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ORGANIC CATATONIA

To the Editor:

I read with great interest the case series of catatonic syndrome in young people (Cohen et al., 1999). Catatonia in children and adolescents has hitherto received scant clinical and research attention, and the authors make a valuable addition to our growing knowledge of catatonia. I also strongly agree with their concluding remark that "clinicians should be able to recognize the (catatonic) syndrome outside the limits of a diagnosis of schizophrenia . . ." (p. 1045) as well as with their emphasis on the frequency of neuroleptic-induced adverse effects in patients with catatonia.

However, I was a little surprised by their comment regarding the status of catatonia in *ICD-10*. The authors erroneously suggest in their introduction that, "In the *ICD-10* . . . , catatonia is associated *only* with schizophrenia, and stupor with melancholia" (p. 1040, emphasis added).

There has been a tendency in the past to consider catatonia as a purely psychiatric disorder, despite many case reports demonstrating a wide range of medical and neurological diseases associated with catatonic symptomatology. This, along with the noninclusion of organic catatonia in *DSM-III-R* and *ICD-9* (and the earlier classifications), led to underrecognition and underemphasis of the organic etiology of catatonia.

Several workers (e.g., Ahuja and Nehru, 1989; Fink and Taylor, 1991) have argued for the inclusion of "organic catatonia" in the classificatory system, and currently both *ICD-10* and *DSM-IV* incorporate organic catatonia in the classification.

The "organic catatonic disorder" (F06.1) is described in both versions of *ICD-10*, CDDG (Clinical Descriptions and Diagnostic Guidelines) and DCR (Diagnostic Criteria for Research), though the DCR criteria are more stringent. In addition to the condition that the general criteria for F06 ("Other mental disorders due to brain damage and dysfunction and to physical disease") are met, the *ICD-10* requires the presence of stupor and/or negativism, and catatonic excitement, with rapid alteration between stupor and excitement. The confidence in the diagnosis is increased by the presence of additional catatonic phenomena (e.g., stereotypies, waxy flexibility). The DCR criteria also stress the need to exclude delirium before diagnosing organic catatonic disorder.

The authors (Cohen et al., 1999) themselves point out the importance of organic factors in catatonia, in their literature review as well as their own patient sample. However, while searching the English and French literature on the subject between 1977 and 1997, the authors have inadvertently left out the case report of catatonia in an adolescent patient with Prader-Willi syndrome (Dhossche and Bouman, 1997), as well as the case of a 12-year-old boy who presented with neuroleptic malignant syndrome (NMS) without fever (Hynes and Vickar, 1996). Increasingly, neuroleptic-induced catatonia and NMS are being viewed as disorders on the same spectrum.

It needs to be emphasized that organic catatonic disorder (or catatonic disorder due to general medical condition) must be first considered in every patient with catatonic signs, particularly in a patient with new-onset catatonia. The inclusion of organic catatonia in both *ICD-10* and *DSM-IV* augers well for the detection of new cases of organic catatonia.

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FLUVOXAMINE AND ENURESIS

To the Editor:

We read with great interest the letters describing children with depression or dysthymia and nocturnal enuresis receiving relief of enuretic symptoms with fluoxetine (Mesaros, 1993; Feeney and Klykylo, 1997), sertraline (Sprenger, 1997) and paroxetine (Murray, 1997). Meanwhile, selective serotonin reuptake inhibitors (SSRIs) are also effective in treating obsessive-compulsive spectrum disorders (OCSDs) as well as depression or obsessive-compulsive disorder (OCD) (Hollander, 1998). We have observed 2 similar cases with OCSDs and nocturnal enuresis receiving relief of enuretic symptoms with another SSRI, fluvoxamine.

The first case involved a 9-year-old boy who was referred with a history of intermittent explosive disorder, encopresis, and nocturnal enuresis. Encopresis and nocturnal enuresis occurred every day. The results of his previous urological workup had been negative. He was initially treated with alprazolam (a minor tranquilizer), imipramine, and desmopressin. His intermittent explosive disorder was partially improved by

alprazolam, but nocturnal enuresis was not improved by imipramine and desmopressin. We changed these drugs to fluvoxamine (25 mg orally every morning and night). Fluvoxamine completely improved the intermittent explosive disorder and encopresis, and for 3 months decreased the frequency of nocturnal enuresis from every night to 2–3 nights per week. Three months after initiation of fluvoxamine therapy, an increase in the patient's fluvoxamine regimen from 25 mg to 50 mg orally every morning and night completely improved his nocturnal enuresis. This patient was followed up for 7 months in our hospital while receiving fluvoxamine therapy, and he continued to be free of symptoms. No side effects of fluvoxamine were evident.

