Letters to the Editor

Paroxetine for Treatment of Obsessive-Compulsive Disorder and Comorbid Stuttering

TO THE EDITOR: Antidepressants have been reported to be beneficial in the treatment of stuttering (1). Stuttering has also been conceptualized as a form of obsessive-compulsive behavior (2). A review of the literature reveals no studies of the effectiveness of selective serotonin reuptake inhibitors (SSRIs) in the treatment of stuttering. However, two reports have described stuttering as a side effect of SSRI treatment (3, 4). Here we describe the successful treatment with paroxetine of a patient with obsessive-compulsive disorder and comorbid stuttering.

Ms. A was a white, 27-year-old single woman with a history of prominent stuttering since adolescence. When she had entered psychiatric treatment 3 years earlier, she had been complaining of anxiety, intrusive thoughts, and poor social functioning. At that time she was treated with psychotherapy and a brief trial of trazodone, which resulted in some decrease in symptoms. At the time of her current presentation she had remained in psychotherapy but had discontinued treatment with trazodone approximately 6 months earlier. She complained of distress related to intrusive thoughts, which were violent, abusive in content (e.g., sexual and physical harm to children), persistent, and difficult to suppress. Ms. A also described a fear of inadvertently verbalizing her inappropriate thoughts at her workplace and subsequently losing her job. Psychological evaluation and psychiatric consultation resulted in a diagnosis of obsessive-compulsive disorder and stuttering.

Treatment with paroxetine, 20 mg/day, was initiated, and weekly psychotherapy continued. After 1 month of paroxetine treatment, Ms. A spontaneously noted a marked decrease in her stuttering. Her therapist also observed the decrease in stuttering during their weekly sessions. Ms. A also experienced a decrease in the frequency and intensity of her intrusive thoughts. After 3 months, the paroxetine dose was increased to 30 mg/day in an attempt to further reduce residual obsessive-compulsive disorder symptoms. One year later, while still taking 30 mg/day, Ms. A displayed marked improvement: the frequency and persistence of her stuttering and intrusive thoughts had decreased, and her social functioning had substantially improved.

It appears that paroxetine may be an effective treatment for patients with obsessive-compulsive disorder and comorbid stuttering. This finding suggests that the repetitious or obsessional behavior associated with some forms of stuttering may be ameliorated when the overriding obsessive-compulsive disorder is properly treated. Further studies are needed to explore this hypothesis and the therapeutic value of SSRIs in the treatment of comorbid obsessive-compulsive disorder and stuttering.

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Hallucinogen-Induced Relief of Obsessions and Compulsions

TO THE EDITOR: The following case report describes the rapid and sustained relief of obsessive-compulsive disorder symptoms with the use of psychedelic drugs.

Mr. A was a white, 34-year-old single man who was incapacitated by obsessive-compulsive disorder. His symptoms, which began at age 6, included excessive preoccupation with the cleanliness of his clothing and self, contamination fears, and preoccupations with order. These led to compulsive counting, showering, ritualistic and repeated washing of his clothes and hands, and ritualistic cleaning and arranging. When showering, he needed to lather 17 times and proceed in a specific order. Failure to do so resulted in an inability to carry on his daily activities.

Mr. A began to use alcohol and marijuana at age 12; large amounts would relieve his anxiety but not his obsessive-compulsive symptoms. When he was 19, he snorted about one-third of a gram of cocaine twice every other week for 4 months. Cocaine markedly worsened his obsessive-compulsive symptoms immediately after use. He first ingested freeze-dried psilocybin mushrooms at age 20, and by age 21 he was ingesting 2 grams of psilocybin twice weekly. He would experience dizziness, nausea, and occasional vomiting about 30 minutes after ingestion of the drug and psychedelic phenomena for the next 6–8 hours. Mr. A readily recognized substantial improvements in his obsessions and compulsions during psilocybin intoxication. His contamination fears disappeared, and he stopped the washing and counting rituals. Such improvement would last 4–5 days but would be followed by gradual symptom return. Further psilocybin use would result in immediate remission of the obsessive-compulsive disorder symptoms.

Mr. A had ingested peyote cactus on five different occasions, which resulted in improvements in his obsessive-compulsive symptoms that were very similar to those improvements seen with psilocybin. However, the psychedelic and adverse effects were more potent and longer lasting.

