

## Resolution of Palinopsia With Carbamazepine

**Sir:** Palinopsia is an unusual phenomenon characterized by the recurrence of visual images after the stimulus object has been removed.<sup>1,2</sup> In most cases of palinopsia, neuropathology has been identified.<sup>1,2</sup> Infrequently, palinopsia has also been described in association with schizophrenia.<sup>3</sup> We present the case of a man who suffered from psychosis and anxiety disorder, whose palinopsia responded to carbamazepine.

**Case report.** Mr. A, a 40-year-old man, was hospitalized complaining of combat-related "flashbacks" and insomnia that he had begun experiencing at age 21. These had diminished since his initial exposure to combat. By age 26, he suddenly began to experience visual abnormalities unrelated to his combat experiences, describing them as perseverations of images of objects he had previously visualized. He stated that, for about 10 seconds after seeing an object, he would continue to see its colored image, then the image would enlarge, lose shape, and disappear. Concurrently, he also began to experience paranoid delusions and tangential thinking. Results of Mr. A's physical examination, including a visual examination, were normal. Blood chemistry, CBC, urinalysis, head CT scan, and EEG findings were normal.

He met DSM-IV diagnostic criteria for psychosis not otherwise specified. He also qualified for anxiety disorder not otherwise specified because, although he experienced some post-traumatic stress disorder (PTSD) symptoms, he did not meet full criteria for PTSD. He was treated with imipramine 200 mg and trifluoperazine 10 mg daily. After 3 weeks, his flashbacks of war content, delusions, and insomnia diminished considerably; however, he continued to experience perseveration of visual images.

Disorders of an episodic or paroxysmal nature have been successfully treated with carbamazepine.<sup>4</sup> Because palinopsia presents as a repetitive, paroxysmal phenomenon reminiscent of an epileptic process, we added the antiepileptic agent carbamazepine to Mr. A's medication regimen. Forty-eight hours after carbamazepine treatment was initiated at 400 mg/day, Mr. A experienced a decrease of image perseveration. After 6 days of treatment, on 800 mg/day of carbamazepine, his palinoptic experiences disappeared. No carbamazepine level measurements were made because the patient refused them. He left the hospital soon thereafter.

Mr. A experienced chronic perseveration of visual images after the stimulus object was removed, consistent with palinopsia.<sup>1,2</sup> The patient also experienced macropsia of the perseverated images, a phenomenon associated with palinopsia.<sup>2</sup> Most cases of palinopsia have been associated with organic brain etiologies.<sup>3</sup> In the present case, however, no brain abnormalities were found by head CT neuroimaging or EEG. Nevertheless, it is still possible that an occult neurologic process is present, which may become manifest at a later time. It is likely that Mr. A's anxiety disorder associated with PTSD features was unrelated to palinopsia because the symptoms associated with the palinopsia had a different history of onset than his anxiety symptoms and the perseverated images were generalized to many objects. Furthermore, the flashbacks, paranoid delusions, insomnia, and loose associations subsided after 3 weeks of treatment with concurrent neuroleptic and antidepressant

medication. His image perseveration, however, continued until he received carbamazepine.

Carbamazepine has antimanic and antidepressant properties that require days to weeks for onset of therapeutic effects. However, the antiepileptic effects of carbamazepine may be noted within 24 hours of treatment.<sup>5,6</sup> Although our patient did not manifest any seizure activity, given his quick response, we suggest that carbamazepine may have its antipalinoptic effect by a mechanism similar to this drug's antiepileptic effect. More systematic treatment with carbamazepine may lead to a better understanding and treatment of palinopsia.

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## Paroxetine Prevents Migraines

**Sir:** There have been several reports indicating that serotonin selective reuptake inhibitors (SSRIs) can produce worthwhile improvement in migraine treatment or prophylaxis. In particular, fluoxetine<sup>1,2</sup> and fluvoxamine<sup>3</sup> have been useful in prevention, and paroxetine<sup>4</sup> has been effective in the treatment of chronic daily headache.

