# Practice Parameter for the Assessment and Treatment of Children and Adolescents With Obsessive-Compulsive Disorder

Research in etiology, neurobiology, genetics, clinical correlates, and evidence-based treatments in children and adolescents with obsessive-compulsive disorder indicate a need for the revision of the Practice Parameters for the Assessment and Treatment of Children and Adolescents with Obsessive-Compulsive Disorder first published a decade ago. The present article highlights the clinical assessment and reviews and summarizes the evidence base for treatment. Based on this evidence, specific recommendations are provided for assessment, cognitive behavioral therapy, pharmacotherapy, combined treatment, and other interventions. J. Am. Acad. Child Adolesc. Psychiatry, 2012;51(1):98–113. **Key Words:** Practice Parameter, obsessive-compulsive disorder, child and adolescent psychiatry, assessment, treatment

bsessive-compulsive disorder (OCD) is a common psychiatric disorder affecting children and adolescents and causing significant disability. In the previous decade since the Practice Parameters for the Assessment and Treatment of Children and Adolescents with Obsessive-Compulsive Disorder were published,1 knowledge of pediatric OCD has increased with large family-genetic studies; the elaboration of phenotypic dimensions; descriptions of comorbid disorders and their moderating effects on treatment response and outcome; research on immune-based neuropsychiatric causes (Pediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcus [PANDAS]); the publication of randomized controlled trials of selective serotonin reuptake inhibitors (SSRIs) and concern and scrutiny on the safety of these SSRIs in children; the first large-scale randomized controlled trials of cognitive-behavioral therapy (CBT); new approaches in behavior therapy including intensive in- and outpatient treatment, family-based treatment, group therapy, and behavioral intervention for very young children with OCD; and emerging data on the moderators and predictors of response to specific treatments. This revision of the Practice Parameters is intended to incorporate recent research and empirical clinical wisdom to guide child and adoles-

cent psychiatrists who treat children with OCD and the other medical and mental health providers involved in their care.

#### METHODOLOGY

Information and recommendations used in this Parameter were obtained from literature searches using the Medline, PubMed, PsycINFO, and Cochrane Library databases and by an iterative bibliographic exploration of articles and reviews, beginning with more inclusive and sensitive searches employing the search term "obsessivecompulsive disorder", multiple free text and relevant medical subject headings (MeSH terms), and an initial period from 1980 to the present day (749 citations). The search was narrowed using delimiters and filters such as age 0 to 18 years, English language only, human studies, published in the previous 10 years, and using the Boolean operators 'AND' clinical trial 'OR' meta-analysis, practice guideline, randomized controlled trial, review, classical article to decrease the citations to 322. Using similar strategies, obsessivecompulsive disorder AND randomized controlled trial were searched to yield 353 citations, including 11 reviews. Key quality domains were examined including descriptions of the study population (inclusion and exclusion criteria), randomization, blinding, interventions, outcomes (including "last observation carried forward" data and description of dropouts), sources of sponsorship or funding, and statistical analysis. For this Practice Parameter, 65 publications were selected for careful examination based on their weight in the hierarchy of evidence attending to the quality of individual studies, relevance to clinical practice, and the strength of the entire body of evidence.

#### **EPIDEMIOLOGY**

The high prevalence of OCD in children was not generally recognized until the first epidemiologic study just over 20 years ago. In that study, most subjects identified through screening who were later diagnosed with OCD had been previously undiagnosed, leading to the notion of pediatric OCD as a "hidden epidemic." The secretive nature of OCD symptoms and the isolated and idiosyncratic functional deficits that may be severe but variable and domain specific contribute to the finding that OCD was under-recognized and underdiagnosed in youth. Early epidemiologic studies were conducted in adolescent populations and most used school surveys for sample ascertainment. The prevalence rates of pediatric OCD are around 1% to 2% in the United States and elsewhere.<sup>2,3</sup> In the more recent British Child Mental Health Survey of more than 10,000 5- to 15-year-olds, the point prevalence was 0.25% and almost 90% of cases identified had been undetected and untreated.<sup>4</sup> There appears to be two peaks of incidence for OCD across the life span, one occurring in preadolescent children<sup>5</sup> and a later peak in young adult life (mean age, 21 years). If all pediatric cases of OCD persisted in adulthood, one would expect an increasing cumulative prevalence of OCD across the life span as more cases are added to the population. Studies have shown that this anticipated cumulative increase in prevalence is modified by the variable outcome of childhood-onset OCD, with a substantial number becoming subclinical over time.<sup>6</sup>