Mr. A ingested psilocybin on a daily basis for the next 4 years. He had developed a tolerance to the psychedelic effects, but he continued to note improvements in his obsessions and compulsions after psilocybin ingestion. Mr. A stopped taking psilocybin and had minimal interference

from obsessive-compulsive symptoms for the following 2 years. However, over the next few years the symptoms gradually returned to the levels they were before Mr. A had started taking psilocybin.

This case is remarkably similar to two prior cases reported in the literature (1, 2). In such cases, specific improvement for symptoms of obsessive-compulsive disorder or body dysmorphic disorder began acutely during the period of intoxication. Mescaline and psilocybin are potent agonists at serotonin (5-$HT)_{2A}$ and 5-HT_{2C} receptors, and their binding potency to these receptors is correlated with their human potency as hallucinogens (3). The acute improvement in symptoms described in this case may involve effects in 5-HT_{2A} and 5-HT_{2C} receptors. The observations that administration of the nonselective 5-HT antagonists metergoline and ritanserin exacerbates obsessive-compulsive disorder symptoms further support this view. These reports support the need for prospective controlled studies with potent 5-HT_2 agonists such as mescaline in the treatment of obsessive-compulsive disorder.

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Nefazodone and Visual Side Effects

TO THE EDITOR: The following is a brief case report of "visual trails," a side effect of nefazodone treatment seen in a patient with depression.

Ms. A was a 54-year-old woman who was being given nefazodone for treatment of a 6-month episode of depression. She had been experiencing recurrent episodes of depression with generalized anxiety since she was 23 years old. Her nefazodone regimen was 200 mg t.i.d. (three separate doses were given to avoid mild nausea). The dose had been titered over 3 weeks (100 mg/day for the first week, 200 mg/day for the second week). After 2 weeks of nefazodone treatment, while still taking only 200 mg/day, Ms. A reported that she began to experience visual trails at a frequency of about twice a week, mostly upon awakening, before her morning dose, and occasionally after her evening dose. The morning trails last about half an hour until she was fully awake. Over 6 weeks the visual trails decreased in frequency from twice weekly to once weekly or less. She said that the visual trails never bothered her and that she thought of them as "ghost shadows."

This patient had an excellent response to nefazodone: a decrease in insomnia and increases in appetite, energy, and ability to concentrate and work as well as improved mood. It should be noted that she was not taking other medications. On the nefazodone package insert, visual trails are listed under the heading of "Abnormal Vision" as a side effect that had occurred during the clinical trials.

KENNETH SCHWARTZ, M.D.
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Inpatient Treatment of Combat Veterans

TO THE EDITOR: I was very pleased to read the study by David Read Johnson, Ph.D., and colleagues (1). Their conclusions that 1) intensive inpatient treatment exacerbates symptoms and 2) shorter inpatient stays that attempt to stabilize and support veterans may be more beneficial and less costly are both welcome news to those of us who are veterans and involved in the treatment of combat veterans.

There is certainly a need for more studies of this kind. Unfortunately, studies that seem to indicate the cessation of a well intentioned government program are rarely undertaken. This comes primarily because we continue to confuse social compassion with adequate care. These inpatient treatment programs arose out of a genuine recognition that this nation had exacerbated the trauma of Vietnam combat veterans by not providing them sufficient debriefing upon their return to civilian life. These social and clinical failures are well documented in the massive literature on the Vietnam veteran. Apparently and unfortunately, one of the consequences of these well-intentioned inpatient programs has been the exacerbation of symptoms that the programs were designed to correct. If I understand Johnson et al.'s table correctly, 18 months after discharge these veterans were feeling much less hopeful than they did upon entering these programs.

This leaves the fundamental question of what can be done? Perhaps one thing that we ought to remember is that these veterans are now in their late 40s to early 60s. They no longer have the psychological resiliency of the 19-year-old self that was initially wounded. These veterans may also have an additional 25–35 years of social failure and drug abuse overlaying whatever the initial psychological wound is. These very real handicaps must be taken into account before we rush off madly to create one more government program, however well intentioned.