**Case reports.** Three of my patients with preexisting classical migraine (two with a family history of that condition and one adopted) received paroxetine for depression. In one case, paroxetine was not effective, and the patient stopped taking it. On doing so, his migraines returned, having been in abeyance. The second patient had severe and frequent migraines. Her depression responded to the paroxetine, and her migraines virtually ceased. She stopped taking paroxetine for surgery, and the migraines returned until she resumed the medication. If she missed or delayed a dose of paroxetine, the migraines would return; if a migraine began, she could advance her next dose of paroxetine by an hour or 2 and abort the attack. The third patient's depression and migraines both responded to paroxetine. In this instance, she noted that fluvoxamine, which she had taken earlier for her depression without benefit, had also helped her migraines, although to a lesser degree. In all instances, the patients

had been warned to expect headaches, these being among the common side effects of SSRIs.

Breslau and associates<sup>5</sup> investigated the association between migraine and major depression. They noted the usefulness of tricyclic antidepressants in both conditions and the possible role of serotonin. Studying a sample of 1007 young adults, they found that migraine predisposed to depression, and depression to migraine, and proposed that a shared mechanism explained this bidirectional comorbidity. It may be that the more effective the antidepressant, the more likely it will alleviate migraines.

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**An Open-Label Study of SSRI Treatment in Depressed Hispanic and Non-Hispanic Women**

**Sir:** Symptoms of depression are common across cultures, but the clinical presentation of depression may differ from country to country and, within the same country, differ between ethnic groups.<sup>1,2</sup> Among Hispanic patients, somatization is a more common presentation of depression than among non-Hispanic patients with major depression, and an increased prevalence of somatic symptoms in Hispanic patients with major depression suggests that they present more frequently at primary care rather than psychiatric facilities.<sup>3-7</sup> It is reported that Hispanic subjects may be more sensitive to the somatic side effects of pharmacotherapy.<sup>8</sup> Marcos and Cancro<sup>9</sup> found that depressed Hispanic women received lower doses of tricyclic antidepressants and had more problems with side effects than depressed non-Hispanic patients.

Twenty-six female patients (13 Hispanic and 13 non-Hispanic) were enrolled in an open-label study of paroxetine (N = 18) and fluoxetine (N = 8) at the Aurora Hispanic Center and the Mental Health Clinical Research Center, University of California, San Diego. Subjects were medically healthy and had DSM-III-R diagnosis of major depression.<sup>10</sup> The Hamilton Rating Scale for Depression (HAM-D)<sup>11</sup> and a 17-item somatic checklist were administered weekly. Data were checked for normal distribution and homogeneity of variance: analysis of variance (ANOVA), t tests, chi-square tests, and the Fisher's exact test were performed.

The mean  $\pm$  SD age of the Hispanic group was 33.0  $\pm$  14.0 years versus 44.8  $\pm$  12.1 for the non-Hispanic group (t = 2.3, df = 24, p = .03). The mean  $\pm$  SD years of education for the Hispanic group was 8.7  $\pm$  2.8 years versus 14.9  $\pm$  2.6 for the non-Hispanic group (t = 5.9, df = 24, p = .0004). One Hispanic patient reported a previous episode of major depression; 9 non-Hispanic patients reported previous episodes ( $\chi^2 = 11.5$ , df = 1, p = .0007). Four subjects in each group presented with lifetime

comorbid DSM-III-R Axis I diagnoses (diagnoses similar for both groups). The initial HAM-D score was 19.0  $\pm$  3.8 for the Hispanic group and 19.5  $\pm$  4.7 for the non-Hispanic group (t = .32, df = 24, p = .7). The final HAM-D scores were 6.6  $\pm$  6.8 for the Hispanic group and 5.3  $\pm$  7.8 for the non-Hispanic (F = .70, df = 1,15; p = .415). When age was covaried, the result did not change.

The groups were identical in type and number of somatic symptoms endorsed at baseline. Six Hispanic and 3 non-Hispanic subjects terminated early ( $\chi^2 = 1.5$ , df = 1, p = .216); reasons were identical for both groups: noncompliance, intolerable side effects, and pregnancy. Hispanic subjects averaged 2.2  $\pm$  2.0 side effect complaints versus 5.1  $\pm$  2.5 for non-Hispanic subjects (t = 3.18, df = 24, p < .005). There were no differences in response, side effects, or attrition rates between fluoxetine- and paroxetine-treated subjects.