#### **ETIOLOGY**

#### Genetic Factors

The contribution of genetic factors to the development of OCD has been explored in twin, family-genetic, and segregation analysis stud-

ies.<sup>7–9</sup> Twin studies have shown that the concordance rates for monozygotic twins are significantly higher than for dizygotic twins. Although family studies also have consistently demonstrated that OCD is familial,7 the morbid risk of OCD in first-degree relatives appears to be greater for index cases ascertained in childhood. For example, in their multisite family study of adult OCD probands, Nestadt et al. 10 found a risk for OCD of almost 12% in first-degree relatives, whereas relatives of pediatric OCD probands showed age-corrected morbid risks from 24% to 26% in more recent studies.<sup>11</sup> Genetic linkage studies of OCD have found evidence for susceptibility loci on chromosomes 1q, 3q, 6q, 7p, 9p, 10p, and 15q.9 There is increasing evidence that glutamate receptor/modulating genes may be associated with OCD.<sup>12</sup>

#### Nongenetic Factors

Although these studies have emphasized genetic factors, they also have pointed clearly to the major effects of "nongenetic" influences on the expression of OCD. For example, twin studies have shown that, even among monozygotic twins, OCD is not fully concordant. Clearly then, nonheritable etiologic factors are as great or greater than genetic factors for the risk of developing OCD. In fact, many, if not most, cases of OCD arise without a known positive family history of the disorder, the so-called sporadic cases. Information on the environmental triggers of the disorder may be especially relevant for the sporadic form because in such cases the OCD cannot be explained by the presence of an affected relative. To date, studies have focused on the perinatal (intrauterine [including potential teratogens such as alcohol and tobacco], birth, and postnatal) experiences of affected subjects<sup>13</sup> and immune-mediated neuropsychiatric models of illness.

Perhaps no issue has been as controversial in OCD as that of PANDAS. The central hypothesis of PANDAS derives from the observations of neurobehavioral disturbance accompanying Sydenham chorea, a sequel of rheumatic fever. An immune response to group A  $\beta$ -hemolytic streptococcus (GABHS) infections purportedly leads to cross reactivity with, and inflammation of, basal ganglia, with a distinct neurobehavioral syndrome that includes OCD, tics, and perhaps hyperactivity. The diagnostic criteria were laid out by Swedo et al.,  $^{14}$  but detractors have argued

that GABHS may be but one of many nonspecific physiologic stressors that can trigger an increase in tics or OCD. <sup>15,16[ct]</sup> At this time, the epidemiologic evidence and expert clinical experience support the belief that a small subset of children with OCD and Tourette's disorder have onsets and clinical exacerbations linked to GABHS. <sup>17,18</sup>

#### CLINICAL PRESENTATION

#### Phenotype

Despite continuity in the phenotypic presentation of children and adults, issues such as limited insight and the evolution of symptom profiles that follow developmental themes over time may differentiate children from adults with OCD. 19 Symptoms of OCD are frequently hidden or poorly articulated, especially in younger children. In addition, children with OCD may display compulsions without well-defined obsessions and rituals other than the typical washing or checking (e.g., blinking and breathing rituals).<sup>20</sup> Most children exhibit multiple obsessions and compulsions (mean numbers over the lifetime have been reported as 4.0 and 4.8, respectively).20 Neither gender nor age at onset has been reported to determine the type, number, or severity of OCD symptoms. Children's obsessions often center on a fear of a catastrophic family event (e.g., death of a parent). Contamination, sexual, and somatic obsessions, and excessive scruples/guilt are the most commonly reported obsessions, and washing, repeating, checking, and ordering are the most commonly reported compulsions. 19 OCD symptoms tend to wax and wane and are persistent in most patients, changing over time so that the presenting symptom constellation is not maintained.<sup>20</sup> Efforts have been made to parse the heterogeneous symptoms of OCD into a few consistent and temporally stable symptom dimensions using factor or cluster analytic methods. The Dimensional Yale-Brown Obsessive-Compulsive Scale<sup>21</sup> measures the presence and severity of OC symptoms within several distinct dimensions that combine thematically related obsessions and compulsions.

