

Letters to the Editor

Scientifically Unfounded Claims in Diagnosing and Treating Patients

TO THE EDITOR: We greatly appreciated the thoughtful book review by Andrew F. Leuchter, M.D. (1), published in the May 2009 issue of the *Journal*, on Daniel Amen's *Healing the Hardware of the Soul: Enhance Your Brain to Improve Your Work, Love, and Spiritual Life* (2). Dr. Amen claims that numerous psychiatric illnesses can be diagnosed and treatments prescribed based on resting single photon emission computerized tomography (SPECT) images. Dr. Leuchter correctly points out the absence of empirical data to support the claims of Dr. Amen. Several years ago, following conversations with Dr. Amen on how to address such concerns, the Brain Imaging Council of the Society of Nuclear Medicine offered Dr. Amen the opportunity to submit his analyses of a blinded set of SPECT scans (to have been prepared by the Brain Imaging Council) to determine how effective his technique is at correctly diagnosing subjects. Although this proposed study could have provided support for his approach, the offer was declined. Nevertheless, for more than two decades, Dr. Amen has persisted in using scientifically unfounded claims to diagnose and treat patients (over 45,000 by his own count).

There are several dangers to patients that can accrue from this approach: 1) patients (including children) are administered a radioactive isotope without sound clinical rationale; 2) patients pursue treatments contingent upon an interpretation of a SPECT image that lacks empirical support; and 3) based on a presumed diagnosis provided by Dr. Amen's clinics, patients are guided toward treatment that may detract them from clinically sound treatments.

Just as serious is the danger to our field. It is likely that, within the next decade, Dr. Amen's claims will be realized in that psychiatrists will enjoy the ability to diagnose and prescribe treatments based, in part, upon neuroimaging findings. Unfortunately, if previously led astray by unsupported claims, patients and their doctors may be less inclined to utilize scientifically proven approaches once these are shown in the peer-reviewed literature to be effective.

It is therefore incumbent upon all of us to monitor and regulate our field. We encourage physicians to remain vigilant of unproven approaches practiced by our peers and to immediately report these trespasses to their state medical boards.

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Psychosis Associated With Medical Marijuana: Risk vs. Benefits of Medicinal Cannabis Use

TO THE EDITOR: Over the past 15 years, it has become increasingly evident that cannabis use carries an increased risk for the development of psychosis (1, 2). At the same time, medicinal cannabis (medical marijuana) has been legalized in many states, with minimal restrictions on prescribing indications. The present case illustrates the evolution of a psychotic disorder, in the setting of medicinal cannabis use, in a young man at high risk for psychosis.

"Mr. Z" was a 24-year-old man who was first hospitalized for insomnia, irritability, and aggressiveness 2 years after military service. On admission, he displayed heightened religiosity and mild suspiciousness. Urine toxicology screening revealed cannabinoids, supporting the patient's endorsed semi-daily cannabis use via water pipe for the past 18 months, without other substance abuse. He was started on quetiapine (100 mg/day), with rapid resolution of symptoms, and discharged after 10 days.

The patient subsequently discontinued quetiapine and was lost to follow-up. Four months later, he presented to a marijuana clinic complaining of chronic pain, insomnia, and anxiety and was given a diagnosis of posttraumatic stress disorder (PTSD) and pain, along with a medical recommendation for cannabis. No psychotic symptoms were elicited. He later explained that he switched from "street" marijuana to medical marijuana in order to obtain a more potent product as well as to avoid illegal activity and getting "ripped off" by drug dealers. He also increased the frequency of his daily use from approximately once to twice daily.

Six months later, Mr. Z was rehospitalized with new-onset auditory hallucinations (multiple voices speaking to each other and urging violence) and delusions (believing that people were tampering with his windows and eavesdropping on his conversations and that he was Jesus Christ). Aripiprazole (15 mg/day) was prescribed, with gradual symptomatic improvement, and then tapered to a lower dose (7.5 mg/day) due to tremor. The patient reported that he believed smoking cannabis helped his chronic pain but that it worsened his psychotic symptoms, such that he wanted help to stop smoking the drug. After 4 weeks, he was discharged to residential substance abuse treatment with only mild, residual psychotic symptoms and a discharge diagnosis of psychotic disorder not otherwise specified, PTSD, and cannabis dependence. At a 3-month follow-up evaluation, while still taking aripiprazole, Mr. Z remained off cannabis and free of psychotic symptoms.

