JOURNAL OF CHILD AND ADOLESCENT PSYCHOPHARMACOLOGY
Volume 20, Number 4, 2010

Pp. 235–236
DOI: 10.1089/cap.2010.2041

Guest Editorial

Obsessive-Compulsive Disorder and Tourette's Disorder: Where Are We Now?

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This special issue of the Journal provides an update on pediatric obsessive-compulsive disorder (OCD) and Tourette's disorder (TD): where are we now, and where are we going? Tic disorders and OCD are quite common in clinical practice; up to 20% of school-age children develop tics, and 2–4% of prepubertal children may develop OCD. There is also a bidirectional overlap of tics and OCD symptoms, in that OCD symptoms have been reported in as many as 60% of patients with TD, while patients with OCD may have a 20% lifetime risk of having tics. There are similarities in phenomenology, psychiatric co-morbidity, genetic vulnerability, and approaches to treatment in both pediatric-onset disorders. (Swain et al. 2007; Stewart et al. 2004)

Treatment includes both behavioral treatment and pharmacotherapy; often combinations of habit-reversal therapy for tics or exposure and response prevention for OCD and medications are used in clinical practice. However, to date, predictors, moderators, and mediators of treatment response are still very much needed. Deepening understanding of the neurobiology of these disorders will elucidate the biological mechanisms that are necessary to guide the development of effective and symptom specific treatments.

Another area of great interest to both practitioners and families is the role of the immune system in pediatric OCD and TD. Since 1998 when Dr. Susan Swedo described the first 50 cases of pediatric autoimmune neuropsychiatric disorders associated with streptococcus (PANDAS), a host of studies have examined this (Swedo et al. 1998). Although substantial data have accumulated, the jury is still out on the specificity of streptococcal bacteria in triggering explosive tics and/or OCD. Indeed, understanding the role of streptococcal bacteria and/or potential other infectious triggers has lagged behind the strong call of parents and pediatricians for effective treatment.

We hope that the articles in this issue, a compilation of reviews and new clinical work, will both update our readers and inspire needed studies.

To set the stage, Dr. Leckman and colleagues have provided a thoughtful review of the neurobiological substrates of TD. Considered a disorder of disinhibition of the cortico-striatal-thalamic-cortical tracts, neuropathological and imaging studies of TD have provided data on the limbic and associative circuits as well. Tics are thought to result from dysfunction in cortical and subcortical regions that are involved in habit formation, and, like habits, tics are routines that link sensory and motor phenomena. Advances in knowledge of the neurotransmitter systems, structural and functional imaging have added to our understanding of the neural circuits involved in TD, which will allow clinicians to target specific types of tics with more specific treatments.

Dr. Parraga and colleagues review pharmacotherapy for TD; an interesting historical report is included. For example, Itard (1825) described "application of leeches along the spine, and thighs... or cold river baths, ... massages and gymnastics"; Gilles de la Tourette himself in his original case reports in 1885 acknowledged tremendous difficulties in treating his patients, and reported using "isolation, tonics, hydrotherapy and static electricity. ..." We have come along way since then, but there is still a long way to go in identification of effective and safe treatments.

Dr. Wu and colleagues review the medical model of tic suppression from the perspective of pediatric neurology. Their article covers evidence for the treatment of tics in TD with a focus on tics themselves. As the majority of clinically referred children and adolescents with TD usually has one or more psychiatric co-morbid disorders as well, guidelines for evaluation of these disorders in association with tics are also suggested.

Although only two typical neuroleptics, haloperidol and pimozide, are formally approved medications to treat TD, adverse effects, such as extrapyramidal symptoms, fatigue, weight gain, and cognitive dulling may be associated. In recent years, atypical neuroleptics have been used preferentially. Dr. Kompoliti and colleagues report a study in which they compared the effects on weight and body mass index (BMI) in patients on first-generation and second-generation antipsychotics with patients who were not receiving either. Not surprising, but of concern, was higher BMI in antipsychotic-treated patients; there was no reported difference between first- and second-generation antipsychotics.