Early-onset cases have a high frequency of subjective sensations known as "sensory phenomena" preceding or accompanying their compulsions. Physical sensations include localized tactile and musculoskeletal sensations, and mental sensations include "just-right" perceptions (to tactile, visual, and auditory sensory stimuli) and "incompleteness" (or need for accuracy).<sup>22</sup>

Pediatric OCD is characterized by a 3:2 male-to-female ratio, with more boys at younger ages. The mean age of onset of pediatric OCD ranges from 7.5 to 12.5 years (mean,  $10.3 \pm 2.5$  years) and the mean age at ascertainment ranges from 12 to 15.2 years (mean, 13.2 years), 5 documenting that, on average, the age at assessment was 2.5 years after the age at onset, a finding of considerable clinical importance. Pediatric-onset OCD is increasingly recognized as a putative developmental subtype of the disorder, based on increased familial aggregation, psychiatric comorbidity, and outcome data. 11

#### Psychiatric Comorbidity

OCD in youth is usually accompanied by another psychopathology that may complicate the assessment and treatment of affected children. Even cases derived from epidemiologic studies, which avoid the referral bias inherent in many clinical studies, have demonstrated rates of comorbid psychiatric diagnoses in more than 50% of affected children.<sup>2</sup> Irrespective of current age, a younger age at the onset of OCD predicts increased risks for comorbid attention-deficit/ hyperactivity disorder (ADHD), separation anxiety disorder, specific phobias, agoraphobia, and multiple anxiety disorders. Mood and psychotic disorders are associated with increasing chronologic age. Tourette's disorder has shown associations with age at onset (tics are more frequent in younger patients), gender (tics are more prevalent in boys), and chronologic age (tics usually improve or remit in the second decade of life).<sup>23</sup>

#### Neuropsychological Findings

Although not part of the core diagnostic symptoms, interest in a neuropsychological "endophenotype" in children with OCD has grown during recent years out of clinical and anecdotal experiences that many children have academic difficulties that are not wholly explained by their primary disorder. Given the potential involvement of frontostriatal systems in OCD, several aspects of neuropsychological performance have been especially relevant, including measurements of visuospatial integration, processing speed, short-term memory, attention, and executive function. Although not yet well characterized, deficits in visual spatial performance and processing speed appear common.<sup>24</sup>

#### CLINICAL COURSE AND OUTCOME

Precipitating psychosocial stressors have been described in several reports indicating that these are occasionally associated with the onset of OCD, sometimes dramatically.<sup>25</sup> However, most pediatric non-PANDAS OCD cases do not provide a history of clear precipitating triggers and has a subclinical onset. The long-term prognosis for pediatric OCD is better than originally conceived. Many children will remit entirely or become clinically subthreshold over time.<sup>5</sup> A younger age of OCD onset, an increased duration of OCD, inpatient treatment, and perhaps specific symptom subtypes, such as sexual, religious, or hoarding obsessions, predict greater persistence. Comorbid psychiatric illness and poor initial treatment response are adverse prognostic factors. In contrast, gender, age at assessment, and length of follow-up are not reported as predictors of remission or persistence. Psychosocial function is frequently compromised. Studies have reported high levels of social/peer problems (55–100%), isolation, unemployment (45%), and difficulties sustaining a job (20%). However, at follow-up in one study, pediatric subjects with OCD showed no difference from controls in educational achievement, with 30% to 70% having attended college.<sup>5</sup>

#### DIFFERENTIAL DIAGNOSES

#### Normal Development

Toddlers and preschoolers frequently engage in ritualistic behavior as a part of normal development. Examples include mealtime or bedtime routines that are insisted on. As a rule, they do not cause impairment in family functioning and an interruption of the rituals does not create severe distress in the child.

