

## Orthomolecular Treatment Cannot Overcome the Tranquilizer Psychosis

In this issue we have a wonderful educational article about the orthomolecular treatment of schizophrenia and other psychotic disorders by Dr. John Hoffer, the son of the late Dr. Abram Hoffer. I strongly encourage you to read and re-read John's article since it describes the basics and nuances of good orthomolecular psychiatric care, while also emphasizing important practical guidelines for clinicians seeking (who want to know how best to use) the right combination of micronutrients, in the right amounts, for patients struggling with psychosis.

John's article illuminates the simplicity of the orthomolecular approach and cogently articulates the reasons why it should not become diluted by extraneous laboratory evaluations or too many micronutrient interventions. In many ways the complexity of orthomolecular psychiatry rests in its simplicity, and yet so many clinicians at the present time are practicing it in a manner that unfortunately diminishes its value and effectiveness. Just like his father, John reminds us that good orthomolecular psychiatric care begins with the development of an effective clinician-patient relationship built on a solid foundation of trust, education, patience, commitment, encouragement, evidence-informed orthomolecular treatments, and detailed inquiries about patients' signs and symptoms, their social relationships with family and friends, their employment status (which may also include steady volunteer work or being an effective homemaker), and their ability to pay income tax.

I would also like to remind our readership about an important aspect of orthomolecular psychiatric care that Abram fully discussed in two papers.<sup>1,2</sup> Unfortunately, the concepts discussed in these papers continue to be neglected by many clinicians to the detriment of their patients, and can lead them to conclude that the orthomolecular treatment failed to produce significant ben-

efits. It should be known that when a patient is taking an antipsychotic drug, the drug will lessen symptoms of psychosis but will also induce a medication-induced condition that Abram referred to as the "tranquilizer psychosis." Abram stated the following about the iatrogenic effects of antipsychotics when describing the tranquilizer psychosis:

"Tranquilizers do initiate the recovery process in schizophrenic patients and this produces the illusion that they will eventually lead to recovery. However, as the recovery process continues, that person's biochemistry becomes more normal and then begins to respond to the drug as if they were normal, i.e. they become sick. Tranquilizers make normal people (including schizophrenics) sick."<sup>1</sup>

When explaining the tranquilizer psychosis, or essentially why the antipsychotic drugs negatively impact patients, Abram characterized this condition as having the following clinical features: (1) less intense psychotic signs and symptoms; (2) memory and concentration problems that have either worsened or remained as they were at diagnosis; (3) depression that would be no different than at diagnosis; (4) less anxiety and agitation; (5) greater apathy and disinterest; (6) socially controlled behavior; and (7) physical toxicity including tardive dyskinesia, nausea, brain damage (cerebral cortex atrophy), weight gain, diabetes, and impotence.<sup>1,2</sup>

Abram also described how antipsychotic drugs and perhaps especially the atypical antipsychotic drugs cause a dependency state accompanied by the following: psychiatric relapse following rapid withdrawal; persistent or unsuccessful efforts to cut down or control substance use; stoppage or severe curtailment in social, occupational, and recreational activities; and continued use despite evidence of unremitting and recurrent physical harm or psychological problems that are probably related to or worsened by the drugs themselves.<sup>2</sup>

Abram argued that without orthomolecular treatment, many patients on atypical antipsychotic drugs have little chance of improving, normalizing, or being cured of

their psychosis. Patients need to be on the orthomolecular treatment for a minimum of two months (often more in my experience, usually four to six months, for patients to understand the patience and commitment needed when on the orthomolecular approach), and then the atypical antipsychotic drug should be very slowly decreased or withdrawn based on the patient's clinical status.<sup>1,2</sup> It is so vital that patients are afforded the opportunity to see how they do with a gradual decrease in atypical antipsychotic; otherwise, Abram has noted that:

"...if the tranquilizer drugs are not withdrawn as the patients begin to recover on orthomolecular therapy there will be no response or no apparent response. There may have been a response of the original schizophrenic state but this will be masked by the tranquilizer psychosis...It is vital that the amount of drugs be reduced as recovery begins, for only then will the investigator see the real effect of the treatment, and only then will patients and their families observe the real recovery which has occurred...If the drugs are not withdrawn the tranquilizer psychosis will develop and this will not be prevented or ameliorated by the nutritional therapy. Vitamin B<sub>3</sub> does not cure the tranquilizer psychosis."<sup>1</sup>

The point here is that all clinicians should follow the advice of John Hoffer when implementing orthomolecular psychiatric care with patients. Clinicians should likewise heed Abram Hoffer's advice and work with patients and their prescribing psychiatrists or physicians so that attempts are made at minimizing patients' dependence on atypical antipsychotic drugs to ascertain the full benefits of the orthomolecular treatment. If attempts at minimizing (and hopefully discontinuing) atypical antipsychotics are never made, clinicians and their patients (as well as the patients' family members) will simply assert or discredit the effectiveness of orthomolecular treatment.

