Letters to the Editor

Gabapentin as a Potential Treatment for Anxiety Disorders

TO THE EDITOR: Gabapentin is currently indicated as an adjunctive therapy for the treatment of partial seizures with and without secondary generalization (1). Although gabapentin is initially synthesized as a γ-aminobutyric acid analogue, its mechanism of action is unclear. In vitro studies demonstrate a gabapentin binding site in neocortical and hippocampal areas of unclear functional significance (2). An emerging body of experience suggests the potential utility of gabapentin for a number of psychiatric disorders including intermittent explosive disorder (3), mania (4), and pain syndromes (5).

Preclinical data suggest the potential anxiolytic effect of gabapentin (6). Recently, Beauclair et al. (7) reported reduction in anxiety symptoms and syndromes in 18 patients with primary psychotic disorders and in one patient with generalized anxiety disorder treated adjunctively with gabapentin, 200–1800 mg/day. We report here on four patients with primary anxiety disorders refractory to standard anxiolytic interventions; they experienced marked clinical improvement with gabapentin therapy.

Mr. A was a 40-year-old attorney with a 20-year history of panic disorder and a past history of alcohol dependence. He had therapeutic trials of sertraline, paroxetine, and desipramine of adequate dose and duration without significant improvement in panic frequency. Taking no medication at evaluation, Mr. A was having daily panic attacks (one or two per day) with limited phobic avoidance. Treatment was initiated with gabapentin, 100 mg/day, and titrated up over 2 weeks to 300 mg t.i.d. During the first week, Mr. A began to experience a gradual reduction in panic frequency, anticipatory anxiety, and sleep disturbance. By week 5, he was panic free with a reduction in phobic avoidance. This benefit persisted to 4-month follow-up.

Ms. B was a 36-year-old woman with a history of panic disorder with limited phobic avoidance since adolescence; in addition, she had a history of recurrent major depression, past alcohol abuse, and mixed character pathology distinguished by marked irritability. She had trials with numerous antidepressants, mood stabilizers, and benzodiazepines, which were either intolerable or modestly successful. A trial with valproate, up to 1000 mg/day, led to notable improvement in panic attacks and decrease in irritability, but the valproate was discontinued because of marked weight gain. Gabapentin, 100 mg t.i.d., was added to her current regimen of fluoxetine, 60 mg/day, and alprazolam, 5 mg/day, and titrated up to 1200 mg/day but was later reduced to 400 mg b.i.d. because of sedation. Ms. B experienced marked reduction in her panic frequency and substantial improvement in irritability and depressive symptoms. This improvement persisted over 6 months while Ms. B continued on a regimen of gabapentin along with her other concurrent medications.

Mr. C was a 54-year-old man with a history of generalized anxiety disorder dating back to childhood and intermittent checking rituals not fully satisfying the criteria for obsessive-compulsive disorder. Over the years, Mr. C had numerous trials with tricyclics and selective serotonin reuptake inhibitors of adequate dose and duration without significant improvement. He derived moderate benefit from diazepam, up to 30 mg/day, but was not able to tolerate higher doses because of sedation. Treatment was initiated with gabapentin, 100 mg t.i.d. Over a 2-week period, Mr. C experienced marked improvement in generalized symptoms of worry and autonomic arousal. This improvement was sustained at 3-month follow-up, and the diazepam was reduced to 10 mg/day. There was no change in his checking rituals.

Ms. D was a 52-year-old woman with attention deficit hyperactivity disorder (ADHD), generalized anxiety, and major depressive disorder, recurrent type. Her ADHD and depression had been effectively treated with dexedrine, 5 mg p.o. t.i.d.; however, she was intolerant of or unresponsive to multiple trials of antidepressants, benzodiazepines, and buspirone for treatment of her anxiety. A trial of gabapentin, titrated up to 100 mg p.o. b.i.d., was initiated, resulting in marked improvement in her anxiety and reduction in her intermittent use of alcohol to self-medicate. Gabapentin also reduced the overstimulation she occasionally experienced with dexedrine. Benefit persisted on this regimen at 3-month follow-up.

Gabapentin is a generally safe anticonvulsant without significant drug and alcohol interactions, need for plasma monitoring, or the liability of abuse or dependency. As illustrated here, patients with an alcohol abuse history for whom benzodiazepines are relatively contraindicated may apparently safely use gabapentin. Our preliminary observations suggest a role for gabapentin as monotherapy or for adjunctive use in patients with panic disorder or generalized anxiety disorder. The promising preliminary results encourage further clinical exploration and systematic study of gabapentin for the treatment of anxiety disorders.