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Dr. Johnson and Colleagues Reply

TO THE EDITOR: We appreciate Donald Denton's comments on our study. Certainly the Department of Veterans Affairs (VA) was well intentioned in its funding of the specialized inpatient units for treatment of posttraumatic stress disorder (PTSD). These programs represent one of the largest efforts a government has ever taken to respond, even though belatedly, to the psychological needs of its veterans. As leaders of the clinical team of one of these units, we, too, had committed ourselves to this effort and were filled with hope for its success. We were disappointed with the results of our own study as

well as with other studies, which suggested that these programs had little long-term effect. The many veterans whose lives did substantially improve continue to cushion us from the profound impact of these results.

Yet our scientific vantage point encourages us to rely on the results of empirical inquiry, even if they suggest our hopes were unfulfilled. Thus, on the basis of a number of empirical studies, including ours, many VA programs are considering shifting their resources from specialized inpatient programs to briefer inpatient, day treatment, and rehabilitation-oriented outpatient programs. Fortunately, these, too, will be subjected to ongoing empirical evaluation.

Nevertheless, these disappointing results should not be used as justification for reducing efforts to help veterans with PTSD, for the results are as much evidence of the strength of this disorder as they are of the inadequacy of the treatment model. These studies should instill respect for the seriousness of this disorder and spur us on to search for more effective treatments. The prolonged and debilitating effects of psychological trauma on its victims and their loved ones are indeed impressive. Although we agree that compassion should not be confused with adequate care, we believe that the former will always remain a prerequisite to the latter.

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Comorbidity Between Personality and Dysthymic Disorders: Historical and Conceptual Issues

TO THE EDITOR: In their very interesting article, Lawrence P. Riso, Ph.D., and colleagues (1) proved that there is a close relationship and frequent comorbidity between early-onset dysthymia and cluster B personality disorders both in patients and their families. Borderline personality disorder was by far the most common cluster B personality disorder, followed by histrionic personality disorder. No such data were presented for family members. We think that these results are of great importance and should make us rethink current concepts of both dysthymic disorder and cluster B personality disorders. A historical viewpoint can show that some of these questions are anything but new.

While Kraepelin (2) understood cyclothymia as a constitution, out of which manic-depressive insanity could develop, and while he postulated a continuum between both disorders, he did not differentiate between unipolar and bipolar disorders. Thus, his concept of cyclothymia consisted of cases that we nowadays would diagnose as dysthymic. Schneider (3) strongly opposed Kretschmer's idea of a cyclothymic constitution or temperament as a continuum from normal to psychosis (4). Schneider saw no connection between personality and psychosis. Nevertheless, his concept of psychopathic personality is of great interest still today. Atheoretically, "psychopaths" are seen as having special forms of abnormal personalities, which are merely variants of the norm. One form of such psychopathy is depressive psychopathy. These patients can be either fearful and anancastic or hyperthymic, irritable, and emotionally instable. This comorbidity between depressive psychopathy (broadly similar to the current concept of dysthymic disorder) and emotional and dramatic personality characteristics (as cluster B personality disorders are defined) was already known to Schneider and has now been confirmed through the use of high methodological standards by Riso et al. Nevertheless, we see a danger: the definitions of psychopathy or personality disorders are by no means self-evident but rather are a mere matter of convention. This means that their diagnostic thresholds are deliberate and derive from clinical necessities. Therefore, a dimensional assessment of personality and personality disorder would add valuable information to the categorical approach. This assessment, especially in cases in which family studies and subclinical forms are concerned, would allow for the testing of the concept of an affective spectrum (5). We believe that the results of the study by Riso et al. 1) stress the importance of dimensional assessment of personality and personality disorders and 2) raise once again serious questions about the relation and connection between "subaffective" illness and personality disorders, and whether it is appropriate that they are listed on two different axes in DSM-IV and in two different chapters in ICD-10.

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Drs. Riso and Klein Reply

TO THE EDITOR: We appreciate the comments by Drs. Brieger and Marneros, which add an important historical backdrop to our findings and raise important methodological and nosological issues. We agree that some rethinking of the concepts of dysthymia and cluster B personality disorders may be warranted. Rather than viewing the co-occurrence of these disorders as the result of one disorder "driving" the other, our family data suggest they co-occur because they *share* etiological factors. Comorbidity in this sense differs somewhat from viewing comorbid conditions as etiologically distinct. However, the rethinking of dysthymia should also be qualified. Our findings suggest that the overlapping factors were only *partially* overlapping. Thus, there may be unique as well as shared etiological factors between these disorders. We anxiously await additional work and discussion on this issue.