#### Other Psychiatric Disorders

Perhaps the most difficult differential diagnosis occurs in the context of a more pervasive developmental disorder (PDD or "spectrum" disorder). Core symptoms of these disorders include stereotypic and repetitive behaviors, a restricted and narrow range of interests, and activities that may be confused with OCD, especially in young children. A small number of children with OCD ( $\sim$ 5%) may also meet criteria for Asperger's disorder or PDD.<sup>23</sup> In addition to the core social and communication deficits that are a diagnostic

hallmark of "spectrum" disorders, the most helpful criterion for clinicians to differentiate PDD from OCD is whether symptoms are egodystonic and are associated with anxiety-driven obsessional fears. Children with PDD frequently engage in stereotypic behaviors with apparent gratification and will become upset only when their preferred activities are interrupted. Another helpful factor is whether symptoms are typical of OCD (such as washing, cleaning, or checking) from which one can infer some obsessional concern.

Another diagnostic dilemma occurs in the context of the poor insight of obsessional thoughts, which merge into overvalued ideas and even delusional thinking suggesting psychosis. In fact, insight in children with OCD is not static but varies with anxiety levels and is best assessed when anxiety is at a minimum. Although OC symptoms may rarely herald a psychotic or schizophreniform disorder in youth, especially in adolescents, other positive or negative symptoms of psychosis will usually be present or emerge to assist in the differential diagnosis, and the nature of obsessional ideation in these patients is often atypical.

Although the diagnosis of obsessive-compulsive personality disorder (OCPD) is rarely used with young children, OCPD features (defined as a pervasive pattern of preoccupation with orderliness, perfectionism, and control at the expense of flexibility and efficiency, beginning by early adulthood) are sometimes present and documented on Axis II in adolescent evaluations. Some children also demonstrate a preoccupation with minute details and facts, follow rules and regulations rigidly, adhere strongly to routines and schedules, and are inflexible, even relentless, in their thoughts or in pursuing their wishes. Although these behaviors are typically egosyntonic and insight is lacking, these children do not meet the diagnostic criteria for Asperger's disorder because they do not have core deficits of empathy and social pragmatic skills. Such children may be critical or judgmental toward others, or angry and even aggressive when events do not conform to expectations or wishes, leading to significant family disruption. Only longitudinal studies can show if these children develop OCPD later. Serotonergic medications are of limited help for such children and treatment is primarily behavioral.

### EVIDENCE BASE FOR PRACTICE PARAMETERS

In this Parameter, recommendations for best assessment and treatment practices are stated in accordance with the strength of the underlying empirical and/or clinical support, as follows:

- Clinical standard [CS] is applied to recommendations that are based on rigorous empirical evidence (e.g., meta-analyses, systematic reviews, individual randomized controlled trials) and/or overwhelming clinical consensus.
- Clinical guideline [CG] is applied to recommendations that are based on strong empirical evidence (e.g., nonrandomized controlled trials, cohort studies, case-control studies) and/or strong clinical consensus.
- Option [OP] is applied to recommendations that are based on emerging empirical evidence (e.g., uncontrolled trials or case series/reports) or clinical opinion but lack strong empirical evidence and/or strong clinical consensus.
- Not endorsed [NE] is applied to practices that are known to be ineffective or contraindicated. The strength of the empirical evidence is rated in descending order, as follows:
- Randomized controlled trial (rct) is applied to studies in which subjects are randomly assigned to two or more treatment conditions.
- Controlled trial (ct) is applied to studies in which subjects are nonrandomly assigned to two or more treatment conditions.
- Uncontrolled trial (ut) is applied to studies in which subjects are assigned to one treatment condition.
- Case series/report (cs) is applied to a case series or a case report.

#### **RECOMMENDATIONS**

Recommendation 1. The psychiatric assessment of children and adolescents should routinely screen for the presence of obsessions and/or compulsions or repetitive behaviors. [CG]

Clinicians should screen for OCD even when it is not part of the presenting complaint. Symptoms may be of mild to moderate severity, wax and wane over time, be prominent in one setting and not another, and be kept secret from others (including family). The simplest probes are those that derive from the diagnostic criteria of the *DSM-IV*: "Do you ever have repetitive, intrusive or unwanted thoughts, ideas, images or urges

that upset you or make you anxious and that you cannot suppress?" For younger children the question might be phrased, "Do you have worries that just won't go away?" It is reasonable to offer some examples at this time such as "worries about things not being clean" or "worrying that something bad might happen to you or someone you love."