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Intravenous Clomipramine for a Schizophrenic Patient With Obsessive-Compulsive Symptoms

TO THE EDITOR: Obsessive-compulsive symptoms, documented in as many as 25% of schizophrenic patients, pose a substantial therapeutic challenge. The addition of clomipramine to the neuroleptic regimen has alleviated both schizophrenic and obsessive-compulsive symptoms (1). In some patients with obsessive-compulsive disorder (OCD), intravenous administration is superior to the oral administration of clomipramine (2). We report on the beneficial effects of adding intravenous clomipramine to a regimen of neuroleptics for a stabilized schizophrenic patient with severe obsessive-compulsive symptoms.

Ms. A, a 25-year-old woman who had had schizophrenic disorder, paranoid type (according to DSM-IV), since the age of 17 years, was hospitalized twice because of acute psychotic exacerbation. After treatment with various antipsychotics, she was stabilized with perphenazine (8 mg/day). Ms. A also manifested ego-dystonic checking and cleaning rituals related to an obsessive fear of contamination. The subsequent addition of fluvoxamine (250 mg/day for 12 weeks) and fluoxetine (40 mg/day for 8 weeks) to the neuroleptic treatment failed to control her obsessive-compulsive symptoms. Further deterioration of the compulsive behavior resulted in her rehospitalization. At this point, in addition to the previously observed rituals, Ms. A was exercising compulsively for 12–14 hours per day. Despite her awareness of the irrationality of her behavior, our attempts to stop the exercising caused her severe anxiety. We initiated a course of intravenous clomipramine (75 mg in 1500 cc of normal saline) in addition to the ongoing perphenazine (8 mg/day); we repeated the 4-hour infusion procedure 24 hours later. Five days after the second infusion, Ms. A's score on the Yale-Brown Obsessive Compulsive Scale dropped from 19 to 4, an indication that her compulsive behavior had almost completely disappeared. This was the first notable improvement since the onset of her obsessive-compulsive symptoms. In addition, Ms. A's score on the Brief Psychiatric Rating Scale dropped from 41 to 29, and her monitored ECG, pulse, and blood pressure were within normal limits. The only side effect was mild sedation. We subsequently gave Ms. A prescriptions for oral clomipramine (150 mg/day) and perphenazine (8 mg/day) and discharged her from the hospital. We observed no recurrence of her obsessive-compulsive or schizophrenic symptoms during a 6-month follow-up.

The combination of clomipramine and neuroleptics has been recommended as a first-line treatment in schizophrenia with comorbid OCD (3). In the case described here, intravenous clomipramine was effective and well tolerated in a stabilized schizophrenic patient with refractory obsessive-compulsive symptoms who was receiving concurrent neuroleptic treatment. Our observation is consistent with the recently documented beneficial effect of pulse loading intravenous clomipramine in treatment-resistant OCD (2). However, we used a lower dose (75 mg/day versus 150–200 mg/day) and a slower rate of administration (4 hours versus 90 minutes). The robust and rapid therapeutic response to intravenous clomipramine pulse loading has been explained by immediate achievement of the therapeutic plasma or brain concentration, with subsequent effect on gene expression (2). If these findings are confirmed under placebo-controlled conditions, intravenous clomipramine may expand our options for treating obsessive-compulsive symptoms in schizophrenic patients.

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Hypersalivation Coincident With Olanzapine Treatment

TO THE EDITOR: Drooling, often manifested as pillow wetting during sleep, is a common adverse effect of the atypical antipsychotic clozapine (1). In light of clozapine's propensity to cause anticholinergic effects, it is surprising that patients complain of drooling rather than of dry mouth—the expected salivary anticholinergic response.

Olanzapine, a derivative of clozapine, is an atypical antipsychotic with a receptor-binding profile similar to that of clozapine (2). To our knowledge, there are no published descriptions besides the following one of drooling as a side effect of olanzapine treatment.

Ms. A was a 20-year-old Caucasian woman with a 3-year history of schizophrenia. After an initial 4-week trial of olanzapine, 10 mg/day, with substantial clinical improvement, she was treated solely with olanzapine, 15 mg/day. Her early spontaneous complaints of adverse effects included experiencing morning grogginess and soaking her pillow with saliva during sleep. When we increased her olanzapine dose to 15 mg/day, her morning grogginess and pillow wetting worsened. A physical examination revealed a normal gait and no muscular rigidity.

