Letters to the Editor

Relationship of Panic Attacks to Paranoia

TO THE EDITOR: In the Epidemiologic Catchment Area Program of the National Institute of Mental Health survey, panic attacks occurred in 28% to 63% of individuals with schizophrenia (1). Investigators have suggested that panic may be related in some way to paranoia (2, 3; unpublished report by Young et al., 1998). To further explore this intriguing connection, we report on a patient with self-described paranoid attacks.

Mr. A was a 42-year-old man who had had schizoaffective disorder since he was 22 years old; he had five hospitalizations for psychosis, depression, and suicidality. Early in his illness, he heard voices threatening to cut off his genitals. He became convinced that his neighbor wanted to shoot him, so he attempted suicide "to die before they could…torture me." Voices telling him that the Mafia was after him and that he was being watched and would be tortured continued episodically. Others sent their thoughts to him telepathically, he said. These symptoms were in good control on a regimen of clozapine, 200 mg/day, and lorazepam, 2 mg b.i.d., except when he had panic attacks.

Mr. A had panic attacks with severe anxiety, shortness of breath, choking, racing heart, sweating, dizziness, lightheadedness, chest pains, a feeling that he might lose control or "blow up," and, occasionally, numbness and tingling. During these episodes, he had a return of paranoia, commonly reexperiencing the thoughts that his genitals would be cut off and that others were talking about him behind his back. He referred to these episodes as paranoid attacks.

Siris and colleagues (4) presented three patients with schizophrenia or schizoaffective disorder and panic, one of whom also described his panic-like attacks as paranoid attacks. Galynker and colleagues (5) described four patients in whom panic attacks were closely followed by, or coincided with, the development of psychotic symptoms.

What is the relationship between panic and paranoid attacks? Panic attacks appear to be sudden explosions of extreme anxiety, with a host of associated physiological components, including rapid heart rate and breathing, sweating, and the like. Panic attacks include a cognitive component, a feeling of dread attributed to a sense of impending catastrophe, loss of control, or "going crazy." These cognitive elements might be the mind elaborating while seeking to explain sudden, inexplicable fear. The paranoid thoughts expressed by a person with paranoid attacks likewise might represent the cognitive elaboration of a panic attack, the result of a mental search to attribute such danger to a suitably malevolent component of the environment. Perhaps panic and paranoia share some underlying biological substrate.

Are paranoid attacks the form that panic attacks take in certain psychotically vulnerable individuals? Other patients with schizophrenia have told us, when questioned, that they have paranoid attacks. The rate of panic episodes in schizophrenia may reflect the presence of such a process, because respondents responded affirmatively to the panic question on the National Institute of Mental Health Diagnostic Interview Schedule: "Have you ever had a spell or attack when all of a sudden you felt frightened, anxious, or very uneasy in situations when most people would not be afraid?" How common are they? This phenomenon deserves further consideration from several perspectives, including implications for etiology and treatment (3).

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New Behavioral Approach to Trichotillomania

TO THE EDITOR: According to DSM-IV, the essential feature of trichotillomania is the recurrent pulling of one's hair, which results in noticeable hair loss and great tension before pulling out hair or when attempting to resist the behavior, followed by relief when pulling out hair.

Trichotillomania shares features in common with obsessive-compulsive disorder, especially with drug response (1). Unfortunately, patients do not seek treatment for years because of embarrassment. Patients with trichotillomania respond best to a combination of psychopharmacology, psychotherapy, and behavior-modification therapy rather than to psychopharmacology alone (2). We present the cases of three young women who responded well to a combination of medication and habit substitution (3).

Ms. A was a 17-year-old adolescent with a history of low self-esteem, depression, anxiety, and hair pulling. She was described as a perfectionist and a loner. She reported hair pulling because of nervousness but was concerned about the way it affected her looks. She was treated with paroxetine, 30 mg/day, and clomipramine, 100 mg at bedtime. Her depression improved, but her hair pulling continued. One day, her mother asked her to pull the weeds in their flower beds. She noticed being fascinated by pulling each weed and looking at its roots. When she pulled her hair, she noted that her hair also had roots. She reported feeling relief by pulling weeds. In a few weeks, she stopped

pulling her hair, which started growing back, and realized that her nervousness was calmed by pulling weeds instead of her hair.

