Letters to the Editor

Worsening of Obsessive-Compulsive Symptoms Following Treatment With Olanzapine

TO THE EDITOR: We report a case of olanzepine-induced worsening of obsessive-compulsive symptoms in a patient who had a similar response to both clozapine and risperidone in previous trials. Although the current literature on atypical antipsychotic drug induction of obsessive-compulsive symptoms consists of 16 case studies, one retrospective report, and a small prospective investigation (1, 2), to our knowledge this is the first such report involving olanzapine. We undertook the current trial with this patient following the report by Baker et al. (3), who suggested that olanzapine did not cause de novo obsessive-compulsive symptoms in inpatients with schizophrenia following a 6-week trial.

Mr. A was a 35-year-old man who lived with his parents and suffered from partially responsive schizophrenia and comorbid obsessive-compulsive disorder (OCD). He had previously experienced clozapine- and then risperidone-induced worsening of obsessive-compulsive symptoms. Before treatment with olanzapine and at 4-week intervals, obsessive and compulsive symptoms were identified and rated with the Yale-Brown Obsessive Compulsive Scale (scale range=0–40). In the past 5 years Mr. A's best level of functioning was attained on a regimen of haloperidol, 25 mg/day, plus some combination of antiobsessional drug (Yale-Brown scale score=20). The most recent antiobsessional drug was fluvoxamine, 200 mg/day. On this regimen, before the olanzapine trial, Mr. A's symptoms included compulsive hand washing and his sentinel somatic delusion of "muscles breaking up."

Olanzapine was started at a dose of 10 mg/day concurrent with Mr. A's other medications, and over a 2½-week period haloperidol was tapered and discontinued. Between weeks 3 and 4 of olanzapine treatment (1 week after discontinuation of haloperidol), accompanied by his mother, Mr. A presented with gross worsening of his OCD symptoms (Yale-Brown scale score=23). His mother stated, "I can't keep him supplied with all of the paper products!" referring to Mr. A's increased hand washing and drying and preference for disposable paper towels and toilet tissue. At week 4 fluvoxamine was increased to 300 mg/day, which was the maximum tolerated. The increase in fluvoxamine, however, did not alter the frequency, duration, or quality of Mr. A's OCD symptoms, and the dose was returned to 200 mg/day at week 10.

Despite the more severe OCD symptoms (Yale-Brown scale score=30 at weeks 12 and 16), Mr. A had a global subjective report of improvement and stated, "I feel more like myself . . . more awake and energetic." He expressed interest in continuing the olanzapine trial, and he suggested "behavioral" treatments for his OCD. Adjunctive behavioral treatment had been suggested or attempted for many years without benefit, primarily because of Mr. A's unwillingness to accept this form of treatment. Although his parents were frustrated with the return of previously controlled OCD symptoms, they also agreed with and supported continuation of olanzapine treatment. Mr. A reported increased motivation, and after months of indecision he decided to join a health club (week 11) and entertained the idea of part-time work or school. His psychotic symptoms, including the somatic delusion, remained at a stable baseline.

This case is one of several to suggest that atypical antipsychotic drugs as a group may share the potential of uncovering or increasing obsessive-compulsive symptoms in patients with schizophrenia or with schizophrenia and comorbid OCD. We undertook the present trial on the basis of a controlled clinical investigation suggesting that olanzapine, despite its pharmacologic resemblance to clozapine and risperidone (i.e., 5-HT_2 antagonism), did not show an association with the emergence of obsessive or compulsive symptoms in patients with schizophrenia (3). All patients in this trial had either no or slight obsessions or compulsions at baseline (i.e., before olanzapine treatment). Therefore, there may be a biological predisposition to developing atypical antipsychotic drug-induced obsessions or compulsions, and this may be prominent in schizophrenic patients with comorbid OCD or in schizophrenic patients with a strong obsessional component. Finally, this report highlights the significance (to the patient) of unrecognized negative or dysphoric symptoms related to use of typical antipsychotic drugs. Although Mr. A did not complain of classic extrapyramidal symptoms or akathisia or have obvious parkinsonian symptoms, he had a fairly robust subjective feeling of improvement and general well-being following discontinuation of haloperidol.

REFERENCES

1. Baker RW, Bermanzohn PC, Wirshing DA, Chengappa KNR: Obsessions, compulsions, clozapine, and risperidone. CNS Spectrums 1997; 2:26-36
2. Dodt JE, Byerly MJ, Cuadros C, Christensen RC: Treatment of risperidone-induced obsessive-compulsive symptoms with sertraline (letter). Am J Psychiatry 1997; 154:582
3. Baker RW, Ames D, Umbricht DSG, Chengappa KNR, Schooler NR: Obsessive-compulsive symptoms in schizophrenia: a comparison of olanzapine and placebo. Psychopharmacol Bull 1996; 32:89-93

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Risperidone-Associated Agranulocytosis

TO THE EDITOR: We report a case in which a patient developed agranulocytosis after risperidone was administered. She previously had developed agranulocytosis after treatment with traditional neuroleptics.