Among patients treated with antipsychotics, parkinsonism-related drooling is due to throat muscle rigidity and a resultant decrease in saliva swallowing. With clozapine, drooling and pillow wetting presumably result from increased saliva production (hence, the usual descriptors, "hypersalivation" and "sialorrhea"), although the mechanism has not been definitively demonstrated. It is unlikely that Ms. A's drooling was related to parkinsonism because she had no extrapyramidal symptoms.

Two proposed mechanisms for clozapine-induced hypersalivation may be relevant here (3–5). Increased saliva production results from salivary sympathetic α-adrenergic receptor antagonism or from parasympathetic cholinergic

(muscarinic) receptor agonism. Ms. A's sialorrhea may have been a result of olanzapine's potent α-adrenergic antagonism. Alternatively, sialorrhea may stem from stimulation of salivary muscarinic receptors. Both clozapine and olanzapine have high affinity for the five muscarinic receptors (M1–M5), and both are antagonists at M1–M3 and M5, consistent with anticholinergic effects. Clozapine (4, 5) and olanzapine (5) are reported to be agonists or partial agonists at M4 receptors. This explanation is speculative because the importance of M4 receptors in human saliva production is unknown.

While the cause of pillow wetting for Ms. A as well as clozapine-treated patients is not understood, anticholinergic and adrenergic agonist therapies are used for clozapine-induced pillow wetting (1, 3). Although Ms. A declined pharmacological treatment, she welcomed discussion of nonpharmacological management (e.g., covering her pillow with a towel or sleeping on her side propped up with extra pillows). Physicians should monitor patients treated with olanzapine for this potentially troublesome adverse effect.

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Screening for Depression in Elderly Patients

TO THE EDITOR: Chochinov et al. (1) compared four brief screening measures for depression in terminally ill patients. They concluded that for diagnostic purposes, brief screening measures do not approach the validity of a single-item interview that asks, in effect, "Are you depressed?" We have a number of reservations with regard to this conclusion.

First, Chochinov and his colleagues compared the brief measures with the standard of a semistructured interview and the Research Diagnostic Criteria. Such standard criteria may not be valid for elderly (mean age of the study group was 71 years) depressed patients (2). For example, community surveys of geriatric depression using instruments designed and validated for the elderly report much higher rates of depression than do epidemiological studies using standard criteria (3).

Second, depressed patients over 65 years old are less likely than younger patients to complain of low mood (4). This observation suggests that the single-item interview could be expected to have low sensitivity for significant depressive illness in this population.

Finally, a comparison of liaison versus consultation models for geriatric inpatients (5) reported a much higher rate of diagnostic accuracy for depression by referring doctors in the liaison model. On the basis of data from this study (5), the approach suggested by Chochinov et al. would be expected to have a low specificity. A more appropriate approach to detection of depression in the physically ill would be an emphasis on liaison rather than encouragement of nonpsychiatric colleagues simply to ask their patients, "Are you depressed?"

In conclusion, we believe that the standard used by Chochinov et al. may be invalid for the population studied. A more appropriate standard would have been an instrument validated for an elderly population with physical illness or a positive response to psychiatric intervention.

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Dr. Chochinov and Colleagues Reply

TO THE EDITOR: Drs. Swanwick and Wrigley raise a number of important points to which we are pleased to respond. While standard systems such as the Research Diagnostic Criteria may or may not be ideal for elderly depressed patients, an alternative "gold standard" for this population has yet to achieve common acceptance. Alternative criteria have been proposed for elderly patients who are physically ill (1), but they largely identify the same depressed patients as standard criteria (2, 3). Hence, we doubt that their application in this particular study would have made much difference. We would, however, encourage additional research to develop and validate appropriate techniques to identify depression in this patient population.

Depressed elderly patients may indeed be less likely than younger patients to complain of low mood. We, in fact, recommended a two-item screening approach (assessing loss of interest as well as depressed mood), since it provides complete coverage of the core criterion symptoms of depression. The same logic is incorporated into most structured interviews, which allow skipping the remaining symptoms if depressed mood and loss of interest are both absent. In our study, however, addition of the loss-of-interest item did not actually improve diagnostic accuracy over the single-item approach. The accuracy of single-item screening in identifying depressive disorders in older adults has also been reported by other investigators (4).

Finally, our results are unarguably criterion specific. It is worth noting that even with standard criteria, depression among the terminally ill tends to be underdiagnosed (5). This may reflect, in part, clinician discomfort in probing too deeply into the psychological experiences of dying patients (6). To

suggest that single-item screening diminishes psychiatry's important role in addressing these issues is simply reading too much into our results. We hope our study serves as a gentle reminder to clinicians to ask their dying patients—even in a simple way—about their mood states. Doing so may help increase recognition of depression in this vulnerable population.