Ours is the first prospective study to compare response rates, side effect profiles, and attrition rates for Hispanic and non-Hispanic patients from the same area who were treated with serotonin selective reuptake inhibitors. We believe a larger masked prospective study is warranted comparing treatment of depressed patients from different ethnic groups.

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**Akathisia as Violence**

**Sir:** Manifestations of akathisia, a side effect of antipsychotic drugs, include restlessness, muscular tension, and a compulsion to move.<sup>1</sup> Infrequently, agitation and violence have been reported to be associated with antipsychotic treatment and

could be related to akathisia.<sup>2,3</sup> Differentiating between akathisia that manifests itself as violence and generalized psychotic agitation is clinically important in order to avoid a vicious circle of violence in patients who are being treated with antipsychotics. We report a case of persistent agitation and violence in a patient with bipolar mood disorder that was probably a manifestation of akathisia.

**Case Report.** Mr. A, a 47-year-old white man with a diagnosis of bipolar mood disorder, was brought to the emergency room because he was screaming in the streets. Mr. A had over 30 past psychiatric admissions associated with agitation and violence and was often discharged against medical advice. He was nearly always noncompliant with his antipsychotic medications, claiming that they made him "jump and lose my temper." Prior to the present admission, Mr. A's daily medications included haloperidol 20 mg, lithium carbonate 1500 mg, divalproex sodium 500 mg, and benztropine 1 mg. At admission, the patient was grandiose, had loud and pressured speech, and admitted he was not taking haloperidol. He was given haloperidol 15 mg q.h.s. and benztropine 1 mg q.a.m. Within 24 hours he started pacing; became restless, agitated, and violent; complained of feeling "jumpy"; and attacked a staff member. On Day 5 of his hospitalization, haloperidol and benztropine were discontinued; chlorpromazine was started, and the dose was increased to 950 mg/day. Mr. A, although sedated, remained threatening and violent. On Day 13, chlorpromazine was discontinued, and haloperidol was restarted at a higher dose of 15 mg p.o. b.i.d. Mr. A again complained of "jumpiness" and punched a television cabinet, causing a self-inflicted fracture. On hospital Day 17, owing to an error, haloperidol was discontinued. The patient became calmer, less irritable, displayed no angry outbursts, and required no further room restrictions. After 5 days, when the error was discovered, haloperidol was restarted at a lower daily dose of 10 mg. Within 3 days, the patient became violent and required room restriction. Haloperidol was then discontinued, the patient's agitation and violence resolved, and a week later he was discharged. His daily medications were lithium carbonate 1500 mg (serum level = 0.9 mEq/L; this dose had not been changed during his hospitalization), lorazepam 1 mg, and divalproex sodium 500 mg. On these medications, he remained well 6 months postdischarge, his longest period as an outpatient.

The association between antipsychotic administration, akathisia, and violence in psychiatric patients has been noted in two reports.<sup>2,3</sup> Herrera et al.<sup>2</sup> showed a trend for more violent episodes to occur with haloperidol than with placebo or low-potency neuroleptics. Crowner et al.<sup>3</sup> found that for violent psychiatric patients taking antipsychotics, half of the assailants had akathisia before the assaults, while only 20% of nonviolent patients had akathisia. However, to support a causal relationship between antipsychotic administration, akathisia, and violence, it is necessary to document a clear onset of akathisia and violent behavior upon initiation of antipsychotic treatment and resolution of both with antipsychotic discontinuation. Although agitation and violence result from a severe manic episode, Mr. A's case documents such an association: on two occasions, the onset and the resolution of both his "jumpiness" and his violent behavior coincided with the beginning and the ending of antipsychotic medication treatment. The fact that the jumpiness occurred with haloperidol and not with chlorpromazine is another factor indicative that Mr. A has exhibited akathisia rather than nonspecific activation of mania; this is because akathisia is more common with higher potency as compared with low-potency neuroleptics. One can also specu-

late that Mr. A's rocky clinical history was related to aggressive behavior perpetuated by antipsychotic administration. The possibility that aggressive and violent behavior unresponsive to antipsychotic treatment could be a variant of akathisia should be included in the differential diagnosis of acute psychosis and in alternative treatment strategies for bipolar mood disorder. Benzodiazepines in combination with lower neuroleptic doses, lithium, or valproate should be considered.