Anna was a 9-year-old child who was admitted to an inpatient hospitalization program. Her main complaint was, "I am pulling my hair out." Her head was bald, and she was isolated from other children. A mental status examination showed depressed mood and hostile behavior. She was treated with fluoxetine, 20 mg/day. Encouraged by Ms. A, she was advised to practice weed pulling at home. After 3 months of outpatient visits, her condition showed improvement, and her hair started growing. Anna reported that feeling the roots of the weeds made her less tense and reduced the urge to pull her hair.

Ms. C was a 23-year-old woman treated on an outpatient basis for trichotillomania. She presented with severe depression related to the divorce of her parents and reported pulling her hair out and creating balding areas on a regular basis since the age of 12. She was placed on a regimen of clomipramine, 50 mg/day. Encouraged by Ms. A and Anna, Ms. C was advised to practice weed pulling. Also, as with these two patients, Ms. C began to show significant improvement after the initiation of medication and habit substitution.

As in the cases mentioned, patients with trichotillomania report an overwhelming urge to pull their hair. After this hair pulling, their inner tension is relieved until the return of the compulsive urge and its accompanying anxiety. These symptoms are similar to those of patients with obsessive-compulsive disorder, who have a similar response to treatment. It is, however, noteworthy that all three patients may have responded either partially or fully to psychopharmacology alone, because not all studies have found drug therapy beneficial for trichotillomania.

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Adverse Effects of Donepezil in Treating Alzheimer's Disease Associated With Down's Syndrome

TO THE EDITOR: Many individuals with Down's syndrome who are over 40 years of age develop clinical and neuropathological evidence of Alzheimer's disease. Cholinergic deficits have been linked to neuronal loss in the nucleus basalis of Meynert in patients with Alzheimer's disease, and the nucleus basalis of Meynert in patients with Down's syndrome contains fewer neurons than are found in normal comparison subjects (1). This implies that cholinergic deficits are also present in the nucleus basalis of Meynert in patients with Alzheimer's disease associated with Down's syndrome. Plasma concentrations of donepezil are related to the percentage of red blood cell acetylcholinesterase inhibition and to improvements in cognitive functioning (2). Patients with Down's syndrome were excluded from donepezil clinical trials (2), and therefore much fewer data regarding its pharmacological effects are available for this population. We report on three patients with Alzheimer's disease associated with Down's syndrome who were treated with donepezil.

Ms. A, a 59-year-old woman with Down's syndrome, demonstrated evidence of progressive cognitive impairment over 2 years, which was manifested as consistent confusion but without aggressive behavior. She started treatment with donepezil, 5 mg/day, and after 8 weeks, she had improvement in mood and was less confused. Ms. A's dose of donepezil was increased to 10 mg/day, and 2 weeks later, she became agitated and aggressive. Her donepezil treatment was then discontinued, resulting in decreased agitation and aggression.

Mr. B, a 57-year-old man with Alzheimer's disease associated with Down's syndrome, demonstrated poor short-term memory and impulsive behavior for several months. He had difficulty with basic activities such as dressing. He started donepezil treatment, 5 mg/day, and after 4 months, he developed urinary incontinence and became increasingly forgetful. His donepezil treatment was discontinued; his urinary incontinence stopped, and he returned to baseline cognitive functioning.

Ms. C, a 65-year-old woman with Down's syndrome with depression, apathy, and obsessive-compulsive features that were unresponsive to multiple antidepressants, started treatment with donepezil at 5 mg/day. She developed urinary incontinence that stopped when the treatment with donepezil was discontinued.

Patients with Down's syndrome may respond to medications used to treat Alzheimer's-associated psychopathology (3). However, further studies are required to adequately assess the safety and efficacy of donepezil for Alzheimer's disease associated with Down's syndrome. In these cases, donepezil treatment resulted in adverse effects related primarily to peripheral cholinergic overstimulation, with symptoms of urinary incontinence in two of three patients. Administration of a peripherally acting cholinergic antagonist or possible use of a lower dose of donepezil may block these side effects, allowing for safe use of this medication in this population. Clinicians should be alert to the possibility of adverse effects when administering donepezil to individuals with Down's syndrome.