The suggestion that our findings extend to the relationship between depressive personality and unstable traits is intriguing. Indeed, Schneider suggested that a subgroup of depressive psychopaths are related to the explosive and labile psychopaths. Of course, our findings are only applicable to this relationship to the extent that dysthymia is equivalent to depressive personality. As Drs. Brieger and Marneros mention, Schneider's writings are probably not consistent with this view, since depressive psychopathy represented the extreme end of normal personality traits rather than being related to major affective disorders. Indeed, differences in the clinical presentations of dysthymia and depressive personality do ex-

ist. Diagnostic overlap is not complete, and depressive personality appears to be a milder and less symptomatic condition (1). Nonetheless, the relative equivalence of these two conditions is implied by evidence that suggests that both dysthymia and depressive personality (as it is currently operationalized) reside along an affective spectrum (1, 2).

While our results support a model of shared etiological factors between dysthymia and cluster B personality disorders, we do not believe they support a spectrum relationship between the two. When disorders lie along a spectrum, they can be thought of as "alternative expressions" or "different phases" of the same disorder (3). Thus, the etiological factors for spectrum conditions should be largely overlapping. In contrast, our family data suggested that the overlap in etiological factors between dysthymia and cluster B personality disorders was far from complete, which is inconsistent with a spectrum relationship.

We agree with Drs. Brieger and Marneros that a dimensional approach would be superior in a variety of ways, including providing greater sensitivity to detect spectrum relationships. Thus, a dimensional approach to analyzing family data has perhaps been underused.

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Academic Versus Clinical Views on Lithium Use

TO THE EDITOR: I would like to comment about the article by Richard E. Johnson, Ph.D., and Bentson H. McFarland, M.D., Ph.D., regarding lithium use in our clinic (1). This article points out some of the limitations and pitfalls that have, unfortunately, existed between academic and clinical settings and some of the lessons that can be learned through close collaboration.

In the 12 years that I have been practicing as a clinic physician at Kaiser Permanente Northwest Region (the last 2 years as Chief of the Department of Mental Health), I have never seen the authors of this article, even though they are but one geographic block away from my clinic. The authors evidently used computer-generated data from a 5% sample, as well as other computer-accessible information. This methodology certainly has some utility but is only a small part of the picture. It seems obvious to the 20 practicing physicians in our department that had Drs. McFarland and Johnson taken the time to walk across the street they would have been made aware of delivery system details that would have allowed them to reach more valid conclusions and hypotheses.

The collection of data not only epitomizes the most unfortunate part of the traditional split between sequestered academicians and their clinically oriented colleagues but does the entire profession a disservice, since the data presented are only the partial truth and, therefore, create a powerful distortion. It may be sensational and the current vogue to make statements that "HMOs might do their care better" and then, as the authors did, list several items that might help. In fact, we currently do most of those things that were recommended in the article, but these things would not show up on the database.

For example, health education classes and nurse case management, which we provide, are not charged against the mental health benefit. Therefore, they do not show up as mental health visits in the computer-driven database. Similarly, many of the patients in our practice lose their coverage because of job changes and often purchase their prescriptions at community pharmacies. Therefore, these data would also be lost, since only the HMO pharmacy data were used. Also, one briefly mentioned hypothesis was not pursued: perhaps these patients were switched to other legitimate mood stabilizers. Our physicians believe that a large percentage of patients started on lithium regimens are switched to valproate treatment, which also would not have been reflected on the computer database. Had Johnson and McFarland asked our clinic physicians who treat these patients about all these data, they would have received helpful, intelligent, and collaborative comments from which they could have developed a database that more closely reflected the clinical reality.

Our access for care is excellent; emergent care is provided and accessible 24 hours a day. Any patient that has an urgent need to be seen is seen within 24 hours, and those who require or request routine visits are seen within 2 weeks. Our customer satisfaction ratings for the care our clinicians provide are very high. Our hospitalization rates match the best regional practices. Our Health Plan Employer Data and Information Set scores are high. The authors even noted that the amount of lithium and laboratory monitoring were within APA guidelines. We have innovative funding of projects related to reminding patients to come in for laboratory tests. We have group psychoeducation classes for patients and families, and measurements from before and after these classes have shown a demonstrated improvement in functioning and skills.

