due to inhibition of cytochrome P4502D6 by sertraline, for which both are substrates.[4] Further, there is a generally decreased clearance of aripiprazole in women.[4] Since aripiprazole levels were not obtained, this hypothesis remains untested. Caution when using aripiprazole with sertraline or another P450D26 inhibitor, especially in women, would seem appropriate.

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## Paroxetine-Induced Bruxism Effectively Treated With Tandospirone

*SIR:* Several case reports have shown that selective serotonin reuptake inhibitors (SSRIs) induced bruxism.[1] This letter reports a patient with paroxetine-induced bruxism who was effectively treated with tandospirone, a partial agonist of the 5-hydoxytryptamine $(5\text{-HT})_{1A}$ receptor. Tandospirone is less potent at dopamine $D_2$ receptor than buspirone.[2]

**Case Report**

"Mr. Y," an 81-year-old depressed Japanese man, received a 4-month trial of sulpiride, a common antidepressant medication used in primary care clinics in Japan. Since his depressive symptoms did not improve, he was referred to the psychiatry clinic. At that time, he experienced all nine criteria for major depressive disorder outlined in DSM-IV.[3] He agreed to a trial which replaced sulpiride with paroxetine.

A regimen of paroxetine was started with a bedtime dose of 10mg. After 7 days at this dosage with no reported adverse effects, the dosage was increased to 20mg/day. At the 14-day follow-up, depressive symptoms remained, although his mood had improved. The dosage was again increased to 30mg/day. At the 21-day follow-up, his depressive symptoms were under better control. He was very pleased with this treatment. However, he complained of "night-and-day jaw clenching" and "sore jaws and teeth." His wife also witnessed his symptoms. Within a week of adding tandospirone, 10mg p.o. t.i.d., to the regimen, he reported that his jaws no longer clenched and that sore jaws and teeth improved.

**Comment**

This case report shows that the patient developed bruxism after starting on a regimen of paroxetine. It has been postulated that disturbances in the central dopaminergic system, especially within the mesocortical tract, are linked to bruxism.[4] SSRI-induced bruxism is considered to be a consequence of serotonergically mediated inhibition of the dopaminergic system. In addition, in this case, elderly age and prior neuroleptic exposure (sulpiride: $D_2$ and $D_3$ antagonist) before starting paroxetine predisposed him to develop bruxism.

It has been reported that buspirone, a partial agonist of the $5\text{-HT}_{1A}$ receptor, ameliorates SSRI-induced bruxism.[1,4] This case suggests that tandospirone also improves paroxetine-induced bruxism. Several mechanisms can be considered in explaining why $5\text{-HT}_{1A}$ agonists relieve SSRI-induced bruxism. It has been shown that systematic administration of $5\text{-HT}_{1A}$ receptor agonists increases dopamine release in the prefrontal cortex.[5] This effect is considered a major factor in ameliorating bruxism.

The precise mechanism of how $5\text{-HT}_{1A}$ receptors regulate dopamine release is not known; however, several mechanisms have been postulated. Presynaptically, agonist activation of the somatodendritic $5\text{-HT}_{1A}$ autoreceptors in the raphe, which reduces 5-HT cell firing, synthesis, and release of 5-HT, is considered to increase dopaminergic neuron firing in the ventral tegmental area and synaptic release of dopamine in the prefrontal cortex.[4] Postsynaptic effects are also suggested since $5\text{-HT}_{1A}$ receptors are localized on nonserotonergic neurons in various brain regions, including the prefrontal cortex. It has been shown that dopamine release is modulated by postsynaptic $5\text{-HT}_{1A}$ receptors in the prefrontal cortex.[6]

This case adds to the literature by suggesting that SSRI-induced bruxism can be treated with tandospirone, a $5\text{-HT}_{1A}$ receptor agonist.

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## Digoxin-Like Immunoreactive Factor in Human Cerebrospinal Fluid

*SIR:* Mammals produce a family of endogenous compounds that resemble the sodium pump-inhibiting cardiac glycosides, digoxin and ouabain.[1] These endogenous cardenolides have been proposed to be important in the pathophysiology of bipolar illness, where it is relatively deficient in the serum of manic subjects.[1] The measurement of these compounds in the CNS has not been previously documented.

Refrigerated cerebrospinal fluid (CSF) waste was frozen at −70°C within 18 hours of collection. Digoxin-like immunoreactive factor (DLIF) was measured by radioimmunoassay as described by Grider et al.,[1] using components provided by New England Nuclear (Billerica, Mass.). The correlation among four samples quantified twice was 0.88.

Ten samples were studied from three women and seven men. Mean age among the women was 41.9 years (range = 30 to 53), and among the men it was 61.6 years (range = 32 to 94). Mean DLIF concentration was 91.7 pg digoxin equivalents/ml.

**Comment**

Human CSF contains a substance which cross reacts with antidigoxin antibodies. This supports previous reports of CSF factors that bear immunologic resemblance to cardiac glycosides[2] and sodium pump inhibitory activity of CSF.[3] Though the source of CSF DLIF is not known, similar compounds have been purified from mammalian hypothalamus.[4] Given that the hypothalamic-pituitary-adrenal axis may be dysregulated in bipolar illness, that DLIF production is responsive to dexamethasone and adreno-corticotropin hormone (ACTH),[5] and that DLIF may be dysregulated in bipolar subjects,[1] it would seem important to study CSF DLIF in bipolar illness.

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## Postpartum Catatonia Associated With Atypical Posterior Reversible Encephalopathy Syndrome

*SIR:* Postpartum psychosis is a psychotic illness that usually begins within 2 weeks of childbirth. It often manifests in manic symptoms and confusion, while catatonia is less frequent.[1] In contrast, posterior reversible encephalopathy syndrome (PRES) is a reversible encephalopathy characterized by various neurological and psychiatric symptoms. It generally involves the parieto-occipital lobes and is closely related to hypertension. Atypical manifestations of PRES in which other brain regions are disturbed have also been reported.[2] We report here a case of postpartum catatonia associated with atypical PRES.

**Case Report**

The patient was a 19-year-old Japanese woman without a history of psychiatric illness. She was treated in an obstetric hospital from the 36th week of pregnancy because of severe hypertension. She delivered a child by Caesarean section in the 37th week. Postoperatively, she gradually became confused. On Day 13 after childbirth, she fell into a catatonic stupor and was admitted to our hospital. On admission, her blood pressure was 160/110mmHg. Physical and neurological examinations, laboratory tests,