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Olanzapine-Induced Ketoacidosis With Diabetes Mellitus

TO THE EDITOR: Olanzapine is a new atypical antipsychotic compound with a receptor profile similar to that of clozapine, with generally few side effects. Specifically, extrapyramidal symptoms did not change in relation to baseline when olanzapine-treated patients and control subjects were compared (1–3), although weight gain and transient liver enzyme elevation have been found. Given olanzapine's structural similarity to clozapine, it might be expected that some rare side effects seen with clozapine, such as impairment of glucose tolerance, could occur with olanzapine (4). We report on a patient who developed acute ketoacidosis with diabetes mellitus while taking olanzapine.

Mr. A was a 50-year-old, single, African American man with a 30-year psychiatric history with multiple psychiatric hospitalizations. He had been admitted to Manhattan Psychiatric Center 20 years earlier and had been hospitalized continuously since then. His diagnoses included schizophrenia (paranoid type), substance dependence, and antisocial personality disorder. He had a significant history of criminal and deviant sexual behavior. His medical history included hypertension, mild obesity (227 lb), and myocardial infarction but not diabetes. He had been on a regimen of extended-release nifedipine, 30 mg/day, to control his hypertension. He did not have a significant family history of diabetes.

During Mr. A's hospitalization, a variety of antipsychotic medications were used, including fluphenazine decanoate, 25 to 75 mg i.m. every 2 weeks, chlorpromazine, 1800 mg/day, and haloperidol, 40 mg/day. He had no major side effects from these drugs. Mr. A had also received divalproex sodium, 750 mg twice a day, for 2 years. His psychiatric disorder had remained resistant to conventional antipsychotic medications. He refused to take clozapine because of the blood testing that was required. The results of his annual laboratory tests in 1997, including his blood sugar level, were normal.

Mr. A started olanzapine treatment, 5 mg/day. He continued to receive fluphenazine decanoate, 75 mg i.m. every 2 weeks, and divalproex sodium, 750 mg twice a day. His olanzapine dose was then gradually titrated to 30 mg/day over 6 months with the intent of gradually tapering his dose of fluphenazine decanoate once he was stable. His delusions and paranoid symptoms improved with this therapy. His weight was 227 lb at the start of olanzapine treatment, it increased to 248 lb after 5 months, and then it went down to 205 lb at the end of treatment. That same month, Mr. A was found drowsy, lethargic, and unresponsive to verbal commands. His blood pressure was 98/68 mm Hg, and he had orthostatic hypotension. Mr. A was then transferred to a hospital emergency room and later admitted to the intensive care unit for diabetic ketoacidosis; he had a blood sugar level of 1200 mg/dl. He was given intravenous insulin and 5% dextrose fluid.

Mr. A's olanzapine regimen was discontinued, and his diabetic ketoacidosis disappeared. He received human insulin (NPH), 40 to 70 units for 15 days. His insulin treatment was then discontinued, with subsequent normalization of his blood sugar level. His levels of blood sugar, urine sugar, and acetone remained normal. He remained on a regimen of fluphenazine decanoate, 75 mg i.m. every 2 weeks, benztropine mesylate, 2 mg twice a day, and divalproex sodium, 750 mg twice a day. Both the manufacturer of olanzapine and the Food and Drug Administration's MedWatch were notified of his drug reaction.

Mr. A developed de novo diabetes mellitus after 8 months of adjunctive olanzapine treatment. After he discontinued olanzapine, his diabetes mellitus disappeared completely. We believe that olanzapine constituted an etiological factor in the development of Mr. A's endocrine disorder. According to the manufacturer of olanzapine, diabetes mellitus was reported in 0.6% of patients in olanzapine premarketing trials (G. Rubel, Eli Lilly and Co., personal communication). As with clozapine, the pathophysiology of decreased glucose tolerance is unknown. Weight gain during olanzapine treatment is well recognized and may have contributed to the development of Mr. A's vulnerability to diabetes mellitus. Furthermore, he did not have a family history of diabetes mellitus. It is also possible that the 6 weeks of his high dose of olanzapine (30 mg/day) may have played a role, although it is not clear at what drug level the first symptoms of impaired glucose tolerance appeared. Also, it cannot be ruled out that the combination of olanzapine, fluphenazine decanoate, and divalproex sodium may have had an adverse effect on Mr. A's glucose tolerance. However, he had been taking fluphenazine decanoate and divalproex sodium for several months before the onset of his diabetes mellitus and again after recovery from it, without diabetic symptoms. Olanzapine or one of its metabolites may have contributed to the suppression of insulin release. This effect has been reported in studies with chlorpromazine, another antipsychotic, which can produce a hyperglycemic response in rats during in vivo and in vitro studies (5). Clinicians using olanzapine at a dose of more than 20 mg/day in patients developing symptoms of diabetes mellitus or in patients with a preestablished history of diabetes mellitus may need to check blood sugar levels more frequently.