Ms. A was a 40-year-old woman who had met DSM-IV criteria for chronic schizophrenia, paranoid type, from the age of 22. She had had no episodes of neutropenia or leukopenia, and her WBC count was generally around 5,000/mm^3. At admission, her WBC count was 5,600 mm^3, and the neutrophil rate was 57%. She was initially treated with chlorpromazine, 300 mg/day, and carbamazepine, 800 mg/day (at therapeutic blood levels), for 6 weeks. Her WBC count gradually fell to 2,500/mm^3 and the neutrophil rate to 30%, and treatment was immediately stopped. Her WBC count rose after 2 weeks (5,200/mm^3, neutrophil rate=51.7%). Haloperidol (30 mg/day) and lithium (900 mg/day) were then given for 3 weeks. Her WBC count fell again (2,200/mm^3, neutrophil rate=52%). Haloperidol was suspended, but lithium was continued for another 5 weeks, along with diazepam (30 mg/day) and trihexyphenidyl (5 mg/day) because of extrapyramidal syndrome, until her WBC count normalized (4,500/mm^3, neutrophil rate=53%). Because of Ms. A's psychotic state, treatment with zuclopentixol was started and reached 50 mg/day in the second week; however, zuclopentixol was suspended because of severe extrapyramidal syndrome and a fall in WBC count (2,700/mm^3, neutrophil rate=29%). Ms. A developed agitated catatonia, and one course of ECT was administered (three times a week for 2 months), with partial response. Three weeks after zuclopentixol was discontinued and during the course of ECT, her WBC count normalized (4,500/mm^3, neutrophil rate=62%). Bone marrow analysis was performed but did not reveal any pathology. Because of her resistant psychotic state and the presence of extrapyramidal syndrome, risperidone treatment was started 2 weeks afterward and was gradually increased to 4 mg/day in the second week. At that time a blood sample was drawn, and risperidone was discontinued because of the emergence of agranulocytosis (WBC count=2,400/mm^3, neutrophil rate=32%).

This single report is of heuristic value only. We should emphasize that the patient was sensitive not just to risperidone but to antipsychotics in general; thus, despite normal bone marrow analysis, we guess there was some reduction in granulocyte precursor cells. To our knowledge, agranulocytosis has not been reported when risperidone was prescribed alone (1) and has been described only after the addition of risperidone to clozapine treatment (2). Some reports suggest that risperidone is the preferred alternative when adverse hematological effects have been triggered by classic antipsychotics (3).

We urge caution when risperidone is used with patients who have a history of neuroleptic-associated agranulocytosis.

REFERENCES

1. Addington D: Risperidone: integrating research and practice. Br J Hosp Med 1994; 52:223–224
2. Godleski LS, Sernyak MJ: Agranulocytosis after addition of risperidone to clozapine treatment (letter). Am J Psychiatry 1996; 153:735–736
3. Mahmood T, Silverstone T, Spittle B: Risperidone appears to be safe in patients with antipsychotic-induced blood dyscrasias. Int Clin Psychopharmacol 1996; 11:53–54

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New Mnemonic Tool for DSM-IV Diagnosis

TO THE EDITOR: I wish to propose a mnemonic tool that can assist clinicians in the rapid and systematic recall of the 17 different categories of psychiatric diagnosis in DSM-IV. As a firm adherent of both comprehensive differential diagnostic reasoning and biopsychosocial case formulation, I have found this tool useful for teaching students and residents, and especially for preparing candidates for Board examinations.

The 17 diagnostic categories can be easily recalled by imagining that one is examining—say, a sad patient—not only by means of one's psychodynamic acumen and empathic resonance, but also by deploying one's "DSM-IV-scope." Let us call this patient either Samuel or Samantha. Thus, a mnemonic phrase that captures the (current) universe of descriptive diagnostic possibilities is "DSM-F-SCOPE: SAM IS SAD," with each letter representing a major diagnostic category, as follows:

D—Delirium, dementia, and amnestic and other cognitive disorders
S—Substance-related disorders
M—Mood disorders

F—Factitious disorders

S—Schizophrenia and other psychotic disorders
C—Childhood, infancy, and adolescent disorders
O—Other disorders that may be a focus of clinical attention
P—Personality disorders
E—Eating disorders

S—Somatoform disorders
A—Anxiety disorders
M—Mental disorders due to general medical condition

I—Impulse control disorders not elsewhere classified
S—Sleep disorders

S—Sexual and gender identity disorders
A—Adjustment disorders
D—Dissociative disorders

I hope that others find this to be a user-friendly tool that enhances the likelihood of comprehensive diagnostic assessment.

