Focal Segmental Glomerulosclerosis and Lithium Treatment

Lithium carbonate is an effective, commonly used treatment for manic-depressive illness. The drug is known to produce a number of renal side effects, including impaired urinary acidification, polyuria, and impaired renal concentrating ability, which have been summarized elsewhere (1, 2). Lithium is a rare cause of nephrotic syndrome, a complication that reduces renal lithium excretion and precipitates lithium toxicity (1). We describe here a case in which the clinical features of lithium toxicity appeared after nephrotic syndrome, due to focal segmental glomerulosclerosis, developed in a patient treated with lithium. We report the case of a 59-year-old woman with bipolar affective disorder.

For 3 weeks Ms. A had been experiencing progressive lethargy and diarrhea. She was finally admitted to the hospital because of anemia and leukopenia of unknown origin. She had bipolar disorder, which had been well controlled for 3 years with a regimen of 2.5 mg/day of haloperidol, 50 mg/day of doxepin, 2 mg/day of biperiden, and 800 mg/day of lithium carbonate. There had been no recent change in her medication regimen. Three days after admission Ms. A developed cerebellar ataxia, dysarthria, and bilateral pleural effusions.

The following laboratory values were noted: 6.9 g/dl of hemoglobin, 2.4 $\times$ 10^9/liter of leukocytes, 52 mg/dl of urea, and 268 mg/dl of cholesterol. Both her electrolyte levels and creatinine values were normal. Ms. A's serum lithium level was markedly elevated at 1.9 mmol/liter (therapeutic range=0.6–1.2 mmol/liter). Her serum albumin level was 18 g/liter (normal range=36–47 g/liter). Her renal function had been normal during the previous 3 years of treatment. Her 24-hour urine protein excretion was 12.23 g (normal range: <0.15 g). Serum electrophoresis showed an increase in α_2 globulin, with no paraprotein detected. Antinuclear antibodies were absent. A renal biopsy analysis revealed focal segmental glomerulosclerosis.

Ms. A's lithium therapy was discontinued, and her serum lithium level fell to 1.1 mmol/liter after only 2 days. Six weeks later her edema and effusions had completely disappeared, her serum albumin level had risen to 29 g/liter, and her 24-hour urine protein excretion had fallen to 3.19 g. After 3 months both her red and white blood cell counts had returned to normal, and her renal function had improved markedly, with only mild proteinuria (0.3 g/24 hours). The reason for her anemia and leukopenia remained unclear, despite a bone marrow biopsy.

This patient presented with the symptoms of lithium toxicity and a coexistent nephrotic syndrome due to histologically proven focal segmental glomerulosclerosis. Her nephrotic syndrome was most likely a renal complication of lithium therapy, since no other cause was found (1, 3). We discovered only three cases of focal segmental glomerulosclerosis in association with lithium therapy in the literature (1). However, physicians should be aware of this rare but very important renal complication of lithium therapy, which indicates the necessity of a careful evaluation of proteinuria in patients treated with lithium.

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Amphetamine Misuse and Social Phobia

We report a case of social phobia after amphetamine abuse, which may help explain the neurobiology of this disorder.

Ms. A, a 26-year-old woman, was seen after 2 months of flushing, sweating, palpitations, and shortness of breath, which occurred in a range of social situations. The first episode happened in a staff canteen, when she became aware of her colleagues staring at her and felt embarrassed. At her assessment she had given up work and had started avoiding various activities for fear of reexperiencing similar feelings. She had no past psychiatric history, and before these attacks she had been confident and extroverted. She had been taking 1.5 g of street amphetamine orally almost daily for 6 years.

Initially, she felt good while taking the drug, but she currently used it to help her "get going." There was no temporal relationship between her anxiety symptoms and her amphetamine ingestion; the attacks occurred specifically in social situations. She showed no symptoms of affective disturbance or psychotic symptoms. A diagnosis of generalized social phobia was made.

The neurobiology of social phobia is poorly understood; biochemical abnormalities of the γ-aminobutyric acid (GABA), serotonergic, and adrenergic systems have been suggested. Furthermore, social phobia is the only anxiety disorder for which there is evidence of dopaminergic dysfunction (1).