The second case involved an 8-year-old boy who was referred with a history of onychophagia and nocturnal enuresis. He also had never achieved nocturnal urinary continence and had a previous urological workup that was negative. Two weeks after starting a regimen of fluvoxamine 25 mg orally every morning and night, the frequency of nocturnal enuresis decreased from every night to 4 nights per week; 2 weeks later, its frequency decreased further, to 1 night per week. Onychophagia, which the patient had had since age 5 years, also was decreased by fluvoxamine therapy. No side effects of fluvoxamine were evident. Informed consent was obtained from the parents of these 2 patients, and they gave consent for this report to be published.

The literature describes 3 children treated with SSRIs for nocturnal enuresis (Feeney and Klykylo, 1997; Murray, 1997). These 3 children experienced complete resolution of enuretic symptoms within 1 month of SSRI therapy. One patient was a 13-year-old boy who gained relief of enuretic symptoms with sertraline therapy. His previous treatments, i.e., water restriction, bladder training, bed and pad, and trials of imipramine and desmopressin, were ineffective (Sprenger, 1997). Another patient was 15-year-old boy receiving complete relief of enuretic symptoms with fluoxetine therapy. He was treated with imipramine for nocturnal enuresis before fluoxetine therapy, and imipramine decreased the frequency of nocturnal enuresis from 6 nights per week to 3 nights per week (Mesaros, 1993). Finally, we report a case in which nocturnal enuresis responded to treatment with fluvoxamine.

We have administered fluvoxamine to 11 children with only primary and functional nocturnal enuresis without depression, OCD, or OCSDs. Informed consent was also obtained from both the children and their parents. Fluvoxamine was effective in 8 of 11 children without serious side effects. These data suggest that SSRIs may become a new drug for the treatment of nocturnal enuresis without the serious cardiac arrhythmia associated with tricyclic antidepressants or hyponatremia associated with long-term desmopressin treatment. The exact mechanism by which SSRIs could be beneficial in the treatment of enuresis is unknown. The effectiveness of these 4 SSRIs in treating enuresis suggests that the neurotransmitter serotonin may be involved in the etiology of some cases of nocturnal enuresis, and increased peripheral levels of serotonin as a result of SSRI treatment may directly cause smooth muscle relaxation in the bladder. Serotonergic facilitation controls enuresis without noradrenergic or anticholinergic treatment interventions, and antidiuretic hormone regulation has been linked to the serotonergic system.

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Comment by Drs. Horrigan and Barnhill, at the invitation of the Editor:

This letter presents another observation concerning the potential role of SSRIs as a treatment for nocturnal enuresis. In this instance the discussion involves fluvoxamine. The authors present a small case series, with minimal accompanying data, to support the assertion that SSRIs such as fluvoxamine "may become a new drug for the treatment of nocturnal enuresis." They posit that SSRIs offer a more benign safety profile relative to better-established pharmacotherapeutic agents such as imipramine and desmopressin.

The effects of serotonin, and drugs that manipulate serotonin, on urination have been noted in the medical literature for many decades (Catacutan-Labay et al., 1966). Most of the studies have been conducted on nonprimate mammals. Serotonin has the capacity to directly inhibit ureteral peristalsis as well as to inhibit micturition by interfering with spinal reflexes, primarily through 5-HT_3 receptor agonism at the level of the spinal cord (Catacutan-Labay et al., 1966). The central effects of serotonin on micturition are less straightforward. For instance, agonism of presynaptic 5-HT_{1A} receptors appears to diminish the threshold for micturition (which can culminate in enuresis), while antagonism of these receptors has an inhibitory effect on bladder reactivity (Testa et al., 1999).

The 2-edged nature of central serotonergic manipulation has clinical relevance across various drug classes. For instance, all of the atypical neuroleptics that incorporate 5-HT antagonism have been indicted with regard to secondary enuresis in previously continent individuals (Bennett et al., 1994). In one of the residential treatment settings that we serve, risperi-