Letters to the Editor

Lethal Interaction of Clozapine and Buspirone?

TO THE EDITOR: As more psychotropic medications are used in new combinations for complex and treatment-resistant psychiatric disorders, serious and unexpected adverse pharmacologic interactions increasingly can occur. The following case report describes a patient in whom acute, potentially lethal gastrointestinal bleeding and marked hyperglycemia occurred after buspirone was added to a regimen of clozapine.

Mr. A, a 33-year-old man with paranoid schizophrenia and a history of polysubstance abuse, would engage in repeated, unpredictable, and serious assaultiveness as a result of command hallucinations; extended inpatient care was required. There had been no previous medical problems, and laboratory analysis values were within normal limits. Results of an EEG and a magnetic resonance imaging scan revealed no abnormality. Since treatment with phenothiazines had resulted in dystonia, Mr. A was placed on a regimen of clozapine, 600 mg/day, which led to a substantial reduction in hallucinations and assaultiveness. However, he continued to complain of difficulty concentrating, panicky feelings, and a nocturnal fear of dying. The possibility of comorbid underlying adult attention deficit disorder, as well as generalized anxiety or panic disorder, was considered. Successive trials of lorazepam, clonazepam, and β blockers had very limited benefit and were discontinued. On the basis of Mr. A's report of prior benefit from caffeine, a trial of over-the-counter caffeine tablets was attempted for treatment of residual attention deficit disorder but did not prove useful. At this point Mr. A had been taking clozapine for more than a year without any adverse effects; his clozapine serum level was 390 ng/ml (reference range=100–700 ng/ml).

Because of the persisting anxiety symptoms, a trial of buspirone was begun. In the first week, the dose was increased to 5 mg t.i.d., and Mr. A soon described feeling somewhat better. One month later the buspirone dose was raised to a total of 20 mg/day. One week after this last increase, Mr. A began to look physically ill and complained of nausea and epigastric pain. His abdomen was distended. After a "coffee-grounds" emesis that was consistent with gastrointestinal bleeding, he was rushed to intensive care. He was found to have severe acidosis and a blood glucose level of over 1300 mg/dl. His hematocrit level had dropped to 31 ml/dl. Results of amylase and liver function tests revealed no abnormality. The clozapine and buspirone regimens were discontinued. An upper gastrointestinal series failed to detect a source of the bleeding, such as ulcer disease. An endocrinologic workup revealed the following results: negative testing for antibodies to pancreatic islet cells, normal glycohemoglobin, and normal C-peptide insulin cleavage products. Mr. A was treated with insulin, and gradually the hyperglycemia abated. Eventually his blood glucose level returned to normal, and he no longer required insulin. However, clozapine discontinuation caused the assaultiveness to return. He was then treated with fluphenazine, which did not result in substantial benefit. Consequently, the regimen was switched back to clozapine without buspirone cotreatment. There was no recurrence of adverse effects, and Mr. A returned to his previous level of functioning.

According to the package insert for clozapine, adverse reactions may include gastric ulcer, hematemesis, and hyperglycemia. Case reports of hyperglycemia (1, 2) include its association with moderate and high doses of clozapine. Although pancreatic islet cell necrosis apparently has not been documented with clozapine, pancreatitis has been reported (3).

I have been unable to locate any reports of an adverse clozapine-buspirone interaction, and it is possible that the reaction was only coincidental with the addition of buspirone. However, given that for over a year before the addition of buspirone this patient had had no problems with clozapine and that no such effects recurred when clozapine alone was resumed, the possible role of buspirone in combination with clozapine must be considered. Although the patient's clozapine level was not determined during cotreatment with buspirone, it had previously been within the middle therapeutic reference range. It is possible that the addition of buspirone augmented the serum level of clozapine, either by enzyme inhibition or by displacing clozapine from its binding sites. The latter is a likely possibility, since both drugs are extensively bound to serum proteins, and there does not appear to be evidence that buspirone significantly affects the cytochrome P450 isoenzyme system, which is involved in the metabolism of clozapine (4). On the other hand, buspirone is known to increase the serum level of haloperidol, probably by competitive inhibition of oxidative dealkylation of haloperidol (5; Buspar package insert). In addition, reports suggest that erythromycin (6), fluoxetine, fluvoxamine, and caffeine (7) can increase clozapine levels, probably due to inhibition of the P450 isoenzyme system. Alternatively, buspirone and clozapine might have had additive, receptor-mediated effects, although this seems less likely on the basis of the known side effect profile of buspirone. Clozapine, for example, antagonizes serotonin (5-HT) receptors, and buspirone is a 5-HT_{1A} agonist. In any event, the reaction described in this case suggests taking great caution in the combination of clozapine and buspirone. Had this patient been an outpatient without quick access to acute medical care, the final outcome might have been more serious.

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Clozapine-Induced Stuttering

TO THE EDITOR: Adult-onset, neurogenic stuttering occurs infrequently as a sequela of neurotrauma or stroke or as an adverse effect of medication. Drug-induced stuttering has been reported with psychoactive medications, including selective serotonin reuptake inhibitors (1), tricyclic antidepressants (2), phenothiazines (3), and clozapine (4). We report an additional case of stuttering associated with clozapine.

Ms. A was a 28-year-old woman who was hospitalized with a diagnosis of schizoaffective disorder for which she had been treated since her early teens. Her symptoms had been unresponsive to haloperidol and lithium treatment; thus, these medications were discontinued, and a trial of clozapine was instituted. During the second month of clozapine therapy, shortly after the daily dose was increased to 400 mg, she began stuttering. This dysfluency persisted as the clozapine dose was gradually increased over the next 3 months up to 900 mg/day, during which time her symptoms did not improve. The clozapine dose was then gradually decreased. Her stuttering ceased when the clozapine dose was at or below 700 mg/day. A subsequent session of ECT also failed to ameliorate her symptoms. However, when haloperidol and lithium were prescribed in addition to clozapine, limited improvement was seen. The stuttering did not recur.

Electrophysiological (5) and positron emission tomography (6) studies of developmental stuttering have shown functional abnormalities in a variety of brain regions that serve the planning, execution, and self-monitoring of speech production. Multiple neurotransmitter systems are involved in the interconnections among these regions. It is therefore not surprising that many drugs with a diversity of mechanisms of action can induce stuttering, nor that an equally diverse array of compounds has been employed in its treatment (7), with variable success. Of the neurotransmitters that have been pharmacologically implicated in the etiology of stuttering, clozapine binds to the receptors of at least three (dopaminergic, muscarinic, and α-adrenergic receptors). Thus, clozapine's addition to the list of stutter-inducing compounds is not unexpected.

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Withdrawal Syndrome After Discontinuation of Venlafaxine

TO THE EDITOR: A possible withdrawal syndrome was recently described in a young woman who had been treated with venlafaxine, 300 mg/day, over an 8-month period (1). We report here two cases in which elderly patients who had been taking venlafaxine for some weeks may have undergone withdrawal after its abrupt discontinuation.

Ms. A, a 70-year-old woman with major depression, was treated with a regimen of venlafaxine, 225 mg/day, in combination with lorazepam, 2 mg/day. Although the antidepressive therapy resulted in an elevation of her mood, venlafaxine treatment was discontinued after 26 days because of persistent insomnia; the lorazepam regimen remained unchanged. About 20 hours after venlafaxine treatment was discontined, Ms. A became confused and began to suffer from nausea, a diffuse headache, agitation, sweating, anxiety, and visual hallucinations. Within 1 hour, there was a wide variation in her blood pressure (from 90/70 to 160/110 mm Hg) and heart rate (from 88 to 112 bpm). Her body temperature was 37.8°C. The severity of symptoms required close monitoring for about 8 hours in the psychiatric intensive care unit; over the following 16 hours all symptoms gradually and completely resolved.