High rates of social phobia have been reported in patients with Parkinson's disease (2), and social anxiety can develop after treatment with dopamine antagonists (3). Bupropion, a dopamine enhancer, may be effective in the treatment of social anxiety (4). A study with single photon emission computed tomography found low dopamine reuptake site density in the striatum of patients with social phobia (5).

Animal studies show that chronic administration of amphetamines causes striatal depletion of dopamine, and limited human data indicate a similar loss of dopamine in the striatum (6). We suggest that our case showed that chronic amphetamine misuse causes social phobia as a result of dopamine depletion, evidence of which was the patient's anergia. This case strengthens the hypothesis that dopamine is involved in the pathophysiology of this disorder.

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Ziprasidone Overdose

Ziprasidone is a combined 5-HT (serotonin) and dopamine receptor antagonist (1, p. 101) that has been administered in controlled trials to over 3,000 patients with schizophrenia and schizoaffective disorder (2). Ziprasidone is a unique molecule with a neuropharmacological profile distinct from those of both the standard and newer antipsychotics (2). It possesses a high in vitro 5-HT_{2A}/dopamine D_2 receptor affinity ratio (1) and is also an agonist at the 5-HT_{1A} and 5-HT_{1D} receptors. It is a 5-HT and noradrenaline reuptake inhibitor, which suggests that it may have use in treating psychotic depression (2). Given the patient population for which antipsychotic medications are intended, the potential for overdose is high. We report the details of a patient overdose. The case has been reported to the U.S. Food and Drug Administration.

Mr. A, a 50-year-old man with a well-established diagnosis of chronic paranoid schizophrenia, was admitted to a hospital's emergency department 4.5 hours after an overdose of 3120 mg of ziprasidone (52 60-mg tablets of ziprasidone and 26 placebo tablets). He said he had not taken any other prescribed or illicit drugs or drunk any alcohol; he had not vomited. He was conscious and cooperative.

Mr. A was connected to a cardiac monitor and a pulse oximeter. His Glasgow Coma Scale (3) score was 15. His blood pressure at admission was 200/95 mm Hg; it decreased to 160/80 mm Hg within 1.5 hours; then it remained in the normal range. His axillary temperature was 36.5°C. His pulse ranged from 70 to 90 bpm. His Sao_2 was between 96% and 99% with room air. He was a little drowsy, and his speech was slightly slurred.

An ECG performed on Mr. A showed some minimal QT prolongation (QT/QTc 430/490 msec) and nonspecific flattening of the T wave. He had no arrhythmias. Four ECGs per hour were performed. Blood was taken for a CBC, to check liver function, and to test electrolyte, magnesium, and acetaminophen levels. The results were within normal limits. An intravenous infusion with normal saline solution was begun at a rate of 8 liters per hour. An intravenous infusion of 10 mg of metoclopramide was given, along with 50 mg of oral charcoal and 150 ml of sorbitol.

Mr. A's first two postoverdose ECGs showed minimal QT prolongation and nonspecific change in the morphology of the T wave compared to his pretreatment ECG. This change was maximal at 6 hours postoverdose, which would correlate with the peak plasma level of ziprasidone (4). His ECG readings then returned to normal.

This is the first reported overdose of ziprasidone, to our knowledge. The quantity of the medication ingested appears to be accurate and is substantial, representing a 26 days' supply of ziprasidone at the upper end of the recommended dose range. Charcoal and sorbitol were administered 5 hours postoverdose. A substantial quantity of the drug would have been absorbed because peak plasma levels occur 4–6 hours after oral administration (4).

Throughout the patient's 13-hour stay in the emergency department, his central nervous system functioned normally, with no extrapyramidal side effects. He was oriented and showed no evidence of delirium. No arrhythmias were noted during his cardiac monitoring or on his ECGs. There was only minimal and transient QT change and no hemodynamic abnormality. These observations suggest relative cardiac safety.

It is premature to draw any general conclusions from this single incident of an overdose of ziprasidone, but our findings suggest relative safety. Patients should have general supportive treatment, as described here, and ECG monitoring.