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Olanzapine-Induced Elevation of Plasma Triglyceride Levels

TO THE EDITOR: The relatively recent introduction of the newer atypical antipsychotic medications—risperidone,

olanzapine, quetiapine—has resulted in a shift in the prescribing practices of psychiatrists. This is likely because of both efficacy and safety. These drugs have demonstrated efficacy at least comparable, and possibly superior, to that of typical antipsychotic drugs, with less likelihood of inducing extrapyramidal side effects. One of the most clinically relevant side effects of the atypical antipsychotics at recommended doses has been weight gain. There are limited direct comparisons of the atypical antipsychotics, although olanzapine has been associated with greater weight gain than risperidone (1). The significance of this beyond cosmetic effects is unknown. This letter describes how long-term olanzapine treatment may affect lipid profiles in a group of chronically institutionalized patients with schizophrenia.

Nine fasting patients (seven men, two women), whose mean age was 41 years (range=24 to 67 years), had blood drawn for plasma lipid panels and their weight measured before starting treatment with olanzapine. This information was obtained by means of routine hospital data collection; therefore, written informed consent was not obtained. The panels consisted of cholesterol, triglyceride, and high- and low-density lipoprotein levels. Lipid panel results and patients' weights were reexamined after an average of 16 months of treatment. The patients' mean dose of olanzapine was 19 mg/day (range=10 to 30). Patients also received adjunctive treatments, with mood stabilizers most often prescribed.

Cholesterol levels, although elevated at baseline, and high- and low-density lipoprotein levels remained essentially unchanged. However, triglyceride levels (normal range=25 to 200 mg/dl) increased from a mean of 170 mg/dl (range=69 to 385) to a mean of 240 mg/dl (range=135 to 369). Five of the nine patients had at least a 50% increase in their levels, although this did not reach statistical significance (paired t test). Consistent with previous reports, patients had a mean weight gain of 22 lb.

This report suggests that olanzapine treatment may result in a marked increase in triglyceride levels for some patients. Whether these results can be generalized to less severely ill patients receiving monotherapy is unknown. In univariate analysis, elevated serum triglyceride levels are correlated with a risk for coronary artery disease, although there is much controversy over the link when other lipid risk factors are added to the model (2, 3). Given our limited understanding of the long-term side effects of olanzapine, monitoring lipid profiles may be advisable. This is likely most important for patients with other nonlipid risk factors, such as a family history of early-onset arteriosclerotic complications (4).

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A Colleague's Suicide

TO THE EDITOR: How does one cope with the suicide of a colleague? It has become common knowledge that the risk of suicide among physicians in general (1), and those who work in the mental health system in particular, is high. After many years of concern about the subject, in 1973 a research team was formed under the auspices of the American Psychiatric Association (2).

Despite the many analyses and taxonomies of at-risk personalities, the suicide of a colleague reopens with great personal pain the question of why someone whose job it is to save people would commit suicide (3). Beyond the pain and the consternation that fellow workers undergo when hearing the bitter news, the need to continue functioning and to handle the situation raises pressing questions regarding the treatment of the colleague's patients and what they should be told.

Can the patients cope with the knowledge that their therapist committed suicide—that the same person who helped them cope with excruciating mental illnesses, with their own suicidal thoughts and attempts, decided to throw in the towel and kill himself? On the other hand, is there any justification for hiding the truth from them? Does consideration for their mental health justify telling an untruth, which creates distance between the patient, who does not know, and the therapist, who knows?

There is also the question of whether it is possible to hide information from a segment of the population of a dynamic psychiatric ward. If the information unintentionally becomes known, lack of trust and distance between the therapists and their patients is likely to result.

The staff copes with such questions while dealing with their personal grief as well. Questions arise as to how sensitive they were to their colleague's distress and whether they could have done anything to prevent what happened. The staff must function on several levels at once: the staff level, the patients' level, and the level of their personal sorrow.