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#### Cessation of Self-Mutilation in a Patient With Borderline Personality Disorder Treated With Naltrexone

**Sir:** Self-mutilation and self-injurious behavior may be due in part to dysregulation of endorphin neurotransmitter systems.<sup>1-7</sup> Self-mutilation and dissociation occur frequently in patients with borderline personality disorder and associated histories of traumatic abuse and neglect, suggesting there may be an etiologic relationship between trauma, neglect, endorphin system dysfunction, and self-mutilation.<sup>8,9</sup> This hypothesis is supported by the observation that some patients with borderline personality disorder have elevated pain thresholds, a phenomenon mediated in part by endorphin systems.<sup>10</sup> The interruption of the reinforcing effects of self-mutilation on endorphin systems by opioid receptor antagonists may help to reduce self-mutilation in selected patients with borderline personality disorder. Although the literature is conflicting, several studies from the developmental disabilities literature have in fact demonstrated a reduction or cessation of self-mutilation behaviors with opioid antagonists.<sup>11-17</sup> Their benefit for patients with severe trauma histories and borderline personality disorder remains unstudied, although others have speculated on their potential usefulness.<sup>15</sup> I now present a case in which naltrexone treatment resulted in a dramatic and near complete cessation of self-mutilation in a patient with borderline personality disorder.

**Case report.** Ms. A, a 28-year-old woman with a history of profound trauma and neglect, suffered from borderline personality disorder, recurrent severe major depression, dysthymic disorder, and alcohol dependence. She also experienced "bad" thoughts about herself that were obsessive and ego-dystonic in nature. I initially began working with her 3 years ago in conjunction with her individual psychotherapist to help reduce her depression and suicidality. She experienced strong urges to cut herself during times of interpersonal stress at work or in psychotherapy, and she cut her arms with a razor during these times to relieve these urges and reduce her tension. This behavior occurred as many as several times per week, was accompanied by frequent visits to the emergency room for sutures, and had persisted for almost a decade despite 8 years of psycho-

therapy. Her cutting behavior did not diminish during her intermittent periods of sobriety.

Although she attended Alcoholics Anonymous, she was generally unable to stay sober for more than a week or two between binges. On several occasions she managed to remain sober for 3 to 5 months.

I first started Ms. A on fluoxetine treatment and gradually titrated the dose to 80 mg/day. Her depressed mood lessened, and her suicidality became less intense, more distant, and manageable. With the increase in fluoxetine, her obsessive thoughts greatly diminished. She continued, however, to have difficulties with periodic binge-drinking episodes and self-mutilation. Although these sometimes occurred together, she would also cut herself during periods of sobriety. After 2 years of attempting to address these behaviors via behavioral management and involvement in her 12-step recovery activities, I started her on naltrexone 50 mg p.o.q. day. Since initiating naltrexone 1 year ago, she has sustained continuous sobriety from alcohol with the exception of one brief lapse during a period of severe stress. Her self-cutting behavior ceased as well, with only one minor incident of scratching.

Her sobriety and ability to successfully manage her urges to cut herself have resulted in improved overall functioning, including her ability to enter into an intimate relationship for the first time in her life.

This case report suggests a possible role for naltrexone in diminishing compulsive self-mutilation in patients with histories of trauma and borderline character pathology. Since naltrexone helps to reduce cravings for alcohol and relapse rates,<sup>18</sup> this patient's sobriety was most likely promoted by naltrexone. Her history of cutting during previous periods of sobriety argues against the possibility that her cessation of self-mutilation was an indirect result of her sobriety.

A single case report does not establish a clear benefit of naltrexone for decreasing self-mutilation in this patient group. Further research, such as double-blind, placebo-drug crossover studies, will be necessary to clarify whether naltrexone may benefit patients with self-mutilation and borderline personality disorder.

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