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Euphoric Mania and Rapid Transcranial Magnetic Stimulation

Grisaru et al. (1) reported antimanic effects from rapid transcranial magnetic stimulation of the right prefrontal cortex. In a 14-day double-blind trial, right and left prefrontal stimulations were compared in patients receiving repeated rapid transcranial magnetic stimulation in addition to antimanic drug therapy. We report here on a patient with euphoric mania that was refractory to treatment with sulthiame (2) who experienced marked improvement during monotherapy with right prefrontal rapid transcranial magnetic stimulation.

Ms. A was a 59-year-old woman with a diagnosis of bipolar I disorder. After her first and only depressive episode, which developed postpartum at age 35, she experienced at least four severe manic episodes. Eight weeks before hospital admission she developed euphoric mania despite lithium treatment. Lithium treatment was discontinued, and a trial with the antiepileptic drug sulthiame was initiated—unfortunately, with no effect after 3 weeks of treatment. Sulthiame treatment was tapered off, and mono-

therapy with rapid transcranial magnetic stimulation was begun. Right prefrontal stimulation was performed, as suggested by Grisaru et al. (1): 20 trains per session, a frequency of 20 Hz for 2 seconds per train, and an intertrain interval of 1 minute.

Ms. A was given five consecutive sessions during weeks 1 and 2 and three sessions during weeks 3 and 4. Her range of motor threshold was 66%–76%. Her scores on the Bech-Rafaelsen Mania Scale (3) slowly but continuously fell (28 on day 0, 24 on day 7, 15 on day 14, 10 on day 21, and 8 on day 28). Her sleep disturbance and thought disorder seemed to respond particularly well to rapid transcranial magnetic stimulation. Ms. A was dismissed from the hospital ward. Prophylactic treatment with the third-generation, putative mood-stabilizing anticonvulsant topiramate (4) was initiated for Ms. A for obesity.

Although new strategies in the treatment of acute mania are clearly needed (5), Grisaru et al.'s observation (1) that add-on treatment with right prefrontal rapid transcranial magnetic stimulation is effective in treating mania (showing laterality opposite to the proposed effect of rapid transcranial magnetic stimulation in depression) deserves particular interest. In this case, repeated monotherapy with right prefrontal rapid transcranial magnetic stimulation was effective and well tolerated in a patient with euphoric mania. Further systematic studies are needed.

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Antidepressant-Induced Sexual Dysfunction and Ginkgo Biloba

Sexual dysfunction is a common consequence of treatment with selective serotonin reuptake inhibitor (SSRI) antidepressants (1, 2). Antidote strategies are often used to lessen the sexual difficulty associated with these agents. A variety of medications are reported to reverse SSRI-induced sexual dysfunction. These include cyproheptadine (1, 3, 4), yohimbine (1, 5, 6), amantadine (1, 7, 8), stimulants (9), buspirone (10), bupropion (11, 12), and sildenafil (13–15). In addition, ginkgo biloba has been used to treat antidepressant-induced sexual dysfunction (16). Although the study of gingko biloba had numerous limitations, including problematic statistical analyses (17, 18), it raised hope that ginkgo biloba could be helpful in managing SSRI-induced sexual dysfunction. We report here our experience with use of ginkgo biloba in reversing SSRI-induced sexual dysfunction.

A 1-month trial of ginkgo biloba was prescribed for 22 consecutive patients (nine men and 13 women) seen in a private psychiatric office who complained of SSRI-induced sexual dysfunction. The gingko biloba was purchased at a local health store, and the prescribed dose was 300 mg t.i.d. Oral consent was obtained; written consent was not obtained because the results of this study were not initially for publication. SSRI-induced sexual dysfunction was defined as a new impairment in sexual desire, arousal, or orgasm appearing within 4 weeks of beginning treatment with an SSRI. Ratings of improvement were based on clinical interviews by the patient's treating psychiatrist.

The 22 patients enrolled in this study described 40 sexual complaints. Three (13.6%) of the 22 patients described at least partial improvement in sexual function while taking ginkgo biloba. None of the nine men and three (23.1%) of the 13 women described improved sexual response. In addition, four (10.0%) of the 40 complaints improved with ginkgo biloba treatment. When analyzed by sex, none of the nine complaints of the men and four (12.9%) of the 31 complaints of the women improved. No side effects of ginkgo biloba treatment were reported.