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Clinton, Sex, and Psychiatry?

TO THE EDITOR: President Clinton's sexual imbroglio has created a large literature of political and legal discussion. Surprisingly, the psychosocial implications of this scandal

seem to be barely discussed. Data suggest that 50% to 60% of American men and women have had extramarital sexual activity, although these figures perhaps include single brief encounters. This behavior is not normative or accepted by the betrayed spouse, however, since data suggest that adultery, when exposed to the spouse, leads to divorce in a high percentage of cases (1). The public exposure of President Clinton's adultery has led already in many of my patients to greatly heightened suspicion and insecurity regarding spousal unfaithfulness. Moreover, patients who confronted and overcame spousal unfaithfulness in their marriage in the past have had recurrences of painful feelings. I hypothesize that the psychological aspects of the resolution of the Clinton imbroglio may have had significant effects on the American family. If Hillary Clinton had forgiven her husband publicly, would this have led to a higher rate of stable open marriages? If President Clinton had been forced to resign, would this have strengthened superego vectors in American male sexuality? Did Ms. Lewinsky's confused sexuality and its inevitable entanglements serve as a reality check for individuals confronting seemingly innocent sexual temptation? Can we really discuss this issue only in political or legal contexts and ignore possible major psychosocial implications?

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Janus, Roman God of Doorways

TO THE EDITOR: Nancy C. Andreasen, M.D., Ph.D. (1), began her editorial by referring to the *Journal* as a "stately dowager" with "a wonderful Janusian capacity" and adds in parentheses that "Janus was a Greek god gifted with the ability to look both into the past and into the future."

Although as a longtime subscriber, I have no doubt whatsoever as to the capability of the *Journal* to look into both the past and the future (and especially the latter), I'd like to point out that Janus is, in fact, not a Greek god but, rather, the Roman god of doorways. He is usually depicted with two bearded faces looking in opposite directions—inward and outward, perhaps. The famous *Ianus geminus* of the ancient forum in Rome, dedicated to him, was a double barbican gate, facing east and west, which was open during war and closed during peace time; I leave readers to draw their own conclusions as to the psychiatric implications of this. More generally, all gateways, house doors, and entrances were under the protection of Janus, as indeed were all beginnings (as the name of our month January testifies).

In this sense, at the threshold of the twenty-first century, may I wish the "stately dowager with a youthful spirit" a long Janusian life, with many new beginnings.

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Editor's note: Although many classical gods had both Greek and Roman variants, George C. Lyketsos, M.D., F.R.C.Psych., is quite correct to remind us that Janus was specifically Roman. I realized this error after it was too late to correct it. The benefit accrued from this slip of the editorial brain is Dr. Lyketsos's interesting commentary.—N.C.A.

Adolescent Abuse: Risk for Psychiatric Disorders

TO THE EDITOR: In their recent article, Sandra J. Kaplan, M.D., and colleagues (1) have made a scholarly effort to study whether physical abuse functions as an additional risk factor for adolescent psychopathology. However, several important questions that may limit the contribution of this well-designed and clearly written article to the literature remain partially or fully unanswered. First, what is the impact of age at onset, frequency, and chronicity versus acuity of physical abuse on the development of psychopathology? Second, what is the relative contribution of the emotional abuse that commonly accompanies physical abuse (i.e., insults, slurs) to the development of psychopathology? Third, is the sexual behavior criterion used in this study—"with an adult at least 5 years older"—too lenient? This excludes sexual abuse by peers and siblings.

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Dr. Kaplan and Colleagues Reply

TO THE EDITOR: Yifrah Kaminer, M.D., raises some important considerations in child abuse research. We plan, in future analyses, to examine the impact of some of the variables named by Dr. Kaminer, including age at abuse onset, frequency of abuse, and verbal abuse (an aspect of emotional abuse). Unfortunately, with regard to abuse severity, there does not exist sufficient variability in severity ratings in our study group for its impact on psychopathology to be measured statistically. Finally, we realize that our definition of sexual abuse, which we used to exclude certain cases of sexual abuse, is conservative. However, this definition was selected because it is commonly used by researchers studying sexual abuse and could be reliably assessed by using social services records. We appreciate Dr. Kaminer's insightful comments and interest in our research.

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