Ms. B was a 73-year-old woman with a depressive syndrome and a known arterial hypertension. She had received a regimen of venlafaxine, 225 mg/day over a period of 42 days, that had been discontinued because of persistent feelings of loss of energy and melancholy. Concomitant medication (clonazepam, 1 mg/day; a diuretic [triamterene, 50 mg/day, in combination with hydrochlorothiazide, 25 mg/day]; and metoprolol, 50 mg/day) was continued without any change in dose. About 16 hours after venlafaxine treatment was discontinued, Ms. B began to suffer from nausea, feelings of abdominal distension, headache, restlessness, agitation, and excessive sweating. She also complained of visual hallucinations in the form of persons. Her blood pressure (160/90 mm Hg) and heart rate (96 bpm) were both within normal ranges. The symptoms completely resolved within 2–3 hours after venlafaxine (75 mg) was reinitiated. During the following 4 days the dose of venlafaxine was gradually reduced, and the symptoms did not recur.

In the previously reported case of possible venlafaxine withdrawal (1), the patient suffered from headache and gastroin-

testinal distress, as was seen in the two cases reported here. Although a venlafaxine-benzodiazepine drug interaction cannot be excluded completely, pharmacokinetic and pharmacodynamic data indicate only clinically insignificant interactions (2) that would not account for the patients' symptoms seen here. Venlafaxine-associated withdrawal symptoms may be explained by the drug's neurobiologic properties of blocking both serotonin and norepinephrine reuptake. Since withdrawal symptoms such as gastrointestinal distress and headache have been reported after abrupt discontinuation of tricyclic antidepressants (3) as well as serotonin reuptake inhibitors (4), it may be possible that both transmitter systems were involved in producing withdrawal symptoms in our patients. The potential risk of severe withdrawal symptoms underlines the need for tapering of venlafaxine before discontinuation, especially in elderly patients.

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Paroxetine Treatment of Sexual Disinhibition in Dementia

TO THE EDITOR: Sexual disinhibition in demented individuals can be extremely problematic, especially in institutional settings. Neuroleptics and benzodiazepines are commonly used for this problem but are usually ineffective and poorly tolerated. Antiandrogenic agents (medroxyprogesterone [1] and leuprolide [2]) are better tolerated but require parenteral administration. Selective serotonin reuptake inhibitors (SSRIs) are reportedly effective for treatment of paraphilias and hypersexuality (3, 4); to our knowledge, SSRIs have not been used in the treatment of individuals with dementia. We report here a patient with alcoholic dementia and sexual disinhibition who was successfully treated with paroxetine.

Mr. A, a 69-year-old man with an 8-year history of dementia and a lifetime of heavy alcohol abuse, had been hospitalized five times for inappropriate sexual behavior. Behaviors included fondling or exposing himself to female patients, staff, and visitors at assisted-living facilities, masturbating in public, and repeated graphic requests for sexual favors. During the previous 5 years, he had lived in a number of all-male assisted-living facilities and had been discharged from several because of hypersexuality; however, he had not been rehospitalized. He displayed no psychotic, manic, or aggressive symptoms.

A review of Mr. A's medical and psychiatric history was noncontributory. A mental status examination revealed moderate deficits in memory and orientation, but language, praxis, visuospatial orientation, calculations, and executive function were preserved. Results of a laboratory workup for dementia were unremarkable.

Mr. A failed to respond to a variety of psychotropic drugs (haloperidol, thioridazine, lorazepam, lithium, and nortriptyline) that were often given in combination and in relatively high doses. He ultimately consented to a trial of paroxetine, 20 mg/day. Within 1 week, staff at the assisted-living facility reported that the hypersexuality had improved dramatically, around 95% in their estimate. After 3 months, this improvement had been maintained, with no substantial side effects.

The mechanism of action of SSRIs in such patients is unclear. Several authors have suggested that hypersexual and paraphilic behaviors may be related to obsessive-compulsive disorder and that the efficacy of SSRIs may be due to their antiobsessional effect (3, 4). On the other hand, a more direct antilibidinal effect may apply (3), since reduced libido is commonly seen with SSRI treatment (5). Although not a common problem, sexual disinhibition in institutionalized demented individuals is very difficult to tolerate and frequently results in placement failure and rehospitalization; treatment with SSRIs may constitute a relatively safe, convenient, and effective strategy.

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Extrapyramidal Symptoms From Intravenous Haloperidol in the Treatment of Delirium

TO THE EDITOR: Delirium is a frequent occurrence in critically ill patients in the intensive care setting and is often associated with agitation and behavioral difficulties (1, 2). Intravenous haloperidol is commonly used to control delirium because it is very effective, has few side effects, and may cause fewer extrapyramidal symptoms than if given orally (1–3). Despite its efficacy, safety, and widespread use, intravenous haloperidol may still cause extrapyramidal symptoms, which can present as other psychiatric symptoms and cause substantial morbidity. We present such a case.

Ms. A was a 55-year-old woman whose previous episode of generalized anxiety disorder had been treated with buspirone, 10 mg t.i.d. She was admitted to our hospital because of shortness of breath that would progress upon exertion to dyspnea and a productive cough. She was treated for a pneumococcal pneumonia but subsequently developed

acute respiratory distress syndrome and was transferred to the intensive care unit. She was intubated and given a tracheostomy. Over the next several weeks, her agitation increased despite treatment with benzodiazepines and narcotics. A standing order of intravenous haloperidol, along with as-needed doses, was added to the regimen. Her agitation was well controlled at an average haloperidol dose of 46 mg/day; the maximum daily dose was 150 mg. Over 3 weeks she received a total of 910 mg of haloperidol intravenously and 108 mg intramuscularly. Consultation with the psychiatry service was requested; Ms. A was described as having a "flat affect" and possible depression.

Because she had had a tracheostomy, Ms. A was unable to communicate except by shaking her head and blinking her eyes. Although she admitted to feeling sad and depressed, her presentation was remarkable for masked facies, bradykinesia, cogwheel rigidity of her upper extremities, and a "pill-rolling" tremor of her hands. She was diagnosed with neuroleptic-induced parkinsonism. Her haloperidol treatment was stopped, and she was started on a regimen of benztropine, 1 mg b.i.d. She experienced a subsequent resolution of her parkinsonism, and her mood and activity level normalized over the next 8 days. She was able to participate more actively in her rehabilitation and was discharged 10 days later.

Haloperidol is considered the treatment of choice for control of delirium in the critically ill patient (1). Because of its safety, efficacy, and familiarity, it is commonly used in intensive care settings without psychiatric consultation. This case demonstrates that substantial side effects such as parkinsonism still occur and may be easily overlooked or may mimic other psychiatric diagnoses such as depression. We believe that this case highlights the need for continued education of intensive care staff as well as psychiatrists regarding the side effects of neuroleptic medications.

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Atypical Neuroleptic Malignant Syndrome Associated With Risperidone Treatment

TO THE EDITOR: Controversy exists about the diagnosis of neuroleptic malignant syndrome, especially regarding rigidity as an essential criterion (1–3). Neuroleptic malignant syndrome without rigidity has been reported with clozapine, an atypical neuroleptic (4). Risperidone, also deemed atypical, causes extrapyramidal symptoms at higher doses and has been associated with neuroleptic malignant syndrome (5). A MEDLINE search through January 1997 revealed seven cases of risperidone-associated neuroleptic malignant syndrome, but none that occurred without rigidity (references available on request). We describe a patient who developed neuroleptic malignant syndrome without rigidity during risperidone treatment.

Mr. A, a 51-year-old man with a history of bipolar disorder, was hospitalized with manic and psychotic symptoms 1 week after the death of his brother. A previous mixed episode had been treated with valproate, 500 mg in the morning and 750 mg in the evening, and risperidone, 3 mg b.i.d. He had been compliant with his medication, and his risperidone regimen had been tapered off. On admission he was experiencing auditory hallucinations, delusions of guilt and persecution, pronounced manic symptoms, and formal thought disorder. His valproate dose was titrated to 750 mg b.i.d., a regimen of clonazepam, 0.5 mg b.i.d., was prescribed for anxiety, and treatment with risperidone, 3 mg b.i.d., was restarted. Five days later, he became lethargic. He was diaphoretic, tachypneic, tachycardic, and tremulous; however, there was no cogwheeling or rigidity. His temperature was 102.0°F, pulse was 120 bpm, respiratory rate was 32 breaths/min, and blood pressure was 140/92 mm Hg. Results of a laboratory examination revealed leukocytosis, hypernatremia, a BUN/creatinine ratio of 24:1.2, elevated liver function test results, and a creatine phosphokinase level of 2315 U/liter.

