ety of uncontrollable factors, ranging from basic material constraints to soldiers' psychological condition, when assigning soldiers to a treatment modality. We recognize that we were unable to control for all the factors that might have influenced allocation of treatment modality, but we find it extremely impossible to think about a naturalistic research design during battle that would be able to control for such factors.

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More Evidence for the Role of Persistent Dissociation in PTSD

TO THE EDITOR: The report by Briere and colleagues (1) highlights the importance of persistent dissociation in the etiology of posttraumatic stress disorder (PTSD). On the basis of two cross-sectional studies, the authors conclude that "the primary risk for PTSD is less whether one dissociates during (or soon after) a traumatic event than whether such dissociation persists over time" (p. 2299). We agree with the authors' conclusions. It is of interest to note that our article published in 2002 in the *British Journal of Psychiatry* (2) reported two *prospective* longitudinal studies of motor vehicle accident survivors and came to a remarkably similar conclusion: "Persistent dissociation and rumination 4 weeks after trauma are more useful in identifying those patients who are likely to develop chronic PTSD than initial reactions." Participants were assessed very soon after the accident and followed for 6 months. Persistent dissociation at 4 weeks was a better predictor of chronic PTSD at 6 months than peritraumatic dissociation measured in the immediate aftermath of the trauma. Another prospective longitudinal study of assault survivors published in 2003 in the *Journal of Consulting and Clinical Psychology* (3) showed that persistent dissociation predicted an additional 8% variance of PTSD severity at 6 months over and above what could be predicted from trauma severity and cognitive processing measures, including peritraumatic dissociation. These findings from prospective longitudinal studies complement the cross-sectional results of Briere and colleagues and support their conclusion. The role of persistent dissociation as a maintaining factor is also highlighted in recent psychological models of chronic PTSD (eg., 4).

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Aripiprazole-Induced Acute Dystonia

TO THE EDITOR: Aripiprazole, the new-generation atypical antipsychotic that is a dopamine system stabilizer, is known for its low propensity to cause extrapyramidal symptoms (1). Although several reports of dystonia associated with the use of newer atypical antipsychotics are accumulating, a thorough PubMed search through Nov. 6, 2005, did not reveal a single such case induced by aripiprazole. We report a case of acute dystonia observed during aripiprazole therapy.

"Mr. A," an 18-year-old man with no contributory family, past, or personal history was brought to our community clinic with a 4-week history suggestive of a psychotic manic episode. Treatment was initiated with tablet divalproex, which was increased to 500 mg b.i.d., along with aripiprazole, 15 mg/day. In the next follow up, his parents complained about episodes suggestive of dystonia in the form of torticollis, which started within 3 days of treatment initiation. Mr. A did not receive any other medication in between. Trihexyphenidyl, 2 mg/day, was added to the regimen, and dystonia did not recur.

Exact mechanism of neuroleptic-induced acute dystonia is still unclear and possibly attributable to a higher ratio of dopamine-acetylcholine antagonism (2) or postsynaptic dopamine hypersensitivity (3) in the basal ganglia. However, preclinical studies with aripiprazole have shown that the drug's unique partial agonism at dopamine D_2 and serotonin 5-HT_{1A} receptors, along with antagonism of 5-HT_{2A} receptors, actually protects against emergence of extrapyramidal symptoms (1).

In our case report, Mr. A had two known risk factors for dystonia, i.e., being young and male (1). Additionally, we propose the following neurobiological mechanisms that either alone or in combinations could have resulted in acute dystonia with aripiprazole.

First, aripiprazole—unlike clozapine, which does not induce acute dystonia—lacks protective anticholinergic action (1). This was supported by the fact that, in our case, dystonia resolved with anticholinergic medicines. Second, aripiprazole's action on the D_3 receptor and antagonism of 5-HT_6 and 5-HT_7 receptors is still unknown (1) and could play a potential role. Third, postsynaptic dopamine hypersensitivity was found in bipolar patients (4), which may be a significant factor. Fourth, the possibility of complex drug interaction of aripiprazole with divalproex cannot be eliminated. Last, preclinical studies found inhibitory action of aripiprazole on serotonin transporter (1), which could potentially alter dopamine balance in the basal ganglia region.

In short, our case report reveals that even aripiprazole may cause rare dystonia in vulnerable individuals, despite having a favorable receptor profile. However, this side effect is amenable to conventional anticholinergic treatment.

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