strongly prevalent in every single diagnostic category, and the corresponding \( P_{\text{neg}} \) values were all very high: 25 of 28 of them were 0.87 or greater, indicating high negative agreement.

The present study is the first reliability study conducted on the Italian version of the MINI. The average duration of the interview, 27 minutes in the interrater and 21 minutes in the retest, was relatively short. Since most of the patients have a psychiatric diagnoses, the average length of the interview was longer than that found in general practice settings (15 minutes) but still was longer than that found in general is associated with low serum calcium level, high-dose antipsychotic therapy, and acute medical illness. Mechanism is thought to relate to D2 receptor blockade; the phenomenon is thought to occur only rarely with atypical antipsychotics.

Ziprasidone is an atypical antipsychotic with a high 5-HT2A/D2 receptor affinity ratio and inhibition of norepinephrine and serotonin reuptake. The compound is an agonist at the 5-HT1A receptor, an antagonist at the 5-HT2C and 5-HT2D receptors, and shows antagonism at z1-adrenergic and H1 histamine receptors.

Premarketing clinical trials of oral ziprasidone resulted in torticollis in less than 0.1% of patients and an EPS rate of 5%. A randomized, double-blind comparison of ziprasidone with haloperidol found a 1% rate of EPS with ziprasidone. A literature search found no published reports of torticollis since the drug’s release.

Expert consensus has deemed the EPS risk presented by ziprasidone therapy as equal to that of olanzapine, less than that of risperidone, and more severe than that seen with clozapine or quetiapine. A prospective study of the prevalence of acute dystonia among neuroleptic-treated inpatients found a rate of 3.1% overall. Ten cases of acute dystonic reactions occurred among 766 patients treated with novel antipsychotics; 8 of these were associated with risperidone therapy. No patients in this study were treated with ziprasidone.

Although package information on ziprasidone recommends an initial dose of 20 mg BID, recent expert consensus has advocated initial dosing be increased to 40 mg BID. The following report details the case of a cocaine-abusing, neuroleptic-naive female patient started on ziprasidone 40 mg BID who developed acute torticollis early in the course of treatment.

LS was a 31-year-old African American female admitted to a state psychiatric facility after physical altercation with a peer. Before admission, the patient had been at a halfway house for

Acute Dystonic Reaction With Moderate-Dose Ziprasidone

To the Editors:

Acute dystonia is a distressing and sometimes life-threatening form of extrapyramidal syndrome (EPS). Occurring within 3 days of initiation of antipsychotic therapy in 90% of cases, acute dystonic reactions may be precipitous, frightening, and lead to a breach in therapeutic alliance. A subtype, torticollis, consists of sustained contraction of strap muscles causing a lateral twisting of the neck. Here presented is a case of acute torticollis following moderate-dose administration of a novel antipsychotic, oral ziprasidone.

Previously described risk factors for acute dystonia include young age, male gender, history of acute dystonic reactions, and cocaine use. EPS in general is associated with low serum
2 weeks, following inpatient detoxification from cocaine and alcohol. Subsequent to her attack on a peer at this placement, the patient complained of auditory hallucinations commanding her to attack the woman in her sleep.

At admission, LS reported a several years' history of intermittent auditory hallucinations described as one clear voice issuing violent commands and insults. These had occurred during periods of sustained sobriety as well as while actively abusing drugs. She denied past neuroleptic trials.

The patient denied use of drugs or alcohol in the preceding 2 weeks. Previous use had featured periods of daily crack cocaine use intermittent for 10 years as well as alcohol several times per week. Examination showed no evidence of acute withdrawal. Urine drug screen (UDS) was negative, and serum calcium level was within normal limits. The remainder of the laboratory workup was unremarkable. At hospitalization, the patient was taking sertraline 50 mg QD and trimethoprim-sulfamethoxazole for a urinary tract infection.

To treat the patient's ongoing psychotic symptoms, ziprasidone therapy was initiated at a dose of 40 mg BID. The first 2 doses were well tolerated. Approximately 3 hours following the third dose of ziprasidone 40 mg, the patient complained of a strange sensation in her tongue and subjective dyspnea. Over the next hour, she developed torticollis to the right as well as a writhing lingual dystonia. She was given benztropine and diphenhydramine intramuscularly, which produced full resolution of symptoms within the next 30 minutes. Ziprasidone was discontinued and replaced with olanzapine, which the patient tolerated without further EPS for the remainder of hospitalization.

Acute dystonia is rare with atypical neuroleptics but may still occur. Risk factors in this patient include her age, history of cocaine abuse, and possibly, her recent urinary tract infection. It also cannot be completely ruled out, despite the negative UDS, that recent cocaine use directly precipitated the dystonia. Rapid medication titration may be crucial for severely ill patients; it may also be encouraged by the current atmosphere of managed care, which demands cost-efficient use of inpatient days. However, acute dystonia is in some cases life-threatening and in most cases extremely uncomfortable. Risk factors for this condition, such as age, gender, history, and drug use, should be kept in mind when designing a medication titration schedule, even with an atypical antipsychotic.

Rachel E. Dew, MD
Doreen Hughes, MD
Department of Psychiatry, Wake Forest
University Medical Center,
Winston-Salem, NC
rdew@wfubmc.edu

REFERENCES

SSRI-Induced Enuresis
A Case Report

To the Editors:

Enuresis is the habitual involuntary discharge of urine after the age of expected continence. This term is often used alone, imprecisely, to describe wetting that occurs only at night during sleep. It is more accurate, however, to refer to nighttime wetting as nocturnal enuresis; this distinguishes it from daytime wetting. Despite extensive research, the mechanism of enuresis has not yet been clarified in detail. Tricyclic antidepressants (TCAs) possess established antienuretic properties, while some recent case reports have shown selective serotonin reuptake inhibitors (SSRIs) to also have similar antienuretic properties and a safer side effect profile. We herein report a case of de novo enuresis induced by paroxetine, one of the SSRIs, which was observed in an adult with major depressive disorders. To our knowledge, there have been no previous reports of SSRI-induced enuresis in adult cases, while there has only been one recent case report of paroxetine-induced enuresis observed in a 14-year-old girl.

CASE REPORT

A 28-year-old woman was diagnosed to have major depressive disorders at 26 years of age according to Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition in our hospital. She showed depressive symptoms such as a depressive mood, insomnia, psychomotor inhibition, and suicidal idea. Her personal and family histories were negative for either affective or any other psychiatric disorders. She did not have a history of enuresis. The laboratory data, including the thyroid indices, showed no definite abnormality. Paroxetine was started at a dose of 10 mg/d, and it was gradually increased up to a dose of 40 mg/d within 3 months. Nocturnal enuresis was first observed within 2 weeks after the initiation of 40 mg/d paroxetine. The average frequency of nocturnal enuresis was 4 times a week. Due to the occurrence of enuresis after the use of 40 mg/d paroxetine, the dosage was gradually reduced to 20 mg/d within 2 months. Thereafter, the occurrence of nocturnal enuresis gradually decreased and stopped within 1 month.

The established effective treatments of enuresis have been shown to include both behavioral and psychopharmacologic interventions. TCAs have been demonstrated to possess an antienuretic effect; however, TCAs are