Mr. A was transferred to the intensive care unit for supportive care and observation. All medications were discontinued with the exception of valproate. Results of an extensive workup in the intensive care unit ruled out sepsis, myocardial infarction, pulmonary embolism, or a CNS event. He improved substantially after rehydration and discontinuation of risperidone; no treatment with bromocriptine or dantrolene was needed. He still evidenced manic and psychotic symptoms but was fully alert and oriented. His vital signs, leukocyte count, and liver function test results normalized; the creatine phosphokinase level fell to 195 U/liter. Mr. A was given the maximum valproate dose, and after 2 weeks olanzapine treatment was initiated. Mr. A tolerated this regimen well; the neuroleptic malignant syndrome-like episode did not recur.

Neuroleptic malignant syndrome is the best explanation in this case despite the absence of muscular rigidity. A complete medical workup excluded concomitant medical illness, all other criteria for neuroleptic malignant syndrome were present, and onset and remittance of the syndrome coincided respectively with the administration and discontinuation of risperidone. It appears that there may indeed be a clinical spectrum of neuroleptic malignant syndrome, and atypical presentations (without rigidity) may occur with atypical antipsychotics.

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Risperidone for Treatment of Childhood Schizophrenia

TO THE EDITOR: Although most cases of schizophrenia have their onset in late adolescence or early adulthood, schizophrenia has been recognized in children. Neuroleptic use in children is limited by the side effect profile, which includes sedation, apathy, extrapyramidal symptoms, and especially the risk of tardive dyskinesia. Risperidone is of special interest for pediatric populations because it shares the same therapeutic spectrum as classic neuroleptics but is hoped to be better tolerated (1). Recently, its efficacy in children and adolescents has been reported in case studies on treatment of developmental disorders (2), tic disorders (3), and adolescents with schizophrenia (4).

Andy was an 8-year-old boy with a 2-year history of very-early-onset schizophrenia; he lived with his biological parents. His main target symptoms included odd behavior, stereotypes, visual and auditory hallucinations, bizarre thoughts, aggressive outbursts, severe anxiety, and fearfulness. Results of thyroid function tests, CBC, an EEG, metabolic and chromosomal analysis, and a magnetic resonance imaging scan of the brain revealed no abnormalities. Risperidone treatment was selected because it has fewer reported side effects than typical neuroleptics. Andy had not been taking another neuroleptic. The risperidone dose was raised from 1 to 2 mg/day (0.07 mg/kg) within 1 week. After 4 weeks of treatment with the higher dose, the regimen was reduced to 1 mg/day and after 3 months was further reduced to 0.5 mg h.s. Four weeks after the beginning of treatment Andy was free of auditory and visual hallucinations. At 4-month follow-up, he was more cooperative, engaging, and free of aggressive outbursts at home. He had also started school in a special education class. Side effects included drowsiness during the 2-mg dose administration and a 21% weight gain (6.3 kg) during the 4-month treatment period.

After 4 months there had been a 61% improvement in positive symptoms and a 67% improvement in negative symptoms as measured by the Positive and Negative Syndrome Scale. His Children's Global Assessment Scale rating improved from 20 to 55. Parents' evaluation with the Child Behavior Checklist before treatment and at follow-up revealed substantial improvement in total raw score (from 105 to 43) and scores for externalizing (from 36 to 12), internalizing (from 28 to 14), thought problems (from 13 to 3), anxious/depressive symptoms (from 15 to 8), withdrawn symptoms (from 11 to 5), and aggressivity (from 27 to 9).

There is a lack of reports on risperidone treatment in children with very-early-onset schizophrenia. Risperidone treatment seemed successful for this child and thus may hold substantial promise in the treatment of children with psychotic disorders.

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E-Mail Therapy

TO THE EDITOR: Patient-doctor communication by e-mail, which has been growing in popularity, holds promises and pitfalls. E-mail obviates "telephone tag," and unlike telephone conversations, messages can be edited, printed, and filed. Time zones and hearing and speech impediments become irrelevant. One message can go to many recipients. Will group therapy be next?

Psychiatric applications vary in complexity. Appointment scheduling is simple. "Med checks" invite lengthier, more frequent communications. An innovative therapeutic adjunct at the Hospital for Sick Children in Toronto, Ability Online, keeps chronically ill pediatric inpatients in touch with distant schoolmates. An emotionally needy patient with low tolerance for intervals between appointments may be encouraged to commit thoughts to the word processor and e-mail them as an alternative to frequent telephoning.

As e-mail expands beyond brief communications and starts to blend with the therapeutic session, concerns arise over dynamic implications and blurring of boundaries. How best to respond, and what are the hazards? Should the therapist who encourages the needy patient's written communications between sessions respond not at all, tersely, or at some length? What if one neglects to check one's e-mail, or it gets lost in transmission? Will this be experienced as abandonment, emotionally or medico-legally? (Of course, this can happen with pagers and answering machines as well.)

E-mail, like "Web-surfing," can be exciting in its novelty and potential. Writes one psychiatrist, "I love email as a means of pts sending psychopharm info to me. Not many do, but boy is it great! They can write as much as they please, I can read it in a trice, at a time convenient for me, and reply in a flash" (personal e-mail communication from J.K. Pearce on PsyCom.Net, Feb. 15, 1997). Given its allure, are we at risk for overencouraging extratherapeutic, albeit digital, contact?

Is there danger of our missing nonverbal diagnostic signs conveyed by the spoken word: the inflection, pauses, or slurred or pressured speech? Need one add "smileys" such as :-) and other digital symbols for affect?

Relationship and affective issues aside, what about confidentiality, malpractice, and other issues? Digital information can be readily hacked, copied, and transmitted elsewhere. I may inadvertently leave my computer screen active for others to see while I momentarily walk away. If my patient travels to another state in which I am unlicensed and e-mails me a question about medication, by replying will I be violating that state's licensure regulations? Will I jeopardize my malpractice coverage?

What about reimbursement? Some health care professionals now charge for "cybertherapy"—unregulated advice, consultation, second opinions, and therapy on the World Wide Web. We have CPT codes for telephone time; why not for e-mail?

Psychotherapy by letter is nothing new; Freud analyzed

"Little Hans" this way. But we must consider the challenges to patient care as we go online into the 21st century.

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Lithium Assay Errors

TO THE EDITOR: Clinicians depend on accurate serum lithium levels to better assess lithium's efficacy and toxicity in order to make dose adjustments. In fact, an article that described important differences in psychosocial function associated with serum lithium levels in patients with bipolar I disorder was recently published in the *Journal* (1). However, very little attention has been paid to the accuracy of lithium measurements. A recent MEDLINE search found no reports in the literature that questioned the accuracy of these measurements, despite lithium's narrow therapeutic index (0.6–1.2 meq/liter) (2). At our institution for the developmentally disabled, significant inaccuracies in serum lithium results were recently identified.

The inaccuracy was discovered when a serum sample of a patient who was not taking lithium was mistakenly sent to our reference laboratory for a lithium determination. The laboratory reported a serum lithium level of 0.4 meq/liter, and on recheck it was 0.3 meq/liter. Sources of possible medication administration errors were ruled out. Over the next several days, 13 additional patients were noted to have higher lithium levels than expected. None of these patients displayed signs of lithium toxicity. After ruling out possible dose changes and errors in administration or sampling, we contacted the reference laboratory with our concerns.

The reference laboratory repeated 10 samples and used the ion-selective electrode method. Of these 10 samples, eight were the same result, and two were 0.1 meq/liter lower. The laboratory then sent five samples to another laboratory for atomic absorption spectrophotometry, which is considered accurate and precise (3). All five samples showed a 0.2–0.4 meq/liter decrease, with a mean decrease of 0.3 meq/liter. The atomic absorption spectrophotometry method provided consistently lower results than the ion-selective electrode method.