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Pulse Clomipramine for Depressed Adolescents

TO THE EDITOR: Dr. Sallee and his colleagues (1) concluded that "the rate of response to pulse clomipramine was robust." However, it is difficult to accept their conclusion. Indeed, when responders were defined by a decrease of 50% or more from baseline in Hamilton depression scale scores, there were seven responders to clomipramine and three responders to the placebo, and this difference did not reach statistical significance. Moreover, this difference should be paralleled with the rate of prior treatment in the two groups: three of the clomipramine-treated patients versus seven of the placebo-treated patients had received previous treatment. Therefore, the higher rate of treatment-resistant depression in the placebo group might account for the difference between the two groups in rates of responders. This hypothesis seems to be confirmed by the low response rate to a subsequent open-label treatment with various selective serotonin reuptake inhibitors (SSRIs) among placebo-treated patients.

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Dr. Sallee Replies

TO THE EDITOR: The comments of Drs. Chabrol and Peresson about our assertion that "the rate of response to pulse clomipramine was robust" reconfirm the need for caution in overinterpreting studies with a small group size. It is true that when responders were defined by a decrease of 50% or more from baseline in Hamilton depression scale scores, differences by treatment did not reach statistical significance. It is not true, however, that treatment-resistant patients were disproportionately assigned to saline placebo. Random assignment of all subjects without regard to prior treatment led to a greater number of previously treated patients in the saline group (N=5) than in the pulse intravenous clomipramine group (N=1) for a total of six previously treated subjects. We did not, however, include in the text a full description of these failed trials. At least two patients in the saline group and the one previously treated patient assigned to the group could be described as treatment resistant (i.e., having failed to respond to two or more trials of adequate dose and duration). These patients accounted for many of the multiple treatments described. Two saline patients, however, were previously treated with a single course of SSRI, which could be characterized as either of inadequate dose or duration (6–8-week course). The low response rate (28%) of the saline cell to subsequent open-label SSRI treatment that we found in our study is not unusual within the adolescent population and cannot confirm a lack of therapeutic responsivity in the saline cell. As with all small studies, more hypotheses are generated than can be adequately addressed by the data. It is important to note that the study supports, but does not confirm, the ability of depression in adolescents to respond to antidepressant therapy.

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Intellectual Decline in Schizophrenic Patients

TO THE EDITOR: Russell and his colleagues reported on the WAIS-R test results of adults with schizophrenia who had been previously tested as children (1). They interpreted their results as suggesting "stable impairment" and provocatively entitled their paper "Schizophrenia and the Myth of Intellectual Decline." Because this "myth" was based on numerous empirical studies, it is important to examine the methodological details of the study by Russell et al. to reconcile the apparent contradictions. There are five major reasons to doubt their conclusions and to infer that the appearance of stability is, instead, likely evidence of intellectual decline.

1. The WAIS-R short form used on the second testing occasion probably overestimates the full-scale IQ of schizophrenic patients because it does not include several subtests on which poor performance is common among this group. To address this issue empirically, I examined the WAIS-R performance of 103 schizophrenic patients at the National Institute of Mental Health (NIMH). The group's mean full-scale IQ was 88.3 (SD= 11.9) with a score of 97.5 (SD=88.3) on the Wide-Range Achievement Test-Revised; those scores suggested a 9.1-point decline (matched pair t test: $t=8.15$, $df=102$, $p<0.0001$). The patients' performance on Russell et al.'s five-subtest short form (mean= 8.77, SD=2.2) differed significantly ($t=6.53$, $df=102$, $p<0.001$) from their performance on the remaining six subtests (mean= 8.02, SD=1.9) as well as on all 11 subtests (mean=8.40, SD=2.0). This significant difference occurred despite a high correlation ($r=0.94$) between short-form and full-scale IQs. Thus, subjects retained their relative positions across short-form and full-scale IQs, but the short-form estimates were systematically higher than the actual full-scale IQs. Because all WISC-R subtests were administered at time 1, the comparison of actual WISC-R full-scale IQ versus short-form WAIS-R IQ is biased against detecting differences.