For compulsions, a similar probe might be: "Do you ever have to do things over and over, even though you don't want to or you know they don't make sense, because you feel anxious or worried about something?" For younger children, the question might be phrased, "Do you do things over and over or have habits you can't stop?" Examples such as washing, checking, repeating, ordering, counting, and hoarding can be offered easily and quickly.

Sometimes adults are left to infer obsessions that are not articulated or even acknowledged by observing behaviors in their children. Examples include avoidance behaviors that imply concerns about some normal and expected activity such as entering a room or handling an object. If screening questions suggest that OC symptoms are present, clinicians should follow with more indepth assessment. The commonly employed parent-report Child Behavior Checklist<sup>26</sup> includes 8 items derived from factor analysis shown to have good sensitivity and specificity as a screen for OCD in children, <sup>27</sup> although even simple positive item scores using item 9 ("obsessions"), item 66 ("compulsions"), and item 112 ("worries") appear equally useful. The message for clinicians is that screening for OCD is straightforward and that simple probes will reveal the great majority of cases.

Recommendation 2. If screening suggests OC symptoms may be present, clinicians should fully evaluate the child using the *DSM-IV-TR* criteria and scalar assessment. [CS]

The diagnostic criteria of time occupied by OC symptoms, the level of subjective distress, and functional impairment, in addition to a standardized inventory of symptoms and a scalar assessment of severity are best captured by a reliable instrument such as the Children's Yale-Brown Obsessive Compulsive Scale (CY-BOCS).<sup>28</sup> The CY-BOCS is a 10-item anchored ordinal scale (0–4) that rates the clinical severity of the disorder by scoring the time occupied, the degree of life interference, subjective

distress, internal resistance, and degree of control for obsessions and compulsions. It has been validated for use with pediatric subjects.<sup>28</sup> The CY-BOCS also includes a symptom checklist of more than 60 symptoms of obsessions and compulsions categorized by the predominant theme involved, such as contamination, hoarding, washing, checking, etc. Scores of 8 to 15 represent mild illness, 16 to 23 moderate illness, and at least 24 severe illness. Equally important are quantitative measurements of avoidance, insight, indecisiveness, "pathologic" responsibility, doubt, and obsessional "slowness." The CY-BOCS is a clinician-administered instrument that is most informative when given to children and their parents, where a "worstreport" algorithm is likely to be most accurate.

Although the CY-BOCS is the current standard assessment tool for pediatric OCD, there are several important limitations to this scale. The first is that the avoidance rating is not included in the quantitative score of the scale, which may therefore underestimate severity when avoidance is a large part of the presenting behavior. Second, the scale is not linear. Three to 8 hours of obsessions or compulsions rates an ordinal score of 3, whereas longer than 8 hours scores a 4 (the maximum) on the scale. It is for this reason that a 25% to 40% decrease in the CY-BOCS scale score is considered a clinically significant response. Third, the heterogeneous nature of OCD is such that atypical symptoms may not be captured by the CY-BOCS symptom checklist. Examples include behaviors driven by sensory discomfort or a fear of a "transformation" into other people or of acquiring an unwanted character trait from another (an uncommon form of contamination). The mean CY-BOCS score at the ascertainment of OCD in children and adolescents in several studies was 23 (standard deviation, 6.5), indicating moderate to severe illness.<sup>5</sup>

Other OCD scales, such as the Leyton Obsessional Inventory, <sup>29</sup> and interviews that assess more broadly for internalizing symptoms (Ten Year Review of Rating Scales II: Scales for Internalizing Disorders)<sup>30</sup> and anxiety, such as the Anxiety Disorders Interview Schedule for Children, <sup>31</sup> the Pediatric Anxiety Rating Scale, <sup>32</sup> the Screen for Child Anxiety Related Disorders, <sup>33</sup> and the Multidimensional Anxiety Scale for Children, <sup>34</sup> may also be helpful.