This brief study demonstrates little reversal of SSRI-induced sexual dysfunction from treatment with ginkgo biloba. There are several obvious limitations to this study, including the lack of a placebo control, a potential variation in product potency, a selection bias, and the lack of a standardized outcome measure. Ginkgo biloba may still hold hope for some patients, perhaps with the selection of a particular brand of product, the use of a higher dose, or use for a longer duration. Nevertheless, this study does not replicate prior findings supporting the use of ginkgo biloba for the treatment of SSRI-induced sexual dysfunction.

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Mnemonic Screening Device for Generalized Anxiety Disorder

In DSM-IV, generalized anxiety disorder is characterized by the essential features of excessive anxiety and worry that an individual finds difficult to control. This cognitive symptom of worry must be about multiple events or activities that are not confined to the features of another axis I disorder. In addition, three or more associated symptom criteria must be present, with at least some of them present more days than not for the past 6 months. The associated symptom criteria fall into two categories, each of which contains three symptoms: 1) motor tension—muscle tension, restlessness, and fatigue and 2) hypervigilance—irritability, difficulty falling asleep, and difficulty concentrating.

Generalized anxiety disorder is difficult to distinguish from other mood and anxiety disorders, with which it shares many symptoms, and it is highly comorbid (1). To help differentiate generalized anxiety disorder from mood disorders, DSM-IV stipulates that the diagnosis of generalized anxiety disorder cannot be assigned if its features occur exclusively during a mood disorder. In their article on the empirical basis of generalized anxiety disorder, Brown et al. (2) cited research examining the ability of the item "Do you worry excessively about minor matters?" from the Anxiety Disorders Interview Schedule—Revised (3) to discriminate patients with generalized anxiety disorder from patients with other anxiety disorders. They found that unpublished 1991 research by DiNardo (which is supported by unpublished 1993 research by Borkovec) demonstrates that the positive predictive power of the question is 0.36 (the probability of a generalized anxiety disorder diagnosis, given an affirmative response) and the negative predictive power is 0.94 (the probability of not having a generalized anxiety disorder diagnosis, given a negative response). These findings indicate that, although an affirmative response to the question about excessive worry over minor matters cannot confirm a diagnosis of generalized anxiety disorder, a negative response can rule out generalized anxiety disorder with confidence (2, p. 1277).

To help physicians screen for generalized anxiety disorder, I developed the following mnemonic device: "Does Mr. Fisc worry excessively about minor matters?" Initially, one simply substitutes "you" for "Mr. Fisc" in the mnemonic device and asks, "Do you worry excessively about minor matters?" If endorsed by the patient, one uses the remainder of the mnemonic "Mr. Fisc" to determine if three or more of the associated motor tension and hypervigilance criteria are present. Each letter of "Mr. Fisc" stands for one of the associated symptoms, as follows: M=muscle tension, R=restlessness, F=fatigue, I=irritability, S=sleep (difficulty falling asleep), C=concentration (difficulty concentrating). I hope others find this to be a user-friendly tool that facilitates improved recognition of this disorder.

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Racial Differences in Treatment of Adolescents With Bipolar Disorder

Previous studies have demonstrated that patient ethnicity influences clinical decision making in psychiatry. Specifically, studies report that African American psychiatric patients are more likely to be treated with "as needed" medication and to be placed in seclusion or restraints, and they receive more antipsychotic medication and at higher doses than similar Caucasian patients (1, 2). To our knowledge, there have been no studies examining the relationship between race and the psychopharmacological treatment of adolescents with bipolar disorder. The aim of our study was to compare the medications received by Caucasian and African American adolescents hospitalized for bipolar disorder.

We retrospectively reviewed the hospital records of all adolescents with a discharge diagnosis of bipolar disorder who were hospitalized at Cincinnati Children's Hospital Medical Center's Adolescent Psychiatry Unit between July 1995 and June 1998 (N=74) for demographic and clinical variables. All of the psychiatrists treating children and adolescents were Cau-

casian. There were no differences between the African American (N=14) and Caucasian (N=60) adolescents diagnosed with bipolar disorder in age, sex, co-occurring diagnoses (including substance use disorders, conduct disorder, oppositional defiant disorder, and attention deficit hyperactivity disorder), length of hospitalization, treatment with lithium, treatment with sodium divalproex, number of episodes of seclusion or restraint, or number of as-needed medications received.