The reference laboratory suggested that the inaccuracies might have been due to aging of the ion-selective electrode. As the electrode ages the levels may drift higher. Because of the difference between the two methods, our reference laboratory has instituted a policy to randomly test serums known not to contain lithium and to periodically engage in parallel testing of the ion-selective electrode method versus atomic absorption spectrophotometry.

Clinicians are constantly reminded to treat the patient's signs and symptoms and not laboratory values. Unfortunately, psychiatric patients frequently have communication difficulties that impair their capacity to self-report symptoms of toxicity, and some may have movement disorders that make objective signs of toxicity difficult to interpret.

Accurate lithium levels are an important tool that is relied upon to make meaningful decisions (1). We hope that standardized quality assurance procedures for improving the accuracy of lithium measurements will be adopted by all commercial laboratories.

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Uncontrolled Buying

TO THE EDITOR: The review by Michel Lejoyeux, M.D., Ph.D., and colleagues (1) of uncontrolled buying recalls the work of Wilhelm Stekel. Among the earliest of Freud's followers, Stekel (1868–1940) was an early apostate as well. He was a prolific and wide-ranging writer with a particular interest in symbolism (2–5).

Stekel was highly interested in "peculiarities of behavior." His two-volume work by that title (6) contains extensive chapters on kleptomania (which he traced essentially to ungratified sexuality), and he regarded inordinate buying as a *forme fruste* of this disorder. He included in the book (pp. 286–308) a detailed case report of oniomania in a 23-year-old married woman who presented as depressed and anorgasmic with her husband. The woman reported several incidents of sexual abuse during childhood, together with rape by an uncle when she was 14. While complex, heavily symbolic, and perhaps anachronistic to a modern reader, Stekel's psychodynamic formulations found the buying mania derivative of unconscious wishes for sexual adventure. The patient bought books, for example, because their hard bindings were phallic and also because they represented the "pass-books" of prostitutes. Three months of analysis reportedly left the patient *lebensfreudig*, sexually responsive, and largely freed of her buying proclivities.

Regrettably, Stekel seems to have been deficient in ethical scientific rigor (4, pp. 135–136; 5, p. 215); there is evidence that some of his clinical reports were fabricated. Nevertheless, the work in question seems coherent and at least etiologically plausible. It may be that oniomania belongs in the burgeoning catalog of disturbances that are often associated with childhood sexual abuse. Certainly a number of the comorbid disorders noted by Lejoyeux et al. are in this category.

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THOMAS MAIER, M.D.
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Dr. Lejoyeux and Colleagues Reply

TO THE EDITOR: Dr. Maier has drawn our attention to Stekel's description of uncontrolled buying, which highlighted important clinical features and illustrated the fact that uncon-

trolled buying is frequent in married women with depression. Specific studies of this association have confirmed that 50%–100% of subjects with uncontrolled buying are also depressed. Stekel's patient presented kleptomania, which is also known to be associated with uncontrolled buying. Uncontrolled buying may be included, like kleptomania, in the spectrum of the impulse control disorders.

The psychodynamic explanation of uncontrolled buying, "a derivative of unconscious wishes for sexual adventure," cannot be generalized to the majority of the patients. In most cases, uncontrolled buying is related to depression. It is a means for alleviating low self-esteem and coping with unbearable stress, depression, or frustration. Uncontrolled buying can be used as an escape mechanism from depressive feelings. Patients who feel empty and sad shop compulsively in an attempt to restore a depleted self. We previously presented (1) the case of a 48-year-old woman who reported uncontrolled buying when she was depressed. She would buy, without taking heed of the price, new models of electric household appliances. For example, in 1 month she bought three televisions, three sewing machines, and two videotape recorders. Buying corresponded to periods of intense sadness. At these moments, the patient could not resist the desire to go out shopping and to spend large amounts of money. She described her shopping episodes as an intense urge to "go into a store and get something new." The patient noted that her buyings had an antidepressant effect. When she entered a shop and chose an article, she would feel less anxious and less depressed. After a very short period of euphoria, she would be invaded by culpability and a sense of guilt. She would leave shops, regretting her behavior, and she would not use the new equipment that she had bought. Uncontrolled buying led to many problems with her bank and debts that she could not reimburse. The patient was aware of the gravity of her behavior. She tried to avoid shops and asked her daughter or her husband to go shopping with her in order to avoid buying too much. All uncontrolled buying episodes disappeared after the administration of antidepressants. When the patient was no longer depressed, she stopped her excessive spending. Uncontrolled buying appeared to be directly induced by depression.

In clinical practice, it is of first importance to sytematically search for depression associated with uncontrolled buying. The treatment of comorbid depression with tricyclic antidepressants or selective serotonin reuptake inhibitors can reduce the frequency and the severity of the behavior and even induce a complete disappearance of uncontrolled buying.

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MICHEL LEJOYEUX, M.D., PH.D.
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JACQUELYN SOLOMON, PH.D.
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Self-Reported Life Satisfaction

TO THE EDITOR: Mark Atkinson, Ph.D., and colleagues (1) claim that medical specialties that service acutely ill patients are in a better position to address program effectiveness than specialties that deal with chronically ill patients. They specifically question the validity of self-report measures of life satisfaction. The study could have highlighted an interesting methodological issue, namely the severe drawback of the instrument with which they measured quality of life. However, the direction of the article, including the conclusion, overstates the boundaries of the study and by doing so, does a disservice to the field of mental health. Quality of life measures have been developed for people with mental illness, their psychometric properties have been investigated, and they address issues beyond symptom reporting (examples include references 2 and 3). Moreover, suggesting that someone with a mental disorder cannot be relied upon to report quality of life issues raises the philosophical question of whose life it is. The greater challenge to our field is to translate quality of life, as measured in patients with chronic mental illness, into quality adjusted years of life, which cost-effectiveness studies demand (4). Unfortunately, that issue was not addressed. The best conclusion drawn from Atkinson et al.'s article is to not use their quality of life measure.

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TO THE EDITOR: We read with interest the article by Dr. Atkinson and colleagues, who concluded that the validity of self-report measures of life satisfaction is questionable in affectively disturbed populations, since scores may be influenced by affective bias, poor insight, and recent life events.

At the request of the Iowa Department of Human Services, we surveyed 2,535 Medicaid-eligible subjects in Iowa who had received mental health services in fiscal year 1993 under the preexisting fee-for-service system before the implementation of managed care (1). Persons with schizophrenia constituted 33% of the total cohort. The self-report survey measured mental and physical health status, satisfaction with mental health services, and quality of life in several functional domains. Of surveys sent, 886 (35.0%) were returned, including 300 responses (33.9%) from persons with schizophrenia. Among the seven diagnostic groups surveyed, persons with schizophrenia reported the best mental health status, the highest satisfaction with their mental health, and high levels of satisfaction with their mental health services, economic status, occupational status, and residential status. However, these subjects had had more hospitalizations, were more likely to receive Social Security Disability Income, and were less likely to live independently than persons in other diagnostic categories.

From this study we concluded that self-report of satisfaction (whether of mental health, service satisfaction, or quality of life) was not a useful measure of clinical or functional status in persons with schizophrenia and that it was inappropriate to use satisfaction as the only measure of clinical outcome in

this population. We recommended that information about program quality and effectiveness should be obtained from multiple sources, including both primary data sources (e.g., clinical records, client interviews) and secondary data sources (e.g., utilization claims). Furthermore, if satisfaction was to be used as a primary measure of patient outcome, methodologies other than a mail-out survey of self-reported satisfaction should be employed because of the low response rate and nonrespondent bias.

Despite our expressed concern about the validity of self-reported satisfaction by persons with serious mental illness, mail-out surveys of self-reported satisfaction are, at present, the only direct measure of patient outcome being used by the managed care contractor. The response rates have been 10%–12%, and the proportion of survey respondents that have psychotic or affective disorders is unknown. The usefulness of such data in the evaluation of service efficacy for persons with serious mental illness is questionable. The justification for reducing services on the basis of these data will be even less certain.