Yes, there is always room for improvement, but I invite these authors and all researchers to come down to the clinics and find out what is really happening, instead of imagining and hypothesizing blindly. We are treating these patients within a limited budget and producing excellent results. Please help us to know how to truly improve the care that we actually provide, instead of supporting the current bias against managed care.

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Androgens and Alcohol

TO THE EDITOR: I appreciated the article on androgens by David R. Rubinow, M.D., and Peter J. Schmidt, M.D. (1). However, I noticed that the authors did not review the animal and human data that described possible androgen effects on alcohol metabolism and on alcoholism.

Castration in animal models has been reported to increase liver alcohol dehydrogenase activity (2), and humans who underwent orchiectomy have demonstrated a postsurgical enhancement of alcohol elimination (3). Mardh et al. (4) ob-

served that testosterone inhibited the gamma-subunit-containing isoenzymes of class I human alcohol dehydrogenase.

Since specific alcohol dehydrogenase polymorphisms have been associated with risk for alcoholism (5), it is possible that testosterone-related effects on alcohol dehydrogenase may also affect alcoholism risk. Lakoza and Barkov (6) suggested a link between testosterone and alcoholism by demonstrating that testosterone administration stimulated the development of experimental alcoholism in rats.

Cloninger et al. (7) distinguished a type 1 (milieu-limited) form of alcoholism and a "male-limited" type 2 form with associated family history of alcoholism, early onset, and criminality. The differentiation of a "male-limited" subtype suggests a possible androgenic influence on the development or expression of alcoholism.

Rubinow and Schmidt reviewed many interactions between androgens and neuroregulatory agents (1). Ethanol also has multiple neuroregulatory interactions, including enhancing the actions of γ-aminobutyric acid, inhibiting the release of acetylcholine, acting at the NMDA receptor, and inducing changes in serotonin turnover and dopamine release as well as interactions with opiate peptides and vasopressin (8). It is intriguing to speculate which, if any, testosterone interactions ultimately affect ethanol use in humans.

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EVE J. WISEMAN, M.D.
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Anorexia Nervosa in the Eighteenth Century

TO THE EDITOR: Stuart C. Yudofsky, M.D., states that anorexia nervosa, which Hilde Bruch epitomized as "the relentless pursuit of thinness," was identified as a medical condition at the end of the nineteenth century (1). In fact, Erasmus Darwin described it in 1796 in his *Zoonomia* under the heading of Anorexia: "Some young ladies I have observed to fall into this general debility, so as but just to be able to walk about; which I have sometimes ascribed to their voluntary fasting, when they believed themselves too plump; and who have thus lost both their health and beauty by too great abstinence, which could never be restored."

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A.G. GORDON
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Dr. Yudofsky Replies

TO THE EDITOR: A.G. Gordon is correct that Erasmus Darwin offered an acceptable, although manifestly deficient and prognostically inaccurate, description of anorexia nervosa at the close of the eighteenth century. A far better account can be found over 100 years earlier when English physician Richard Morton, in his *Phthisiologia, seu Exercitationes de Phthisis*, proffered what he considered to be the chief characteristics of the condition: amenorrhea, extreme emaciation, hypothermia, overactivity, and (incorrectly) lack of appetite. However, a more inclusive and accurate description of the condition that, importantly, highlighted the interaction of the patient with the family awaited Lasègue's definitive late-nineteenth century paper, *De l'anorexie hysterique*. As translated from the French by Palazzoli (1), Lasègue's observations merit recounting.

> After a few months, the patient finally arrives at a state that can rightly be called one of hysterical anorexia. The family is in a turmoil. Persuasion and threats only produce greater obstinacy. The patient's mental horizon and interests keep shrinking, and hypochondriacal ideas or delusions often intervene. The physician has lost his authority; medicaments have no effects . . . . The patients claim that they have never felt better; they complain of nothing, do not realize that they are ill and have no wish to be cured. This description would be incomplete without reference to their home life. Both the patient and her family form a tightly knit whole, and we obtain a false picture of the disease if we limit our observations to the patients alone.

Lasègue's treatise also emphasized the value of the physician being sensitive to his own feelings about and attitudes toward the patient. This insight, coupled with Lasègue's understanding of the role of the patient's family in the pathogenesis of the disorder, the persistence of the symptoms, and success of the treatment laid the foundation for the biopsychosocial conceptualization of the etiologies and treatments of anorexia nervosa as advanced by Hilde Bruch and other twentieth century clinical investigators.

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