All of the adolescents received at least one mood stabilizer. The groups also did not differ in reported psychotic symptoms: 14% (two of 14) of the African American and 18% (11 of 60) of the Caucasian adolescents were clinically diagnosed with hallucinations, delusions, or thought disorder. Nonetheless, African American adolescents with bipolar disorder were nearly twice as likely to receive treatment with an antipsychotic as were Caucasians (86%, N=12, and 45%, N=27, respectively) (χ^2=7.5, df=1, p=0.006).

The results of this preliminary study suggest that there are ethnic differences in the pharmacological treatment of hospitalized adolescents with a clinical diagnosis of bipolar disorder. Lewis and colleagues (3) reported that African American adolescents are more likely to be incarcerated, rather than hospitalized, than similar Caucasian adolescents, which suggests that African American patients are perceived by Caucasian clinicians as more threatening and disruptive than similar Caucasian adolescents. Perhaps this perception may explain the racial differences in the use of antipsychotics in this group. The differences in treatment may also be secondary to actual racial differences in the rates of psychotic spectrum symptoms in adolescents with bipolar disorder. Whaley (4) reported that mild forms of paranoia are more prominent in African Americans than Caucasians, which suggests that differences in the diagnosis and treatment of African Americans and Caucasians may be linked to the misinterpretation of African American, culturally based paranoia as a psychotic symptom.

Our findings may be confounded by variables not controlled in the present study, including racial differences in the clinical diagnosis of bipolar disorder, the rates of hospitalization, and socioeconomic status. Despite these limitations, our results suggest that further systematic investigations of the effects of race on the psychiatric diagnosis and treatment of adolescents, using structured diagnostic interviews to evaluate the accuracy of clinical diagnosis, are warranted.

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Treatments to Change Sexual Orientation

The *Journal* recently published a "Position Statement on Psychiatric Treatment and Sexual Orientation" that had been approved by the Board of Trustees of the American Psychiatric Association (1). That statement correctly cautions mental health professionals that there is little scientific evidence to support the efficacy of treatments designed to change sexual orientation. That statement was intended to address the matter of homosexuality and to take a clear stand "against discrimination, prejudice, and unethical treatment…, including discrimination on the basis of sexual orientation" (p. 1131).

The psychiatric profession still correctly considers pedophilia to be a mental disorder. However, like heterosexuality and homosexuality (orientations that differ from one another on the basis of differences in sexual attraction), pedophilia, too, can be thought of as a sexual orientation that is different from others on the basis of age of attraction. As with other sexual orientations, irrespective of the relative contributions of genetics and environment, maturing individuals discover the nature of their own attractions; such attractions are not the consequence of a volitional decision. Historically, untold numbers of human beings have been both demonized and vilified simply because their sexual makeups differ from the norm.

In the case of pedophilia, society must insist, for good reason, that persons who are sexually attracted to children are forbidden from acting on these attractions. As with alcoholism, such persons need to have access to effective treatments that can enable them to successfully resist succumbing to unacceptable temptations. However, just as has been the case historically with homosexuality, society is currently addressing the matter of pedophilia with a balance that is far more heavily weighted on the side of criminal justice solutions than on the side of mental health solutions.

Individuals whose sexual orientation is directed toward children manifest the same range of personality, temperamental, and character traits as individuals whose sexual orientation is directed toward adults. A recent *Journal* article (2) documented that the vast majority of individuals with pedophilia showed no evidence of either antisocial or narcissistic personality disorder. It may be no easier for a person with pedophilia to change his or her sexual orientation than it is for a homosexual or heterosexual individual to do so.

Without condoning sexual misconduct (and without equating the consequences of acting on an attraction toward children with the consequences of acting on attractions toward adults), the American Psychiatric Association should develop a contemporary policy statement regarding pedophilia. It should encourage nondiscriminatory and objective public policies and professional education, increased availability and access to appropriate psychiatric care, and the immediate establishment of an intensive research effort to more effectively address this important societal and mental health issue.