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Critique of Book Review

TO THE EDITOR: I am writing to protest the selection of Dr. Paul C. Horton as reviewer for my book *Ultimate Intimacy: The Psychodynamics of Jewish Mysticism* (1). The book, which was edited by me and which contains contributions by a number of distinguished psychoanalytic colleagues and some equally distinguished scholars of Jewish mysticism, attempts, by example, to initiate the serious psychologic analysis of the phenomena of Jewish mysticism by using primarily a psychodynamic approach. Dr. Horton, on the other hand, is concerned not with understanding the psychology of the mys-

tical experience so much as its celebration. "Nowhere in the book," he says, "do we find resonances with the 'unbound wonder and profound awe . . . and the feeling of unutterable happiness which is never forgotten' that is said to characterize the 'true mystical experience.' " He continues, "Curiously, traditional psychoanalysts, such as the contributors to this volume . . . [set] rules for discourse about mysticism that would keep us in a religious conceptual playpen. Although the analyst may play the role of 'apocalyptic seer' . . . there are no Maggids in psychoanalytic institutes." Apparently, Dr. Horton interprets any psychoanalytic interest in the phenomena of mysticism as an attempt to restrict and thwart the practice of mysticism. Why would he—or anyone—look for maggidim in psychoanalytic institutes?

His resentment toward our endeavor causes him to misrepresent and misquote the text. "Jewish mysticism," he tells us, "took its origin in the ninth through the thirteenth centuries." Actually, as the text makes clear (p. 40), Jewish mystical documents appeared within one or two centuries before or one or two centuries after the Common Era.

Further, he writes, "The 'ultimate intimacy' spoken of in the title is reduced to an unconscious repetition of children's 'intense interest in their parents' toilet functions' (p. 205)." Let me give you the context from which that quotation is taken. On page 204 we find the statement, "Dr. Galenson informs us that in their earliest years, children exhibit intense interest in their parents' toilet functions." The issue is the interpretation of a text from The Ethiopic Book of Enoch, which describes "streams of flaming fire" issuing from beneath a throne upon which the "Great Glory" was sitting. The text is considered in a demonstration of how historical-philological and psychoanalytic interpretation can be coordinated. A similar image occurs in Daniel 7:9–10. The argument assembles several pieces of evidence that suggest that the stream issuing from under the throne occupied by the divinity is a symbol for a urinary stream. The evidence is strongly suggestive and not at all far fetched. The chapter concludes with the statement that if that conclusion is correct, it "leads us right to the matter of our central concern - ultimate intimacy." The rest of the book is concerned with interpreting the mystical experience as a regressive attempt once more to enjoy the comfort of physical closeness to and intimacy with the parents as one might imagine they were experienced in early infancy. It is one thing to contend that the image of the fiery streams leads us again to the issue of ultimate intimacy on the one hand, and taking it as the definition of ultimate intimacy on the other.

Horton takes issue with the author for reverting to archaic psychoanalytic paradigms. Nowhere does he indicate that the chief psychoanalytic parameter considered is affect regulation.

Dr. Horton concludes his "review" by congratulating the contributors "for their willingness to explore what Emerson has called 'the region of grandeur which reduces all material magnificence to toys, yet opens to every wretch that has reason the doors of the universe.' "

It is quite clear that Dr. Horton is not interested in what our book is trying to do and condemns it because it is not a different book.

Yet he ignores the final sentence of the book, which might have been expected to please him. "Many of us who cannot accept the non-rational assumptions necessary to embrace mysticism might nevertheless find a certain degree of comforting spiritual experience in studying what it is, what it does, how it has influenced the lives of millions of people of every religion over the centuries, and where it might take us in the future."

Dr. Horton should not have been invited to review this particular book, and, having been invited, in fairness, he should not have accepted.

I respectfully request that the book be assigned to a more objective referee for a proper review.

REFERENCE

1. Horton PC: Book review, M Ostow (ed): Ultimate Intimacy: The Psychodynamics of Jewish Mysticism. Am J Psychiatry 1996; 153:1499–1500

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Dr. Horton Replies

TO THE EDITOR: Mortimer Ostow is incensed that I have reviewed his book on mysticism. He challenges my qualifications, evidently preferring that someone in his immediate circle should have been assigned the task. However, it is rather bootless to preach to the converted, and scholarly discourse should welcome a diversity of perspectives. As a steady contributor to the literature on the mystical experience and related emotional feeling states (1–5), I find it appropriate that I would review a book on this subject for the broad spectrum of readers of *The American Journal of Psychiatry*.

