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ADDERALL, THE ATYPICALS, AND WEIGHT GAIN

To the Editor:

Atypical neuroleptics are increasingly used in child and adolescent psychiatric populations. A variety of factors account for this state of affairs, including the rapid onset of clinical benefits from atypical neuroleptics across a wide range of diagnoses, their apparent lack of cognitive toxicity, and their alleged favorable safety profile in comparison with the traditional neuroleptics. However, the majority of clinicians who prescribe these agents to children and adolescents are also attuned to their high probability of increased appetite and subsequent weight gain as iatrogenic liabilities (Allison et al., 1999). A variety of palliative strategies have been proposed to combat these phenomena, ranging from dietary interventions to the use of adjunctive pharmacotherapies. However, none of the latter approaches has emerged as particularly appealing. For instance, topiramate is associated with cognitive dulling (in essence, mirroring the problems of a much older anticonvulsant, phenobarbital) (Mohamed et al., 2000), while the latest proposal to deploy adult antidiabetic medications such as metformin (Cottingham et al., 2000) is viewed as an antithetical solution by many clinicians.

More than 40 years ago, the amphetamines were described as "the best available drugs to aid in reducing weight" (Leake, 1958, p. 94). Adderall® is a combination of amphetamine salts that was previously marketed under the trade name Obetrol®, with a primary niche as an antiobesity medication (Berman and Anderson, 1966). Years later it was revived as a pharmacotherapy for attention-deficit/hyperactivity disorder (ADHD), and it has emerged as a successful competitor in this regard. The case described below may provide some reconciliation between Adderall's past and present roles.

M.N. is an 11-year-old boy with ADHD, chronic posttraumatic stress disorder, intermittent explosive disorder, and conduct disorder who was in residential treatment when first prescribed olanzapine as an adjunct to Adderall 10 mg po tid. His response to valproic acid, lithium carbonate, sertraline, clonidine, and methylphenidate at appropriate dosages and trials of suitable duration to establish efficacy (or lack thereof) had been inadequate. While the Adderall was associated with a 50% decrement in M.N.'s Conners Global Index score across the day (and the only side effect was a mildly decreased appetite), M.N. continued to experience paroxysmal episodes of reactive, affectively charged, physical aggression. Adjunctive olanzapine was implemented at a dose of 10 mg po qhs, and this medication was successful almost at once with regard to the target symptom of reactive physical aggression (aggressive outbursts declined by more than 80% in the 2 weeks following the initiation of olanzapine).

However, increased appetite and subsequent weight gain also rapidly emerged. M.N.'s weight increased from 36 to 40 kg (a body mass index change from 16.9 to 18.8 kg/m^2) during the first 6 weeks of olanzapine pharmacotherapy. His weight ascended at a rate of 0.4 kg/week over the next 2 months, which alarmed M.N.'s caretakers despite their satisfaction with his change in deportment. After a review of all reasonable options, the administration time of M.N.'s Adderall was shifted from immediately after meals to 45 minutes before meals (this corresponded to a new dosing schedule of 6:45 A.M., 11:15 A.M., and 4:15 P.M.), in an effort to temper M.N.'s irrational exuberance at mealtimes. In the wake of this intervention. M.N.'s appetite appeared to normalize as did the velocity of his weight gain, and, in fact, he actually lost 2 kg over the next 6 weeks. However, he took a scheduled 2-week drug holiday from the Adderall (but not the olanzapine), and during this time he regained 2.4 kg. This weight was shed within 2 weeks after the reinstitution of the previous Adderall regimen. His discharge weight was ultimately 41 kg after 7 months of residential treatment, which corresponded to a body mass index of 18.2 kg/m^2.

Careful psychopharmacology often requires an appreciation of the utility of a medication's side effects. In this instance, the medication deployed to address M.N.'s comorbid ADHD, namely Adderall, appeared to buffer the detrimental effects of olanzapine following a subtle shift in the dosing schedule. Systematic evaluation of this approach may be worthwhile for children and adolescents who are candidates for dual prescription of a psychostimulant and an atypical neuroleptic medication.

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