In an attempt to improve patients' understanding of niacin (nicotinic acid) treatment for schizophrenia or psychosis, I have included an informational sheet that can be

copied without any copyright infringement for the benefit of patients prescribed niacin, which can be found on the next page.

—Jonathan E. Prousky, ND, MSc  
Editor



## References

1. Hoffer A: Vitamin B-3 does not cure the tranquilizer psychosis. *Townsend Lett Doctors Patients*, 2001; 213: 88-91.
2. Hoffer A: Editorial: atypical anti-psychotics create dependency disorders. *J Orthomol Med*, 2004; 3-10.

## Patient Handout

### Using Niacin (Nicotinic Acid) for the Treatment of Schizophrenia or Symptoms of Psychosis

Please purchase 500 mg pills of niacin. Day one, take one pill (500 mg) with each meal three times daily. Day two, increase to three pills (1,500 mg) twice daily with meals. Within about 10-15 minutes of your first dose of niacin you will experience a flush. The flush is not dangerous and is beneficial in reducing muscle tension, anxiety, general stress, and possibly other symptoms. The flush sensation usually begins on and around the forehead and then travels down the body to the bellybutton and sometimes to the toes. It is accompanied by itching, redness, heat, and then sometimes is followed by cold chills when it subsides. The flush usually lasts for several minutes to over 20-minutes and then subsides. Remember, the flush is not an allergic reaction but an expected reaction from niacin.

Niacin can sometimes lower blood pressure, so when you are changing positions (i.e., going from sitting to standing, please do so slowly to give your body time to adjust). Once you increase your niacin dose to 1,500 mg twice daily with meals, the flush should either go away, or should become about 90-95% less intense and much more tolerable.

Niacin has been used for the treatment of schizophrenia since the 1950s. Niacin has efficacy in treating acute schizophrenia or early psychosis (i.e., receiving treatment within one to two years of diagnosis), and among patients that have chronic schizophrenia or chronic symptoms of psychosis, the results of niacin are much less predictable. Niacin is considered off-label since most patients are treated with antipsychotic medication (whether the older drugs, or newer atypical ones), as well as an assortment of other psychiatric drugs, such as mood stabilizers, antianxiety, and antidepressant drugs. You need to be on niacin treatment for a minimum of two months, and usually four to six months, before beneficial effects will be possible. The only way

in which to determine if niacin (along with other properly prescribed orthomolecular treatments) will benefit you is to (with the cooperation of your prescribing physician) slowly reduce the dose of the drug(s) you are taking. With a slow decrease in drug(s), you and your family members should notice improvements in some combination of symptoms impacting quality of life, perceptual and thought disturbances, cognitive function, mood, behavior, and your willingness or desire to do things. At some point, your clinician might feel that your dose of niacin needs to be increased, perhaps to 6,000 mg daily for increased therapeutic effects.

Although niacin can sometimes elevate liver enzymes, the elevations are usually not worrisome. The elevations only become a clinical concern when the liver enzymes reach more than two or three times the upper limit of normal. Consistent clinical vigilance is required. Reassuringly, liver biopsies among patients with cholesterol problems showed that up to 6,000 mg or 6 grams daily of niacin for one to nine years showed no significant pathological change.<sup>1</sup> Niacin can sometimes affect blood sugar control (in about one-third of patients)<sup>2</sup> and has been associated with gout, even though gouty attacks are very rare from taking niacin.<sup>3</sup> The safety profile of niacin is generally very good, and you need to be consistent with this treatment to ascertain if you respond well to this vitamin.

#### References

1. Baggenstoss AH, Christensen NA, Berge KG, et al: Fine structural changes in the liver in hypercholesteremic patients receiving long-term nicotinic acid therapy. *Mayo Clin Proc*, 1967; 42: 385-399.
2. Hoffer A: Vitamin B<sub>3</sub> and schizophrenia. *Townsend Lett Doctors Patients*, 2001;213:20-23.
3. Paterson ET: Vitamin B<sub>3</sub> and liver toxicity. *Townsend Lett Doctors Patients*, 2001; 207: 23.

*Permission to copy this page is granted.*