The capacity for the mystical experience has important evolutionary and psychodynamic implications. Most readers of this journal are going to want to know if a book about this subject is practical, relevant, and sufficiently comprehensive to be helpful in the practice of psychiatry. From my review, the reader will have no misunderstanding of what this book is about. Very few readers will feel a need to know about the more parochial and esoteric aspects of this subject. A basic fault with Ostow's book is expressed, paradoxically enough, in the sentence he cites, the last sentence of his book. The sentence begins, "Many of us who cannot accept the non-rational assumptions necessary to embrace mysticism" and ends with a little sop for the benighted ones, the "millions" who, through the centuries, have, by implication, abandoned rationality in their search for progressive solace. Such a perspective, i.e., that mysticism is "non-rational," precludes alternative psychoanalytic explanations. Thus, the idea that mysticism is an example of experience in the progressive transitional mode (1–3) is not even considered by Ostow and his colleagues.

Ostow essentially reiterates orthodox Freudian reductionism: Hate is older than love; the solace of religion is always and only mere illusion; and the positive emotional feelings are simply defensive add-ons in the face of a "harsh and intransigent reality" (80). This obsolete perspective ignores the universality of the mystical phenomenon and reduces it to an exclusionary, paranoid, grandiose, and destructive "apocalyptic complex."

Ostow faults me for not highlighting "affect regulation" as the "chief psychoanalytic parameter." However, the "affect regulation" of the orthodox psychoanalytic paradigm, like Ostow's "apocalyptic complex," is about regulating intensely negative and primitive affects. For Ostow, one can embrace, if he thinks they can be embraced at all, higher, more creative, and evolutionarily modern feeling states only by making "non-rational" assumptions.

Indeed, one wonders if Ostow and his colleagues are talking about mysticism at all. A sign of this is his focus on the "practice" of mysticism. One does not practice mysticism any more

than one practices moments of musical genius, scientific inspiration, or, in the case of the apocalyptic complex, madness.

Ostow asks why I—or anyone—would look for maggidim in psychoanalytic institutes. My comment was in response to his statement, "The analyst plays the role of the apocalyptic seer who provides the revelation" (p. 138). One of the unexamined assumptions of such a posture is that the analyst can preempt the role of the maggid. The second is that the analyst, as surrogate maggid, can "practice" the delivery of revelations. This would be like a conservatory student practicing being Beethoven. Yet, the analyst might more appropriately look to the maggid for creative illumination for those whose backs are to the mouth of the cave.

Regarding Ostow's statement that I "misrepresent and misquote the text" about the time of the origin of Jewish mysticism, I found Ostow's discussion on pages 39–40 to be too ambiguous as a frame of reference for the reader. I therefore turned to noted historian Frederick Copleston (6), who dates the *Cabala* from "after the middle of the ninth century A.D." to the "beginning of the thirteenth century" (p. 201). Other historians date the roots of the *Cabala* a little earlier, at the beginning of the Middle Ages, but this may be of marginal significance for other than the professional historian.

REFERENCES

1. Horton PC: The mystical experience—substance of an illusion. J Am Psychoanal Assoc 1974; 22:364–380
2. Horton PC: The mystical experience as a suicide preventive. Am J Psychiatry 1973; 130:294–296
3. Horton PC: Solace—The Missing Dimension in Psychiatry. Chicago, University of Chicago Press, 1981
4. Horton PC, Sharp SL: Language, solace, and transitional relatedness. Psychoanal Study Child 1984; 39:167–194
5. Horton PC: Introduction, in The Solace Paradigm—An Eclectic Search for Psychological Immunity. Edited by Horton PC, Gewirtz H, Kreutter KJ. Madison, Conn, International Universities Press, 1988, pp 3–39
6. Copleston F: A History of Philosophy, Volume II: Mediaeval Philosophy, Augustine to Scotus. Westminster, Md, Newman Press, 1960

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Reprints of letters to the Editor are not available.

Correction

On the seventh line from the bottom in the second full paragraph on page 321 of the editorial "Decline of Dopamine: Effects of Age and Acute Neuroleptic Challenge" by Jeffrey A. Lieberman, M.D., and Richard Mailman, Ph.D. (March 1998, pp. 319–323), the phrase in parentheses should read "(i.e., had increased dopamine overflow)."