catatonia, an eminently treatable condition (Fink and Carlson, 1995). Catatonia responds well to benzodiazepines, and when these fail, as they occasionally do, it responds to convulsive therapy.

In patients with a pervasive refusal syndrome, an alternate diagnosis of an affective disorder with catatonic features should be considered. Such consideration is especially useful when a treatment course is protracted. This possibility is not precluded by the presence of psychological stressors or sexual or family abuse features which may dominate the clinical picture. Catatonia, whether of mind or brain origin, is eminently treatable.

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## METHYLPHENIDATE EUPHORIA

To the Editor:

We would like to report a case of euphoria induced by methylphenidate in an 11-year-old prepubescent boy treated for hyperkinetic disorder, diagnosed according to ICD-10 criteria.

The child was treated with 10 mg of methylphenidate daily, increased after 4 weeks to 15 mg daily after a good clinical response. Within 5 days of starting the medication the child contacted the prescriber by telephone, of his own accord, to ask whether he could increase the dose. At a later review meeting, 6 weeks after commencing the medication, the child volunteered that he liked taking the tablets because they made him feel "nice" and "very happy," describing the sensation produced as a "buzz." He also confessed to having stolen two 10-mg tablets from his grandmother, who had taken responsibility for administering the medication, because he liked taking them so much. As a result of these revelations, the medication was changed to pemoline. The good clinical effect has been maintained, with improved functioning both at home and at school, but with no recurrence of a euphoric effect and no further theft or abuse of medication by the patient.

Drug-induced euphoria, with the risk of dependency and abuse, is a serious side effect of stimulant medication. Although euphoria is a well-recognized effect of amphetamines in adults, the evidence so far available has suggested that such a response does not occur in prepubertal children (Taylor, 1994). To date the youngest reported case of methylphenidate-induced euphoria is that of a 13-year-old boy, in the early stages of puberty, who developed the response after 2 years of prior treatment (Goyer et al., 1979). Our experience suggests that such a reaction can occur in a younger, prepubertal child and may also lead to abuse of medication.

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## RISPERIDONE AND TARDIVE DYSKINESIA

To the Editor:

We wish to report a case of tardive dyskinesia developing during the treatment of an adolescent female with the neuroleptic risperidone (Risperdal®). To our knowledge there has been only one previously reported case of tardive dyskinesia with Risperdal® use, and this involved a 28-year-old woman (Addington et al., 1995). To our knowledge this is the first reported case of presumed Risperdal®-induced tardive dyskinesia in the child and adolescent population as well as in a patient without previous neuroleptic exposure.

C.B., a 14-year-old white female with a history of attention-deficit hyperactivity disorder (ADHD), depressive disorder not otherwise specified (NOS), eating disorder NOS (characterized by frequent binge-eating episodes and obesity), and posttraumatic stress disorder (PTSD) (related to a history of sexual abuse), had been receiving mental health care at a treatment center in Dayton, Ohio, since January 1994. We first saw this patient in February 1996. While her history was highly suggestive of intrauterine exposure to illicit substances and alcohol, it was otherwise unremarkable for substances of abuse, head injury, or other medical or neurological disorders. Her medication history was significant for methylphenidate (Ritalin®) use since April 1994, fluoxetine (Prozac®) use since July 1995, and (Risperdal®) use since July 1995. According to record, the previous provider prescribed Risperdal® for symptoms of behavior dyscontrol and insomnia. The patient had been treated with imipramine up to 200 mg at bedtime for approximately 1 year. The imipramine therapy was discontinued in February 1995 after C.B. had an imipramine serum level of 800 ng/

mL. She subsequently started Prozac® for ongoing symptoms of depression. On her presentation to us, she was on a medical regimen of Ritalin® 20 mg twice/day and 10 mg twice/day, Prozac® 20 mg/day, and Risperdal® 0.5 mg at bedtime.

During the intake evaluation, the patient and her foster mother's greatest concern centered on this "serious twitch" that involved only the patient's face. Specifically, the twitch involved a wrinkling up of the nose with deviation of the upper lip to the right. This symptom had its onset in October 1995 3 months after initiation of Risperdal® in addition to her Ritalin® and Prozac® regimen). The foster mother noted that this symptom had increased in intensity over the following 8 weeks without any periods of waning or alteration in the degree of facial involvement. On C.B.'s last appointment, approximately 1 month prior to seeing us, her Ritalin® dosage had been decreased from 20 mg q.i.d. to 20 mg b.i.d. and 10 mg b.i.d. and her Risperdal® regimen had been decreased from 1 mg at bedtime to 0.5 at bedtime. These medication changes failed to alter the symptomatology, and the abnormal movement continued at a constant frequency and level of facial involvement. The patient confirmed again the involuntary nature of the symptom and noted she was being teased by her peers, creating even greater social difficulty. Her initial Abnormal Involuntary Movements Scale (AIMS) score was 8 on presentation to us. At this point the Risperdal® was completely discontinued. A 0.05-mg dose of clonidine was added in the evening to aid sleep. In addition, given a worsening of ADHD symptoms (particularly problems with concentration), she started Ritalin-SR® 20 mg at noon, and her regular Ritalin® regimen was changed to 20 mg t.i.d. When we saw C.B. 6 weeks later, these was no change in her involuntary movement symptoms or AIMS score. At no time had this symptom waned in severity or involved other regions of her body. She has never had symptoms consistent with vocal tics.

This is, to the best of our knowledge, the first reported case of the onset of tardive dyskinesia in a pediatric patient treated with Risperdal<sup>®</sup>. The dose used in this case was 1 mg/day, which is well within the optimal dose range for efficacy and low side effects established in dose-finding studies involving treatment of adults with Risperdal<sup>®</sup> (Chouinard and Remington, 1993).

Risk factors for tardive dyskinesia in this patient include female gender, presence of an affective disorder, lack of a psychotic disorder, and early neuroleptic exposure (Hales et al., 1994). This case is noteworthy in that the patient had no previous exposure to neuroleptic agents. It is worth considering the possibility that her assumed intrauterine exposure to alcohol and illicit substances may have placed this patient at increased risk for tardive dyskinesia. The

effect of her long history of exposure to Ritalin® as well as Prozac® is impossible to assess fully.

This case supports the need for careful consideration to be given before starting neuroleptic agents in pediatric patients. Conditions considered specific for neuroleptic use in adults (psychoses and major affective disorders) represented only 28% of the diagnoses among the child and adolescent patients who were receiving neuroleptics during a recent study (Richardson et al., 1991). Pediatric patients exposed to even short-term neuroleptic use should undergo baseline and continual monitoring for neurological side effects.

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## CD AND ADHD IN BIPOLAR DISORDER

To the Editor:

Anecdotal reports and claims of intimate associations between conduct disorder (CD) or attention-deficit hyperactivity disorder (ADHD) in children with bipolar illness have been presented by several investigators. However, the true nature of the relationship between these conditions remains unclear. Given that a specific relationship would impact evaluation, treatment, prognosis, and research involving these conditions, we were compelled to undertake a critical review of the relevant literature.

We reviewed six studies examining the relationship between CD and bipolar illness between 1988 and 1995. We eliminated three because we felt that their methodology was inadequate to address the problem of comorbidity (selected population, unstructured evaluation, or very small sample size). Among the three remaining studies, there was a surprisingly consistent rate of comorbidity of 37% to 42% (Carlson and Kashani, 1988; Kutcher et al., 1989; Wozniak et al., 1995).

Carlson and Kashani (1988) used the Diagnostic Interview for Children and Adolescents (DICA) and the Child Behavior Checklist to determine diagnoses in 120 random, nonreferred