ment that would cure the infection he was convinced was killing him. He learned about silver colloid as a possible "antiseptic" agent and instructions on how to make a silver colloid solution from the Internet.

There are two major points of interest in this case. First, as clinicians we need to be aware of the information sources our patients use and how they use them. While there is some good information on the Internet, there is also a lot of potentially harmful information. Many web sites offer easy access to products that may be harmful to patients. For example, an Internet search for "colloidal silver generators" yielded more than 100 sites. Part of our role as physicians is to provide education to our patients, not only about their illness but also about the possible misinformation that is freely available.

The second point is that seemingly innocuous "folk remedies," such as the one in our case, can lead to permanent harm. Given the fact that somatic delusions of infection or disease are not rare in psychiatrically ill patients, the use of colloidal silver might appear attractive to vulnerable patients. Clinicians are urged to be aware of the popularity and potential harm of the "remedy" presented in this case.

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Stabilization of Hypomania Following Initiation of Tamoxifen

TO THE EDITOR: Tamoxifen, an antiestrogen agent used in breast cancer treatment, inhibits protein kinase C and may have antimanic properties (1–3). We present the case of a patient with bipolar disorder who became depressed while receiving tamoxifen.

A 55-year-old Caucasian woman with bipolar disorder presented in December 2006 for evaluation of depression after starting tamoxifen for the treatment of breast cancer. While she was in her 20s, the patient's bipolar disorder was characterized primarily by hypomania. Beginning in her 30s, depressive episodes predominated. She was subjectively euthymic but at times hypomanic, and she was medication free for 3 years before starting tamoxifen. In December 2004, at age 53, she was diagnosed with stage I breast cancer and treated with lumpectomy and radiation, followed by 18 months of tamoxifen. The patient had no other conditions that present with depression. Two antidepressant trials plus stimulants had little effect until tamoxifen was discontinued by her oncologist in June 2006 when a mixed state ensued. Subsequently, lithium was started, which precipitated worsening mood. We feel that tamoxifen, then lithium, "stabilized" her mood in the depressed phase of the illness. We recommended increased dosing of lithium, antidepressant taper, avoidance of stimulants, and the addition of lamotrigine.

Mood stabilizers have been anecdotally associated with stabilization in the depressed phase of bipolar disorder. There is only indirect evidence for this phenomenon in that anticonvulsants, including valproate, are associated with risk of depression (4). Evidence for an association between tamoxifen and depression is conflicting (3, 5, 6).

One possible explanation for these clinical observations is that the pathophysiology of bipolar disorder may involve protein kinase C. Chronic administration of lithium and valproate has been shown to reduce protein kinase C isoenzymes in the frontal cortex and hippocampus in rats, and increased protein kinase C activity has been observed in postmortem brain tissue in bipolar disorder patients (7). Tamoxifen was antimanic in three small clinical studies (1–3), supporting evidence for a possible role of protein kinase C in bipolar disorder. Our case supports the need for larger studies of protein kinase C-inhibiting agents in hypomania. Because tamoxifen is widely used in the treatment of breast cancer, understanding its potential effect on mood regulation is also clinically important.

Limitations of this report include the episodic nature of bipolar disorder and unavailable follow-up data, making the association between tamoxifen use and depression tentative. We therefore advise careful consideration of risks and benefits before discontinuing tamoxifen for symptoms of depression.

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Poor Neonatal Adaptation After in Utero Exposure to Duloxetine

TO THE EDITOR: Risk-benefit analysis is central when forming a treatment plan for pregnant women with mental illness. This task is further complicated by limited data pertaining to the effects of antidepressants on the fetus, neonate, and child. To our knowledge, this is the first case report of in utero exposure to duloxetine.

"Ms. A" was a 36-year-old Caucasian female with a history of recurrent major depression, anorexia, and chronic neck pain. When she sought psychiatric consultation at 34 weeks' gestation, she was 1) in complete remission, 2) being treated by an anesthesiologist, 3) and receiving monotherapy duloxetine (90 mg/day). She was educated about neonatal behavioral syndrome, and duloxetine was subsequently decreased to 60 mg/day.

The child was delivered without complication at 38 weeks. Upon delivery, she was blue, with minimal respiratory effort and oxygen saturations in the 80s. Her Apgar scores were 7 and 9. After birth, she was transferred to the neonatal intensive care unit because she continued to require oxygen. The child was started on antibiotics while possible causes of transient tachypnea were assessed. Basic laboratory examination, blood gas, echocardiogram, and chest and abdominal x-rays were all normal on day 1. Breast feeding was discouraged because of concerns of exposure to duloxetine, and the mother was advised to switch to sertraline. Antibiotic treatment was discontinued after blood cultures remained negative.

On day 3, the child was weaned to room air but developed "jerky rhythmic movements," or "twitchiness." An electroencephalogram (EEG) showed nonspecific encephalopathic findings. The child did have episodes of shaking, and the EEG revealed no correlated changes. Phenobarbital was started, and despite a high blood level the following day, the child continued to experience occasional twitching. Head computed tomography, magnetic resonance imaging, and lumbar puncture were all normal. A repeat EEG conducted at 7 days was suggestive of subclinical seizures. A follow-up EEG at 7 weeks was normal. Phenobarbital was discontinued, and the child was diagnosed with tremors and neonatal seizures associated with neonatal behavior syndrome. At age 2, the child is healthy with consistently normal neurobehavioral development.

This case demonstrates the syndrome referred to as poor neonatal adaptation or neonatal behavioral syndrome, characterized by jitteriness, poor muscle tone, weak cry, respiratory distress, hypoglycemia, low Apgar score, and seizure (1). These symptoms start within hours, generally require only supportive care, and end within 1 to 2 weeks. The syndrome may occur in up to 30% of infants with selective serotonin reuptake inhibitor exposure (2), with a risk ratio of approximately 3.0 (3) or higher for premature infants (4). The mechanism underlying the syndrome is unclear. A dose of fluoxetine or nursing may decrease symptoms assuming they stem from withdrawal. Reports indicate a higher risk with exposure to paroxetine and venlafaxine, agents with the shortest half-lives, but data are very limited pertaining to newer antidepressants such as duloxetine, mirtazapine, and bupropion. Better characterization of poor neonatal adaptation and its etiology could reduce invasive procedures and inform the difficult decisions in treating mental illness during pregnancy.

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Cingulate Gyrus Tumor Presenting as Panic Attacks

TO THE EDITOR: The diagnosis of panic disorder is usually straightforward, but tumors or epilepsy of the temporal lobe may rarely present as panic attacks (1). We report the case of a teenager who presented with short-lasting episodes resembling panic attacks secondary to a dorsal anterior cingulate ganglioglioma.

A 15-year-old boy presented with a 3-month history of recurrent, unexpected panic attacks occurring four to five times daily. His clinical history was unremarkable, and no stressful events were reported. During his panic attacks, he experienced intense anxiety, palpitations, trembling, shortness of breath, feelings of choking, dizziness, lightheadedness, and hot flashes. The episodes were unprovoked and usually lasted 1 to 2 minutes. On two occasions, he reported loss of muscle tone in the lower limbs. He developed concern (after >1 month) about having fur-