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BARBARA M. ROHLAND, M.S., M.D.
DOUGLAS R. LANGBEHN, M.D., PH.D.
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Dr. Atkinson Replies

TO THE EDITOR: I would like to respond to Dr. Arfken's comments by saying that a colleague and I recently used a different quality of life instrument (Quality of Life Inventory [1]) for a different group of persons with affective disorder and replicated the observation that affective state has an impact on life satisfaction ratings (2). Thus, it seems unlikely, at least for this patient population, that such respondent bias is an instrument-specific phenomenon. Indeed, statements made by consumers and family members who are affected by various forms of chronic mental illness indicate a general acknowledgment of the difficulties that self-report measures pose during certain phases of their illness. I am sorry that this reader took our article to imply that consumer reports should be eliminated. Rather, we wanted to caution against a simplistic application of the self-report method and to convey the need for careful scrutiny of self-report data, since they may be confounded with the symptoms of mental illness.

Our findings are nicely complemented by the observations and comments provided by Drs. Rohland and Langbehn regarding response data from persons coping with schizophrenia. In our article, we concurred with their suggestion that such self-report measures be triangulated with data from multiple sources so as to help assure valid interpretation. As we gain greater understanding about the impact of confounding factors such as mood, insight, and mental lucidity on our data sets, survey designs or analytic methods can be tailored so as to account for such factors. These solutions are, of course, less attractive for funding purposes, since they introduce complexity into both the data collection and the interpretation processes.

As a final comment I would like to emphasize that quality of life and satisfaction measures have broader applications than to simply fuel consumer-based methodologies for funding allocation, a fact that may be overlooked in a managed care environment. When carefully gathered, quality of life data represent the life circumstances and personal experiences of the persons we seek to help. As such, they can provide ongoing feedback to primary caregivers and policy makers regarding specific areas of consumer concern. In my opinion these measures should be used not only to determine treatment outcomes but to inform the treatment process—enhancing both the individualization of care and the achievement or maintenance of a basic standard of personal welfare for those we serve.

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Effects of Pregnancy on Suicidal Behavior

TO THE EDITOR: I read with interest the article by Peter M. Marzuk, M.D., and colleagues (1) in which they reported that pregnant women have a significantly lower risk of suicide than women of childbearing age who are not pregnant. They hypothesized that pregnancy may exert a behavioral inhibiting effect on suicidal urges.

Another important reason for the reduced suicide risk may be that pregnancy is associated with a period of relative well-being in some women. For example, patients with panic disorder experience a marked reduction in the frequency and the severity of panic and related symptoms (2). Also, some patients with manic-depressive illness are thought to have no psychic trouble during pregnancy (3). A colleague and I reported a case series of three women with severe bipolar II disorder who did not respond to trials of various psychotropic drugs but had periods of sustained euthymia during their pregnancies (4). In McNeil et al.'s study (5), 80% of patients with a history of affective illness (mainly of bipolar type) reported feeling better, or the same as usual, during pregnancy. Further evidence that pregnancy might confer a protective effect comes from studies in which patients remained symptom free during pregnancy after discontinuation of lithium but experienced a relapse during the postpartum period (6).

The exact mechanism of mood stabilization during pregnancy is speculative because of the complex physiological changes that involve a number of hormonal systems. However, it is possible that the greater production of estrogen and progesterone during pregnancy has a therapeutic effect on depression.

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Dr. Marzuk and Colleagues Reply

TO THE EDITOR: We appreciate Dr. Sharma's letter. We agree that some women with mental disorders may experience a reduction in symptoms during pregnancy. As a consequence, they may be less likely to develop the suicidal mental states that are associated with serious psychiatric disorders and thus may be less likely to display suicidal behavior. In contrast, for other women, pregnancy heightens psychosocial stresses or exacerbates symptoms, particularly if they discontinue their medications. Although these women may feel suicidal, our data could suggest that pregnancy, through either biological or psychosocial mechanisms, inhibits them from committing suicide. Undoubtedly, there are many factors associated with the change in suicide risk during pregnancy.

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Benzodiazepine Receptor Binding and Schizophrenia

TO THE EDITOR: I read with interest the article by Geraldo F. Busatto, M.D., Ph.D., and colleagues (1) in which trend correlations were found between symptom severity and the binding potential of γ-aminobutyric acid$_A$ (GABA$_A$) and benzodiazepine receptors. Severity of positive symptoms was negatively correlated with benzodiazepine receptor binding in the left medial temporal region, and severity of negative symptoms was inversely related to receptor binding in the medial frontal cortex. In the discussion, Busatto et al. address several limitations of the study. One limitation that was not mentioned is the lack of taking into account potential structural abnormalities in the brain of patients with schizophrenia. A number of recent studies have found significant reductions in frontal or prefrontal brain tissue volume in patients with schizophrenia (2). In some studies, these reductions in frontal volume were found to be associated with negative psychotic symptoms. Frontal volume may also be associated with educational level and parental socioeconomic status, factors that were not taken into consideration by Busatto et al. In addition, significant reductions have been observed in temporal lobe volume, either predominantly left-sided or bilaterally (2). Since reductions in GABA$_A$ and benzodiazepine receptor binding potential can reflect a nonspecific measure of neuronal loss (3), correction for structural changes in the brain, i.e., by coregistration of single photon emission computed tomography and magnetic resonance imaging (MRI), is necessary before it can be concluded that the GABAergic transmission is affected specifically in schizophrenia.

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NICOLAAS P.L.G. VERHOEFF, M.D., PH.D.
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Dr. Busatto and Colleagues Reply

TO THE EDITOR: We are pleased to reply to Dr. Verhoeff's comments on our article. We agree that without MRI co-registration, one cannot conclusively exclude the possibility that the reported correlations that involve reduced regional ^{123}I-iomazenil binding were secondary to structural brain changes in schizophrenia. However, if the latter were the case, one would expect to see the same pattern of correlations with data from the early portions of the time-activity curve of ^{123}I-iomazenil, when ligand uptake predominantly reflects regional blood flow. We did investigate possible changes that involve indices of early regional ^{123}I-iomazenil binding taken from the first scanning sequence (1–15 minutes after the injection) by normalizing the regional measures either to the uptake in the white matter or to the mean uptake in the whole brain. No significant correlations between early ligand uptake and clinical variables were identified in the group of schizophrenic patients.

In any case, if the reported abnormalities of regional ^{123}I-iomazenil uptake were indeed secondary to frontotemporal anatomical disturbances in schizophrenia, this would still allow one to conclude that inhibitory GABAergic synapses are lost in consequence to such structural defect. We believe that such regional deficiency of GABAergic transmission, albeit not necessarily specific or of etiological primacy, is of potential relevance to alternative treatment strategies for schizophrenia. Selective GABA-acting agents targeted to the regions identified in our study could relieve schizophrenic symptoms by incrementing GABAergic transmission in remaining synapses that are possibly spared by the disease process.

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Violence and Cocaine

TO THE EDITOR: We read with interest the finding of Kenneth Tardiff, M.D., M.P.H., and colleagues (1) regarding the association of violence and cocaine use during the month before admission in female psychiatric inpatients.

Previously, we assessed behavioral disturbances in patients admitted to a psychiatric emergency room whose urine toxicology results indicated ingestion of cocaine or alcohol (2). In view of the findings of Tardiff et al., we reanalyzed our data for gender differences. We found that female patients who had ingested cocaine had a higher risk of aggression than male patients (18% versus 0%) (p=0.02, Fisher's exact test). Patients who had ingested alcohol had high rates of aggression (40%), and patients who had ingested both alcohol and cocaine had low rates of aggression (5%), but there were no significant gender differences (presence of alcohol: p=0.62, presence of both alcohol and cocaine: p=0.26 [Fisher's exact test]).

Our findings in psychiatric emergency room patients parallel the findings of Tardiff et al. in several, but not all, aspects. Both studies suggest a higher risk of aggressive behavior in female patients with recent cocaine use than in male patients. In contrast, Tardiff et al. report no association of alcohol use and aggression in male psychiatric inpatients. Differences in study design, subject populations, and ascertainment of substance use undoubtedly contribute to discrepant findings.