Attention-deficit/hyperactivity disorder (ADHD) is the most common co-morbid psychiatric disorder reported in clinically referred children and adolescents with TD. Treatment of these two disorders simultaneously is challenging. Although stimulants have been reported anecdotally to increase tics in some youth, there is no substantial scientific evidence that this is the case, and, in fact, studies have supported the benefit of stimulants in these children. Dr. Lyon and colleagues report on a study in which children with ADHD and TD were treated with a single-dose of a stimulant to evaluate the effect on subjects' ability to behaviorally suppress tics. Results were interesting and somewhat unexpected, providing potential pilot data on combination pharmacotherapy and behavioral therapy for tics in future studies.

Aripiprazole has gained more widespread use for TD in recent years; as a partial dopamine agonist, it has the advantage of utility

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for tic suppression, and relative tolerability compared to first-generation and other second-generation antipsychotics. As a testimony to its widespread use, Dr. Cui and colleagues report to date the largest worldwide study of aripiprazole in children and adolescents with TD. The authors report that 72 patients in an 8-week open-label study experienced a significant reduction in both tic severity and behavioral symptoms, and tolerated the drug well.

Dr. Grant and colleagues review the use of the glutamate antagonist riluzole in psychiatry generally and in childhood-onset OCD. This agent, one of the very few new treatments proposed for OCD in adults and children, may act by increasing presynaptic glutamate release. Previous magnetic resonance spectroscopy studies had suggested glutamatergic abnormalities in childhood OCD (e.g. Rosenberg and Keshevan 1998). The off-label use of this agent (it is approved for use in amyotrophic lateral sclerosis) seems promising for OCD. Some rare but disturbing adverse effects such as pancreatitis may be more common in children, and careful monitoring will be warranted if this progresses to more general use.

Dr. Geller's team provides an extensive general review of pediatric OCD, including clinical presentation, outcome, and treatment. In this thorough and balanced review, the need for more data on agents that augment therapeutic effect is made clear. There is intriguing evidence for the usefulness of atypical opiates. This is a particularly important point, since the most effective augmenting drugs are the antipsychotics with their attending greater risks in pediatric populations.

Dr. Murphy and colleagues provide an unusual review of the immunobiology of TD. It has not generally been recognized that clinical reports have linked TD/tics to infectious triggers since 1929; these reports are as convincing as the observations associating Syndenham's chorea with OCD. This article describes the continued controversy over and methodological difficulties in establishing a clear etiologic relationship between streptococcal infection and OCD/tics. There is also in depth review of the several different models of how antibodies to brain regions might cause disorder, at least for subgroups of patients. Research on the humoural responses to anti-neuronal antibodies (e.g., an increase may attack dopamine receptors or promote signal transduction leading to release of excitatory neurotransmitters) with a broad review of laboratory and clinical studies related to TD is discussed. Studies of the cellular responses (e.g., T and B lymphocyte changes) also provide several models for this disorder. The review concludes with a new model of infection-triggered tics/TD linking vulnerability to group A streptococcal (GAS) infections, dopamine, and altered immunological functioning. The article concludes with guidelines for future research covering TD and OCD, other possible infectious agents, and treatment studies monitoring biological markers.

Dr. Bernstein and colleagues report a descriptive comparison of 21 children meeting Swedo's PANDAS criteria and a matched non-PANDAS childhood OCD group. In addition to clinical features described previously (e.g., prominent tics), there was excess in urinary urgency and deteriorating handwriting as well as an excess of acute separation anxiety, for the PANDAS group, but not in the childhood OCD group. In contrast, the childhood OCD group had an excess of chronic separation anxiety. The findings of this study are supportive of previous studies of PANDAS and extend the description in an intuitively reasonable way.

We think that you will find this special issue thought provoking and informative, and helpful in clinical practice. We want to thank the authors for the breadth and depth of their reports, and the challenges that they have set forth. We are hopeful that this issue provides both an update on where we are with TD and OCD right now and where we need to go in the future.

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