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Possible Interaction of Tramadol and Antidepressants

Clinicians wishing to follow the recommendations of Toby D. Goldsmith, M.D., et al. (1) in using tramadol hydrochloride to achieve rapid remission of obsessive-compulsive disorder (OCD) should be advised that drug-drug interactions may result if concomitant antidepressant treatment is used. In the case presented in the letter by Dr. Goldsmith and colleagues, a patient with OCD was receiving fluoxetine, 20–40 mg/day, in addition to tramadol in the course of her treatment. The authors mention the possibility of a serotonin syndrome developing but do not discuss the risk for seizure, which may result from P_{450} inhibition induced by the selective serotonin reuptake inhibitor (SSRI), as well as competitive binding to the substrate. This is a documented warning in the *Physicians' Desk Reference,* and patients receiving both types of medication should be fully informed of this possibility to minimize medical-legal risk.

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We thank Victor I. Reus, M.D., and Lee Rawitscher, M.D., for pointing out the risk of seizures due to drug-drug interactions when tramadol hydrochloride is used. Although it was not specifically stated in our original letter, our patient was appropriately informed of the risk of seizures as well as serotonergic syndrome when fluoxetine was added to her tramadol treatment, which had rapidly alleviated her OCD symptoms.

The risk of seizures associated with tramadol has been reported. As noted by Drs. Reus and Rawitscher, seizures have been reported when SSRIs (as well as tricyclic antidepressants and other tricyclic compounds, monoamine oxidase inhibitors, opioids, neuroleptics, and other drugs that lower the seizure threshold) are combined with tramadol (1). A nested case-control study by Jick et al. (2) investigated the risk of idiopathic seizures in patients taking tramadol and found that patients receiving tramadol alone did not have an increased risk of seizures. Seizures are also increased in those at otherwise greater risk of convulsive activity (e.g., those with epilepsy, head trauma, or metabolic disorders and those taking other substances that lower the seizure threshold) (1).

The importance of treating postpartum psychiatric illnesses cannot be understated, given that mothers are at a greater risk of not bonding with their babies or having normal relationships with their infants while suffering from such illnesses (3). Patients with OCD taking SSRIs often have to wait 8–10 weeks for symptomatic relief and many have an inadequate or partial response (4, 5). In a case series from Sichel et al. (6), OCD symptoms began an average of 2.2 weeks postpartum. Since early diagnosis and rapid treatment of OCD during the postpartum period are imperative for appropriate mother-child bonding, the use of tramadol for the rapid remission of symptoms may be appropriate for some patients during SSRI titration.

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Biology Versus Psychoanalysis

The crude Cartesian dualism decried by Eric R. Kandel, M.D. (1), has been succeeded by much subtler considerations of the relationship between what we still, for lack of better terms, call "mind" and "brain." For instance, philosophers Frank Jackson (2) and Thomas Nagel (3), among others, have emphasized the degree to which subjective and objective descriptions are fundamentally different, and even irreconcilable, categories of explanation. Scientists aspire to a world view that is maximally objective and value neutral, whereas intrinsic to subjective phenomena are specific points of view and biased interests. In this view, a comprehensive objective science of human behavior may not be feasible. This is not to say that objective factors are irrelevant from a subjective standpoint, only that the former cannot, logically, account for all of experience. The issue then may not be some kind of ethereal substance called "mind" but rather the radical epistemological gulf that subjectivity represents.

The hallmark of psychoanalysis, and really all psychotherapy, is the attempt to fathom and, if possible, to ameliorate a patient's subjective reality. Psychotherapy entails such entities as self, person, meaning, values, and free will—all of which may be even less reducible to biology than the vaguer notion of "mind." Psychotherapy presupposes a freedom of subjective will that exists even in the context of great adversity. Psychotropic medication and other somatic treatments are necessary because there are obvious (and sometimes not-so-obvious) limits to freedom of will. The interplay of the two approaches, subjective and objective, touch on paradox and the limits of our understanding, but they are complementary and not mutually exclusive. If this argument turns out to be wrong, and biology does someday fully explain subjectivity, then traditional notions of autonomy and individual freedom will be discounted, psychotherapy will become superfluous, and our views of human beings, self, and other will alter in ways that are difficult to foresee.