The consistency of both sets of data and the advantage of ascertainment by urine toxicology testing in our study support a gender-specific psychopharmacological action. This clinical finding adds to other reported differences between male and female cocaine users in controlled laboratory studies (3), neuroimaging studies (4), and epidemiological investigations (5). Further research on cocaine use and its psychiatric complications, including violent behavior, needs to take into account gender-specific differences.

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Dr. Tardiff and Colleagues Reply

TO THE EDITOR: We are pleased that the study by Drs. Dhossche and Rubinstein has shown findings similar to those in our study of violence among patients admitted to a private psychiatric hospital. By using an accurate measure of cocaine use (urine toxicology screen), they found that a higher risk of aggression was associated with the use of cocaine in women, but not in men, admitted to a psychiatric emergency department. It appears that cocaine may make female psychiatric patients just as violent as male patients. Presumably this is related to the pharmacologic effects of cocaine, which include irritability, impulsivity, hyperactivity, and suspiciousness. Unlike the study of Drs. Dhossche and Rubinstein, in our study alcohol use before admission was not associated with violence for male or female patients. This is probably due to different subject populations in the two studies. All of our patients were ill enough to be hospitalized, whereas many of Dhossche and Rubinstein's emergency room patients were not ill enough to be hospitalized. Degree of psychopathology in our study may have overshadowed any effect of alcohol on violence.

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Psychotherapy's Role in Psychiatry

TO THE EDITOR: Jeffrey A. Lieberman, M.D., and A. John Rush, M.D. (1), take a highly problematic stance with regard to the role of psychotherapy in psychiatry. While the authors claim that their endeavor is based on science and empiricism, no scientific (or other) explanation or data are given for their statement that psychotherapy "can and should be codified and implemented, when medically indicated, by nonmedical professionals." The authors note the absence of data that psychiatrists are more effective psychotherapists than other "less expensive" mental health professionals but do not explain why that means it is best for psychiatrists to discontinue treating patients with psychotherapy. It appears that the determining factors of the authors' strategy for psychiatry are economic and political, rather than empirical, scientific, and patient-oriented.

No one argues the value of incorporating neurobiological developments into psychiatry, but it is unclear why the authors feel it is necessary to jettison our knowledge about and approaches to the patient's mind. We know the mind is a powerful instrument for change and that psychological changes can lead to neurobiological shifts. The authors' approach sets back psychiatry a century, when there was little knowledge about psychological approaches.

Along with the absence of consideration of the mind is an apparent absence of a humanistic focus on what is best for patients. References to the "worried well" minimize the tremendous suffering that such patients frequently experience. Doidge et al. (2) found that a group of analytic patients showed high rates of anxiety and depressive disorders as well as high rates of significant trauma in their history. These patients had already received a variety of treatments that did not result in lasting benefit. The focus of Drs. Lieberman and Rush appears to be on what is best for the economy, rather than patients. The indirectly stated message is that the ship is sinking, and psychiatrists should take the seats on the lifeboats and leave many of our powerful approaches to the mind, along with the patients that benefit from them, behind.

In his editorial (3), Herbert Pardes, M.D., states the importance of psychiatrists having knowledge of what they are prescribing and the value to certain patients of having one practitioner administer both treatments. If psychiatrists stop learning about and performing psychotherapy, they will lose the ability to know when to prescribe it. In an ongoing psychotherapeutic process, psychiatrists may more readily recognize subclinical forms of syndromes that are likely to benefit

from medication. Many studies indicate that a variety of syndromes respond best to a combination of medication and psychotherapy. An individual giving both treatments could best titrate these various treatments in a given patient.

Psychiatrists also have an important role in medical student education on the humanistic side of medical care. Patients do not want assembly line medicine, and they want to feel that their doctor understands them. Physician role models are essential for medical students to gain such understanding.

Psychiatry desperately needs long-term outcome studies in general and for the psychotherapies in particular. Only then can we consider cost effectiveness on a comparative basis. Such long-term outcome studies are only currently getting underway.

We must not forget that our role as psychiatrists is to do what is best to help our patients. To ignore our patients' internal mental life and the power of unconscious wishes, aims, and conflicts—which control lives through fostering symptoms of anxiety and depression and disrupting life goals and relationships—is to deny access to powerful data for psychiatrists to use in relieving patients of their fears and inhibitions. To consign these functions to others outside the profession for the sake of economic and political expediency would be a tragic mistake.

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Drs. Lieberman and Rush Reply

TO THE EDITOR: Drs. Busch and MacKinnon write as if we proposed that psychiatrists provide no psychotherapy. Our article made no such statement, and this is not our recommendation. It is our recommendation that psychiatrists need not be the providers of time-limited, relatively easy to implement forms of therapy any more than a neurologist needs to provide the physical therapy essential to a patient's recovery from a stroke. Since selecting patients for these forms of treatment is an imperfect clinical task, as Drs. Busch and MacKinnon note, we contend that the decision to recommend such treatments, as well as an evaluation of the outcomes of such treatment, should be retained by the psychiatrist—much as neurologists make recommendations in collaboration with physical therapists to initiate and terminate physical therapy. Given the fact that we can measure outcome, it is not required that the provision of the treatment per se must reside in the same individual who determines the indications for, limitations of, and when to discontinue these therapies—whether they are successful or not.

We did recommend that the training emphasis on psychotherapies and psychodynamic theory be reduced to allow for more integration of medical, neurological, and neurobiological exposure. However, this should not be at the expense of the humanistic dimension of psychiatry, which is integral to medicine and an essential part of training. Psychiatry has assumed a leadership role among medical specialties in teaching and using the skills involved in humanistic patient care and should continue to do so.

Drs. Busch and MacKinnon's suggestion that the determinant factors of our strategy are not scientific and empirical but economic and political is inaccurate. We are simply facing reality and applying the analytical and clinical skills of physician scientists. To ignore this reality serves no good purpose, not even that of the practicing medical psychotherapists whose dedicated and skillful efforts are being devalued, and least of all the patients for whose care we as psychiatrists are charged.

Finally, we agree wholeheartedly with their recommendation for long-term outcome studies for psychiatry in general and for psychotherapies in particular.

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Peacekeeping Duty and PTSD

TO THE EDITOR: The recent article by Brett T. Litz, Ph.D., and colleagues (1) reports on an interesting role for frustrated aggression in the development of posttraumatic stress symptoms among soldiers who served in Somalia. However, readers should also be informed of the suspected role of frustration in acute psychiatric illness.

Frustrated aggression during peacekeeping has recently been suggested as a contributing factor to the high psychiatric morbidity and mortality that marked the early weeks of "Operation Uphold Democracy" in Haiti (2). That mission was initially planned as a combat invasion but was changed to a peacekeeping occupation hours before troops landed. Anecdotal reports from soldiers revealed intense frustration with the change from combat to peace enforcement. Clinical activity also suggested an early period of maladjustment for soldiers. The initial 6 weeks of deployment were marked by a high rate of major axis I disorder presentations to the mental health clinic and three suicides (3). Only one case of PTSD was diagnosed (which existed before deployment), but many soldiers were treated for adjustment and major depressive disorders. No cases of acute stress disorder were diagnosed. The prevalence of major axis I disorders dropped markedly and stabilized after 6 weeks. Frustrated aggressive drives appear to have contributed to this early period of maladjustment.

While initial suggestions for psychological support of soldiers on peacekeeping missions have been made (4), further research into the role of frustrated aggression in peacekeeping and improved methods of supporting the men and women involved in these operations are clearly warranted. This information may also benefit police and other areas of law enforcement.