Although cannabis may have some health benefits, it also has a variety of adverse effects, including psychosis, especially among those at high risk (1–3). The patient in the present case was at high risk for psychosis based on attenuated symp-

toms at first presentation, with evolution of frank psychosis potentially explained by his increased use of cannabis and the greater potency of medicinal relative to “street” cannabis (4). This case underscores the importance of 1) aggressively managing cannabis use in patients at high risk for psychosis and those already suffering from psychosis, 2) apprising physicians who prescribe/recommend medicinal cannabis of its iatrogenic and psychotoxic liability among such individuals, 3) educating the public about the risk of cannabis-induced psychosis, and 4) the need for recent evidence about this public health risk to inform policy decisions about medicinal cannabis in the United States (3).

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Do Antidepressants Alter Emotional Processing in PTSD?

TO THE EDITOR: I read with interest the article by Catherine J. Harmer, D. Phil., et al. (1), published in the October 2009 issue of the *Journal*, on the effects of antidepressants on negative affective bias in depressed patients. The authors raised the possibility that antidepressants exert effects by altering emotional processing early in treatment. They also noted that their results are consistent with cognitive theories of depression.

The study's findings remind me of the effects of selective serotonin reuptake inhibitors (SSRIs) on anger, which I have observed in patients with combat-related posttraumatic stress disorder (PTSD). I've noted that treatment with SSRIs often produces a discernible reduction in observed and internally experienced anger preceding any reduction in other PTSD symptoms or depression. Patients report that their “fuse” seems longer and that they see things that used to make them angry but somehow do not bother them as much. This reduced inclination toward anger frequently occurs within a few days of starting treatment and sometimes occurs at lower than usual doses, consistent with the lower dosing of reboxetine conducted by Harmer et al. Sometimes it is the patient's spouse, not the patient, who first notices that the patient

seems less angry. Sometimes the ameliorative effect of SSRIs on anger is reaffirmed with medication discontinuation. I have had spouses correctly suspect that their husband was secretly medication noncompliant based on their perception of his increased anger. One patient, a former Vietnam medic, was able to articulate a change in his perceptions with sertraline discontinuation. Within days, he perceived that people around him were suddenly “lots more angry and difficult.” He realized, of course, that this was unlikely and that it was his appraisal of others that had suddenly changed.

These clinical experiences suggest that SSRIs may alter emotional processing in PTSD patients not unlike that seen with reboxetine in depressed patients. (1) Although there are potential alternative explanations for the aforementioned clinical observations (e.g., improvements in anger in PTSD patients may be one aspect of a general SSRI-induced emotional dampening [2] and improvement in anger might be a manifestation of a global improvement in PTSD), the timing of the improvements (i.e., early in treatment) and the reports of altered perception of external events are reminiscent of Harmer et al.'s findings. It may be that changes in emotional processing by antidepressants play a role in the treatment of PTSD just as they appear to do in depression.

References

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Drs. Harmer, Goodwin, and Cowen Reply

TO THE EDITOR: We thank Dr. Hierholzer for his interest in our hypothesis that antidepressant drug treatments have early effects on the evaluation of emotional material, which are important in the development of clinical mood change over time (1). We agree that this hypothesis of antidepressant drug action may also extend to anxiety disorders. In his clinical observations, he suggests that anger is reduced early on with SSRI treatment in PTSD. These clinical observations are consistent with an earlier study (2), which found a decrease in anger recognition following 7 days of administration of the SSRI citalopram in healthy volunteers. It is encouraging that these findings in healthy people in a laboratory setting may translate into a different patient group and to a real-world setting. Consistent with these findings, Davidson et al. (3) reported that early effects on anger and irritability were predictive of therapeutic response to sertraline in individuals with PTSD.

To test Dr. Hierholzer's clinical observations using a cognitive psychology approach, it will be important to observe whether behavioral and neural biases toward anger-related