2. Age cohort effects confound the use of WISC-R and WAIS-R scores to determine the longitudinal course of intellectual func-

tioning. As reviewed by Kaufman (2), each restandardization of the major IQ tests has documented substantial "gains" in IQ, estimated at 3 points per decade in the United States, where the IQ tests were normed. This cohort effect influences examinations of individual performance over time: the WISC-R was published in 1974; the WAIS-R, in 1981. At time 1, the group was 13.3 years old; their IQs were calculated relative to those of persons born in 1961. At time 2, the group was 32.9 years old; their IQs were calculated relative to those of individuals born in 1948. This 13-year age difference is likely responsible for a 3–4 point WAIS-R advantage relative to the WISC-R. In addition, the WAIS-R norms for 16- to 19-year-olds have been criticized as producing spuriously high scores (2). At time 2, at least one subject fell in this age range. Again, the psychometric problem decreased the probability of documenting intellectual loss.

3. Several studies reviewed by Kaufman (2) have suggested that WAIS-R scores of intellectually limited subjects are systematically higher than their WISC-R scores, with differences as high as 11 points reported. Thus, equivalent WISC-R IQ and WAIS-R IQ may be evidence of actual IQ decrement among these subjects. Russell et al. included six or seven subjects with WISC-R IQs lower than 75; the inclusion of such intellectually limited subjects would be particularly problematic and would raise possible diagnostic issues.

4. As noted by Russell et al., their group was highly unrepresentative of schizophrenic patients. The subjects had childhood-onset psychiatric symptoms, low overall IQs, and a substantial number of childhood-onset psychoses. What, if any, is the possible justification for generalizing the findings of Russell et al. to schizophrenic patients as a whole, as implied by the title?

5. The inclusion of nine subjects who were psychotic at time 1 undermined any examination of decline related to onset of psychotic illness, confusing this issue with deterioration over illness course. The fact that these subjects did not differ significantly from the rest of the group at time 1 or time 2 (a comparison with remarkably limited power) is not relevant to the main argument. The question of intellectual decline over illness course is distinct from the question of loss of intellectual ability with illness onset. Furthermore, the deletion of these nine subjects would have reduced the group to 25, a small number to use to confidently dispel a myth.

In summary, there are several reasons to suspect that the present comparison of estimated WAIS-R scores and WISC-R scores in schizophrenic patients resulted in the appearance of stability when actual loss, obscured by psychometric confounds, occurred. Considering that a decline of 2.3 points was documented without the contribution of these likely artifacts, a true decline of 8–10 points, the extent of decline estimated by using a variety of methods, may actually have been present.

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Drs. Russell and Murray Reply

TO THE EDITOR: In his letter, Dr. Gold puts forward five points from which he concludes that the "appearance of stability" in IQ in our subjects is actually "likely evidence of intellectual decline." We discussed three of Dr. Gold's points in our original paper; the remaining two are matters of test artifact.

1. Dr. Gold raises the issue of the short form of the WAIS-R and refers to data he has collected on 103 adults with schizophrenia by using the WAIS-R. His results revealed a lower mean subtest score when 10 subtests, rather than just five, were administered; however, equivalent IQs were derived from both. The difference between the means of the 10- and the five-subtest scores is statistically significant, but it would never be considered practically significant. The IQ literature suggests that a scaled score should be at least 3 points above or below the mean before it can be considered noteworthy (1). Dr. Gold presents scores that are approximately one-third of a scaled score from the mean.

In the original article, we presented considerable data about the reliability and validity of the five-subtest short form of the WAIS-R. An advantage of the short form is that it omits several of the subtests (e.g., information and arithmetic) that assess educational attainment. Furthermore, we chose the tests to control for the disabling effect that a long period of serious mental illness may have had on this group's ability—for example, their ability to name accurately five prime ministers of Great Britain since World War II. We also considered issues of test fatigue in patients who, in general, remain chronically unwell (2).

2. Gold points out that we included six to seven subjects with IQs lower than 75 and raises the issue of comparing WISC and WAIS scores among subjects termed "intellectually limited." The cutoff for "intellectually limited" is usually a full-scale IQ lower than 70. We have, therefore, reexamined our data in subjects whose full-scale IQs were lower than 70. During the follow-up period, 50% of these subjects showed improved IQ, and 50% showed a decline. There were no significant differences between IQs at time 1 and time 2 in the subjects whose full-scale IQs were lower than 70, a finding similar to the findings for the group overall. Thus, there is no substance to Dr. Gold's argument.

We do agree with Dr. Gold that one must be cautious in interpreting longitudinal data. However, our study did control for the age cohort effects of cross-sectional studies and the questionable validity of IQ estimative methodology upon which the "myth" has been built.

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