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Dr. Litz and Colleagues Reply

TO THE EDITOR: We appreciate Dr. Hall's comments about the similarities and differences between the research he and his colleagues have conducted on U.S. military personnel who took part in the peacekeeping mission to Haiti and our own research that examined the predictors of PTSD severity in Somalia veterans. It is not surprising to us that frustration with shifting priorities and rules was a source of stress for soldiers who were deployed to Haiti. However, it is surprising that there were few lingering problems after the mission that were associated with frustration. In more recent analyses of our Somalia veteran database, we found that global frustration with the negative aspects of peacekeeping (e.g., frustration with Somalis, being restrained from aggressive action when threatened, and reaction to changing rules of engagement) contributed to the risk of a wide variety of subsequent psychopathology (unpublished 1997 study of S.M. Orsillo), which partly supports Dr. Hall's findings. We also found that pressure to uphold restraint is not implicated in the development of PTSD when frustration with being restrained from aggressive action is analyzed separately from other frustrating aspects of peacekeeping (1). Rather, the specific form of frustration that is secondary to such restraint is a unique psychological outcome in peacekeepers in its own right. Our findings and those of Dr. Hall and his colleagues suggest that clinicians and researchers need to pay special attention to mission-specific factors that lead to frustration in peacekeepers, the actual experience of frustration in soldiers, and both the acute and long-term mental health consequences of such experiences.

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Confidentiality Conundrum

TO THE EDITOR: Howard B. Roback, Ph.D., and colleagues (1) provide a fine review of the confidentiality problems in group therapy with substance-dependent physicians but appear to misunderstand some of the relevant law. The Code of Federal Regulations protects confidentiality for federally assisted alcohol and drug treatment, even if the assistance is indirect (42 CFR 2.63–2.65). The federal Health Care Quality Improvement Act provides immunity from liability for disclosure to professional review bodies, not liability for failure to disclose. It thereby encourages but does not require such reporting of physicians. Since there is a legal requirement to maintain confidentiality as well as immunity for reporting to professional review bodies, therapists are legally able to make either choice. Consequently, in most instances they are in the advantageous position of being permitted to base such decisions upon clinical and ethical as opposed to legal factors.

State law governs if federal protection does not apply. In case of discrepancy, federal law trumps state law. Even without other legal protection, all subpoenas can be challenged before disclosure. Claims of lack of relevance or of psychotherapist-patient privilege might be sustained by a court. Additionally, it is unclear whether the subpoenas to testify about group members received by 27% of surveyed therapists included orders from a court of competent jurisdiction, as is legally required in federally assisted programs.

The authors' proposed legislation to clarify that confidentiality supersedes other regulations is unnecessary and potentially counterproductive. In our current political climate such efforts more likely would result in changes that favor disclosure and not confidentiality.

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ROBERT WEINSTOCK, M.D.
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H. WESTLEY CLARK, M.D., J.D., M.P.H.
San Francisco, Calif.

Dr. Moore and Colleagues Reply

TO THE EDITOR: Drs. Weinstock and Clark claim that we misunderstand some of the relevant law. The Code of Federal Regulations explains the manner in which courts should apply the Comprehensive Alcohol and Alcoholism Prevention, Treatment, and Rehabilitation Act and the Drug Abuse Office and Treatment Act. Our article explained in detail the extent to which these acts and the Code of Federal Regulations protect the confidentiality of substance abuse treatment records. Weinstock and Clark apparently believe that we do not understand that the Code of Federal Regulations applies to treatment programs that receive federal assistance "even if the assistance is indirect." In fact, however, we did state that the statutes and regulations apply to programs that "receive either direct or indirect federal support" (p. 1251).

Our article described the extent to which the Health Care Quality Improvement Act provides immunity from damages when a person reports the impairment of a physician to a professional review body. Contrary to the allegation of Weinstock and Clark, our article never states that the Health Care Quality Improvement Act *requires* violations of confidence. Rather, our article emphasized that this act "encourages" violations of confidence.

Weinstock and Clark fail to note our article's demonstration that substance-dependent physicians are likely not protected by the Comprehensive Alcohol and Alcoholism Prevention, Treatment, and Rehabilitation Act and the Drug Abuse Office and Treatment Act because those statutes are very probably trumped by the Health Care Quality Improvement Act. Therefore, when the patient is an impaired physician, Health Care Quality Improvement Act permits the enforcement of *state* laws that *require* the treating physician to violate confidentiality by reporting the impaired physician to the state medical board. Our article indicates that 15 states have such laws.

We stated that in general, federal law trumps state law. Weinstock and Clark are incorrect in claiming that "in case of discrepancy, federal law trumps state law." In fact, federal law does not always trump state law. When a state law differs from federal law, but does not directly contravene federal law, and when the state law provides greater protection of civil rights, then the state law takes precedence.

Weinstock and Clark claim that courts *might* sustain objections to subpoenas for disclosure when the objections are based on assertions of lack of relevance or of psychotherapist-patient privilege. However, inspection of the "Notes of Decisions" rendered pursuant to the Comprehensive Alcohol and Alcoholism Prevention, Treatment, and Rehabilitation Act, the Drug Abuse Office and Treatment Act, and the Code of Federal Regulations shows that courts have issued many subpoenas that forced providers to violate confidentiality by revealing substance abuse treatment records (1).

Finally, Weinstock and Clark provide no evidence to support their allegation that our proposed legislation would further erode confidentiality. In fact, the language of our proposed legislation provides confidentiality protections that exceed the protections stipulated by current laws and by the regulations cited by Weinstock and Clark.

We hope that these comments adequately address Weinstock and Clark's concerns.

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RANDALL F. MOORE, M.D., J.D.
HOWARD B. ROBACK, PH.D.
GLORIA J. WATERHOUSE, PH.D.
PETER R. MARTIN, M.D.
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Genetic Risk Factors for Bipolar Disorder

TO THE EDITOR: I was pleased to see the article by Blanca Gutiérrez, B.Sc., and colleagues (1) and the continued interest in the finding of a catechol *O*-methyltransferase (COMT) activity abnormality in patients with bipolar disorder, which my colleagues and I originally reported (2). I have concerns about Gutiérrez et al.'s conclusion that there is no association between the COMT gene and risk factors for bipolar disorder. Although the gene itself may not be different, its expression, perhaps controlled by other genes, could account for our finding. The role could still be large but mediated by a regulator of COMT gene activity rather than by the gene itself.

It is gratifying to find that work from a quarter century ago has become such a part of the fabric of the history of biological psychiatry that the original authors' names have been forgotten.

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CAL K. COHN, M.D.
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Drs. Gutiérrez and Fañanás Reply

TO THE EDITOR: One of the motives that led us to carry out the molecular analysis of the COMT gene in patients with bipolar disorder was the results from several studies that reported the altered function of this enzyme in patients with affective disorder. We read Cohn et al.'s 1970 study with great interest as well as that of other researchers who also reported lower activity of this enzyme in patients with affective disorder (1).

The growing demand to make articles as brief and to the point as possible precludes the use of exhaustive references, for which reason we unfortunately omitted to mention Cohn et al's study. We opted to refer to recent works that provided a general retrospective, within which the initial study of Cohn et al. is, of course, reported.

On dealing with the COMT enzyme, our hypothesis was novel in relation to the aforementioned studies in that we aimed to recognize that the differences in the enzyme's function for patients with bipolar disorder were genetically determined. Indeed, our study was focused mainly on genetic variability rather than physiological features.

The COMT protein structure seems to be controlled by two common genetic variants described in the general population, each of which gives rise to enzymatic forms with different levels of activity. Our study aimed to recognize the distribution of this genetic variability in an accurately designed association study in which no particular distribution of enzymatic forms differentiated patients with bipolar disorder and healthy comparison subjects. Our results suggest that the *analyzed* genetic variation in the COMT gene does not play a direct role in the origin of this affective disorder. We do, however, agree with Dr. Cohn as to the possibility that other regulating genes may act upon the expression of the COMT gene. We do not reject this possibility at any moment in our article. Indeed, we leave the way open for further genetic studies on the basis of this hypothesis according to the new genetic variability described in the promotor region of this gene.

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BLANCA GUTIÉRREZ, B.SC.
LOURDES FAÑANÁS, PH.D., M.D.
Barcelona, Spain

Borderline Personality Disorder and Transitional Objects

TO THE EDITOR: I read with great interest the article by William Cardasis, M.D., and colleagues (1) on transitional objects and borderline personality disorder. It confirms the unscientific and casual observations of myself and other members of the inpatient treatment teams with whom I have had the privilege to work since my residency. It has become so common for us to see patients who are admitted with either blankets or stuffed animals and whose axis II diagnosis is later confirmed that we have come to refer to the presence of these items as a "positive bear sign." We have further differentiated new animals (often brought to the patients as gifts) from those brought from home. The former usually suggest the presence of mild borderline traits, while the latter often correlate with a more severe pathology.

