in light of the epidemic of opioid use disorder—especially among clinicians working in a treatment program. Some of the challenging aspects about treating patients with opioid use disorder are:

- high prevalence of the disorder
- diversion of existing medication-assisted treatments (ie, buprenorphine), compliance with treatment
- urine drug monitoring.

The article addressed urine drug screening, particularly cross-reactivity of the different drugs. With buprenorphine treatment, cross-reactivity of the

buprenorphine screening assays varies, depending on which assay is being used. In a study comparing the new Lin-Zhi urine buprenorphine enzyme immunoassay (EIA) with the well-known Microgenics cloned enzyme donor immunoassay, investigators concluded that the latter assay generated a higher percentage of opioid cross-reactivity than the former, and that there also was interference from structurally unrelated drugs (ie, chloroquine and hydroxychloroquine).<sup>1</sup> The EIA assay demonstrated more highly specific and sensitive detection of buprenorphine, without opioid cross-reactivity.

In a study<sup>2</sup> that examined cross-reactivity of naloxone with oxycodone immunoassays, researchers proposed that urine samples with a high naloxone concentration produced higher cross-reactivity with oxycodone. They proposed that such high naloxone concentrations could occur in adul-

terated or substituted urine when patients have attempted to dissolve buprenorphine in the urine sample to provide the appearance of compliance. The authors mentioned that typical total urine naloxone concentrations are usually quite low for standard buprenorphine formulations, because of their low bioavailability when taken orally. The clinical recommendation in the article states that it is good practice to confirm positive screens with gas chromatography-mass spectrometry tests.

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**Diagnosing and Managing Depressive Episodes in the DSM-5 Era**

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**DISCUSSION INCLUDES:**

- Applying the mixed features specifier
- Implications of mixed features for illness severity, comorbidities, and treatment response
- Management strategies

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**Diagnosing and Managing Depressive Episodes in the DSM-5 Era**

The premise of the newly introduced mixed features specifier in the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5)* is similar to what was proposed approximately a century ago as part of the "manic depression" unification hypothesis. German psychiatrist Emil Kraepelin (1856-1926) originally conceptualized affective states as a continuum, wherein an individual's diagnosis was arrived at via a confluence of contemporaneous disturbances in mood, thought processes, and volition (behavior). His original description was agnostic insofar as it lacked the 2 categorical constructs, bipolar disorder and major depressive disorder—terms that eventually appeared in the DSM. Kraepelin described a total of 6 types of mixed states (depressive or anxious mania, excited depression, mania with thought poverty, manic stupor, depression with flight of ideas, and inhibited mania) and pure depression. The phenotypic variation of states that Kraepelin described (Figure 1) are similar, but not identical, to the phenotypic heterogeneity of mixed states subsumed under the DSM-5

**specifier**  
is specifier during a major depressive episode whose categorical diagnosis is a depressive disorder. The presence of a major depressive episode in an order historically bridges bipolar disorder and is a tacit endorsement of an "The mixed features specifier in DSM-5 is of mixed states, which was defined as manic and depressive episode." The specifier is applied to an episode of hypomanic depressive features are present, as defined 3 or more proscribed hypomanic reasons and juxtaposes the conceptual DSM-5. As can be seen in the figure, for 8 features, at least 5 core manic symptoms need to be present. For a diagnosis of depression with mixed features, at least 3 core manic symptoms and at least 5 depressive symptoms need to be present.<sup>1</sup> Several core and nonoverlapping symptoms exist in depression with mixed features. Symptoms that are core (ie, allowed) include diminished interest or pleasure, slowed physical and emotional reac-

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