Dr. Kandel is right that on experimental grounds, psychoanalysis never has been a science; rather, it is a hodgepodge of psychological theory, history, and literature. He laments the possibility that psychoanalysis may be relegated to the realms of philosophy and literature. His implication is astute, and his article convincingly advocates the replacement with biology of that part of psychoanalysis that has purported to be objective explanation. What remains is a wealth of thought about history, society, and art. In fact, it is notable that the decline of psychoanalysis within psychiatry has been paralleled by its meteoric rise in the fields of aesthetics and literary theory.

Psychoanalysis is being split asunder. Its scientific and objective aspirations, after long and eminent careers as heuristics for empirical research, are in the process of being consumed by biology, whereas its philosophical aspects live on as gloriously as before. It is no wonder that Freud vacillated between scientific and hermeneutical views of his creation, which, at times, he recognized as a loose and unstable amalgam.

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Use of ECT in Italy

The timing of the remembrance of Ugo Cerletti by Dr. Stefano Pallanti, M.D., Ph.D. (1), is ironic in view of the Feb. 15, 1999, *circolare* (government white paper) signed by Rosy Bindi, Italian Minister of Health, that replaces the government's previously favorable official view of ECT as an "indispensable" treatment, with stringent new restrictions on its use. The *circolare* specifies that private clinics will no longer be able to offer ECT, which may now be administered only as an emergency procedure in government hospitals after other treatments have failed and if the patient is in a "life-threatening" situation.

Because of politically based conflicts, the use of ECT in Italy was already among the lowest in the European community; the new regulations now threaten the very existence of this truly indispensable treatment in the land of its birth.

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Dr. Pallanti Replies

My thanks to Richard Abrams, M.D., who touched on a crucial issue. It is puzzling that despite the recognition in Minister of Health Bindi's *circolare* of the effects of ECT "in 50% of drug-resistant, depressed patients" and its tolerability, "with only transient mnesic effects," a radical curtailment is nonetheless envisaged, supposedly because of its "wild use." This seems unlikely when fewer than 50% of the mental health facilities can perform it, costs are not refunded by local administrations, and patients have to travel long distances for treatment. The incoherence of this *circolare* is unacceptable.

Admissible moral objections must be distinguished from propaganda and consensus seeking. Clearly, the wishes of the nonscientific community should be considered and reckoned with: a case in point is that of American HIV patients who successfully brought about the introduction of zidovudine before the completion of clinical trials. But Di Bella's nontoxic cocktail against cancer, which received financial support for clinical trials despite a lack of scientific documentation, clearly revealed institutional weakness in the face of public opinion, which was readily swayed by the image of the frail 85-year-old Di Bella and encouraged by certain opportunistic politicians too interested in consensus to care about science (1). ECT, on the other hand, is an unpleasant term with a bad image; it lacks powerful support from economic lobbies even though it works. Its virtual suppression represents the premature resignation of the Italian psychiatric community and also indicates the difficulty of scientific communication.

The question of communication needs addressing, especially in Italy, where humanist culture dominates. Physicians must devote more attention to the physician-patient relationship—not always given due consideration in the use of ECT, but still a prime vehicle of communication (2). And greater scientific literacy would allow adequate methodological evaluation, thus avoiding incoherence and the immoral consequences of the "rhetorical" stigmatization of ECT—a biased rhetoric that is neither humanistic nor scientific—which affects patients by prolonging their wait for appropriate therapy. It is worth remembering that Italy produced not only Cerletti but also Galileo, who was persecuted for his scientific achievements.

This letter is not a defense of ECT but a reflection: in scientific medicine, "we do what works" (3), but obviously there are implications, an example being the philosophical debate in neuropsychiatry on topics long considered to be "ontological" (e.g., cerebral death for organ donation, euthanasia). But therapeutic choices cannot acceptably be the result of purely political and ideological decisions. Clearly, science has its own inner ideology that reflects a contingent social context; it is utopic to think decision-making processes are free from social context, especially given the dramatic nature of health choices. Science used to be legitimized *dei gratia*, but now it seems that a superior divine authority is being replaced by a political authority rather than by methodological correctness and widespread informed approval.

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