These findings (often snickered over in morning report) are, as Cardasis et al. purport, often important observational clues that aid in the diagnosis and treatment of these difficult and challenging patients.

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LAUREN D. LAPORTA, M.D.
Paramus, N.J.

Misrepresentational Review

TO THE EDITOR: Any book deserves some negative reviews. However, it is one thing to expose weak points and quite another to grossly misrepresent a book. The book review by Lauri R. Robertson, Ph.D., M.D. (1), of my book *Cultures of Healing* does the latter.

To hear Dr. Robertson tell it, I am a "relentlessly bitter critic" of mental health care. To the contrary, I present a positive way of understanding mental health care that, I argue at length, makes more sense than the conventional public and self-images of the profession. Dr. Robertson neither presents this alternative image (or acknowledges its existence) nor addresses any of the arguments I evince for it. She may not like my view, but by any reasonable measure, failing to mention the main point of a book is a gross distortion.

She says that I not only throw the baby out with the bathwater but appear to "deny there ever was a baby at all." She does not specify the "baby" that she is talking about, but if she means that I deny the existence of care that helps people, she is absolutely wrong. Indeed, the gravamen of my book, as I state clearly at many places, is to find a way of understanding mental health care, since it is important and does good things.

Dr. Robertson would have readers think of my book as "postmodern relativism." Neither postmodernists nor relativists think, as I do, that there is truth and that science is a crucial avenue to it. Neither bemoan the shaky scientific foundation of mental health care, as I do, since both movements deny to science epistemic advantage. I criticize at many places in the book those mental health types who seize on postmodern ideas to justify their lack of sound knowledge. I argue that all mental health professionals need better scientific education in a wider range of sciences—hardly a postmodern or relativist notion.

Dr. Robertson ascribes to me many things that I simply do not say. For instance, nowhere do I address Freud's textual inconsistencies (I do not consider them important). I do not (to my recollection) anywhere mention "brainwashing." I do not criticize "imposition of rationality on emotion" but rather cognitive therapists' extremely unscientific and indefensible idea of how thinking works.

Dr. Robertson concludes that my book contains too much "*ad hominem* gripe." I would challenge her to produce even one ad hominem passage in my book. An ad hominem fallacy argues that the bad character of someone who holds a view is a testament to the falsity of the view. I certainly do argue that it is a vice to claim as true views that one has good reason to believe are false and to profit from this lie. This position, however, is the opposite of ad hominem, since it argues that the falsity of the ideas is a testament to the faultiness of the character.

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ROBERT T. FANCHER, PH.D.
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Italian Psychiatric Reform

TO THE EDITOR: Angelo Fioritti, M.D., and colleagues (1) evaluated the reform of the psychiatric care system in the region of Emilia-Romagna between 1978 and 1994. Their data illustrate the shift from hospital-based to a community-based mental health network. However, some of their results raise further questions. One would assume that the average length of stay during this shift would go down; however, admission rates went up (shorter, but more frequent stays). In view of this, the authors' statements that "the overall rate of inpatient admissions remained stable" seems to be puzzling, especially during the time when several mental hospitals were closed and the number of patients who resided in mental hospitals substantially declined. Fioritti et al. also state that the Italian community psychiatric system is somewhat less costly than the previous system. It would be interesting to know if the number of practicing psychiatrists increased, decreased, or remained stable during this reform. It would also be interesting to know if the needs of the mentally ill in Emilia-Romagna were met after the reform, or if Emilia-Romagna faces increasing numbers of homeless mentally ill and increasing numbers of mentally ill who are unable to get adequate mental health services in overcrowded community outpatient clinics.

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RICHARD BALON, M.D.
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TO THE EDITOR: The article by Fioritti and colleagues reported an interesting evaluation of the psychiatric services in Italy after the 1978 Reform ("Law 180"). We agree with the authors' suggestion that the shift from a hospital-based to a community-based psychiatric system of care seems feasible and less expensive than the hospital system. However, it should be noted that lack of central coordination determined an inadequate implementation of psychiatric services at regional and local levels in the north, central, and southern regions of Italy (1, 2). Although the case of Emilia-Romagna described by Fioritti and colleagues is a good example of the implementation of community psychiatry in the north-central region of Italy and that is also currently performed in districts such as Melegnano, South Verona, Arezzo, and Perugia, it should not be forgotten that a recent survey of the Italian Institute of Social Medicine showed that 17,000 patients are still living in 76 mental hospitals.

Nineteen years after the psychiatric reform, the new 1997–1999 mental health reform should ameliorate some of the drawbacks of Law 180 without changing the basic principles of the Reform, such as the closing of mental hospitals and the focus on psychiatric services in the community. The mental

health project will review community care services for the 500,000 Italians with mental health problems and will be aimed at correcting dysfunction and unevenness on regional and local bases by introducing sanctions on local authorities for noncompliance. The closing of all mental hospitals will be completed in the next 3 years, and psychiatric care will be coordinated by a department of mental health. This department will be financially autonomous and will be composed of psychiatric services such as mental hygiene centers, community care facilities, psychiatric wards within the general hospital, and emergency services. Practical and structural aspects of mental health services will be reviewed, and the optimal demographic ratio for each service will be provided. In conclusion, the 1997–1999 project will allow a better evaluation of the Italian community-based system of care, which could represent a useful model for psychiatric care in a broad international scenario.

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ANDREA PERGAMI, M.D.
MARA GONEVI
ANTONIO GUERRINI, M.D.
Milan, Italy

Dr. Fioritti and Colleagues Reply

TO THE EDITOR: The Italian Psychiatric Reform of 1978 established a "front door" policy in deinstitutionalization that prohibited new admissions to psychiatric hospitals and set up a community-based system of care with a limited number of beds located in general hospitals. Dr. Balon's concerns are legitimate, as problems in the availability of hospital beds, staffing of alternative services, and neglect of the mentally ill have been claimed in many parts of Italy and have led to public discontent. Dr. Pergami and associates thoughtfully underline how these problems require a more careful and thorough administrative action, hopefully at a national level.

In Emilia-Romagna, after all new admissions to psychiatric hospitals were prohibited, patients were placed in general hospital psychiatric wards, university clinics, private clinics, and alternative residential facilities. Throughout the period 1979–1992, the number of beds for acute, subacute, and long-term patients outside the mental hospital increased from 23.72 to 27.91 per 100,000 inhabitants. In the same period the average length of stay in all public and private wards for acute and subacute patients passed from 31.40 days to 25.59 days and was much lower in the public sector (16.21 days in 1992) than in the private (37.13 days in 1992). From 1979 to 1992, the rate of admission to all private and public facilities increased from 212.2 to 286.4 per 100,000 inhabitants (1). These changes can be explained by new young adult psychotic patients entering the system and the gradual entrance into the system of patients discharged from the psychiatric hospitals.

The graduality of this process can, at least in part, explain the virtual absence of homelessness among the mentally ill in most parts of Italy. Better general social background conditions also account for this phenomenon in Emilia-Romagna: a tradition of community mutual support and of careful social welfare policies, as well as an economic development that was based on cooperatives and small scale industries (2), were all of remarkable help in the process of integration and support of the patients discharged by the mental hospitals.

Finally, we can provide only general estimates of the total number of psychiatrists who work in the private sector, since no certification by a professional board is required. Their number has certainly increased since 1974, when the first specialization school in psychiatry was established in Bologna (all specialists previously licensed were neuropsychiatrists). On the other hand, in 1992, 457 psychiatrists, 82 psychologists, 2,085 nurses, and 412 social workers were operating in the public mental health services of the region Emilia-Romagna. The implementation of cost-containment policies is likely to reduce dramatically the number of psychiatrists working in the public sector in the next decade, which allows for some of their current clinical tasks to be incorporated into other professional profiles.

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