Recommendation 3. A complete psychiatric evaluation should be performed, including information from all available sources and comprising standard elements of history and a mental state examination, with attention to the presence of commonly occurring comorbid psychiatric disorders. [CS]

Psychiatric comorbidity is the rule in youth with OCD, seen in clinically referred and epidemiologic samples and specialty and nonspecialty child psychiatry settings.<sup>35</sup> Storch et al.<sup>36[ut]</sup> found that 74% of youth with OCD met the criteria for at least one comorbid diagnosis, and those children with at least one comorbid diagnosis had a lower treatment response and remission rates with CBT compared with those without a comorbid diagnosis. The presence of disruptive behavior disorders in particular may represent a therapeutic challenge for clinicians. The identification of major depressive disorder and bipolar disorder is especially important before the initiation of an SSRI. Because certain comorbid disorders may adversely moderate the outcome of CBT and the medication treatment of pediatric OCD, careful assessment and treatment of other psychiatric disorders before and concurrent with the treatment of OCD may improve the final outcome in subjects with OCD at all ages (see Recommendation 8).

Comorbid eating disorders are infrequent in preadolescent children with OCD but become more prevalent during adolescence.35,37 In these children, medical considerations outweigh other concerns of psychopathology (except suicidality) and must be addressed and stabilized to permit mental health interventions. A "spectrum" of compulsive/impulsive habit disorders such as trichotillomania, compulsive nail biting, skin picking, and other forms of self-injury shares important features with OCD but also has important differences. Although stress may exacerbate these symptoms, they are usually not preceded by specific cognitions (obsessions), but rather a sense of tension that is general or localized. The impulsive behaviors are frequently a source of (temporary) gratification but may be followed by remorse and shame. Behavioral therapy is the mainstay of treatments for these disorders because standard SSRI medications are often less helpful. Body dysmorphic disorder usually onsets in adolescence when normal developmental pressures increase the focus on appearance and attraction among peers, but it may also begin in childhood.

## Recommendation 4. A full medical, developmental, family, and school history should be included with the psychiatric history and examination. [CG]

Family Accommodation. Children are embedded in families and, not surprisingly, families may become deeply enmeshed in their children's OCD. Parental efforts to relieve a child's anxiety may inadvertently lead to an accommodation and reinforcement of OC behaviors such as providing verbal reassurance or other "assistance" to children, for example, handling objects that children avoid such as opening doors, laundering "contaminated" clothes and linens, and even wiping children on the toilet who will not do it themselves. The very high intensity of affect and irritability displayed by some affected children engaged in ritualistic behaviors makes it difficult for parents to react with the supportive yet detached responses needed for effective behavioral management. The role of individual family members in the maintenance and management of OC symptoms is important to assess. The familial nature of anxiety disorders and OCD is an added factor in families' responses to a child with OCD. Detailed and specific questions about activities of daily living may be needed to understand the cycle of OC behaviors at home.

Medical History. Medical inquiry should focus on the CNS during a systems review with attention to trauma and neurologic symptoms (e.g., choreiform movements). Recently, attention to infection with GABHS as a potential precipitant for a PANDASassociated OCD14[ct] has increased. Inquiry of an infection with GABHS is indicated in acute and dramatic onsets or exacerbations in preadolescent patients or when a child in remission suddenly relapses. Neurologic signs, such as chorea, are evidence of rheumatic fever but may not occur for many months after infection. "Soft" neurologic signs, such as tremor and coordination difficulties on examination, are one criterion of the PANDAS diagnosis. 14,17 Antistreptococcic antibodies such as antistreptolysin O and anti-DNase B are present in most children by early adolescence, but a 0.2 log increase (doubling) in titers is considered evidence of a recent infection. Intercurrent titers may be helpful because exacerbations can be assayed with subsequent titers to detect any sudden increase in antibody levels, but a GABHS culture is the investigation of choice. Positive antistreptococcic antibody titers are not, by themselves, an indication for antibiotic treatment. At the present time, no neuroimaging procedures have been validated for the

assessment or diagnosis of OCD or related comorbid disorders.

Educational Assessment. School and educational histories provide an ecologically valid and important measurement of function and of illness severity. OC symptoms that spill into the school setting imply more anxiety, stronger compulsions, less insight, and less resistance and control. Therefore, educational impairment denoted by falling grades, the need for extra help, or special class placement indicate more urgency for treatment and could justify more aggressive interventions, including medications. Beyond this, there is increasing interest in a specific neuropsychological pattern of dysfunction that may be characteristic of pediatric OCD, evidenced by impairments in visual memory, visual organization, and processing speed. Children with evidence of this pattern often are dysgraphic, prefer reading to writing, and have stronger language than math skills. Impairments in planning complicate the generalization of CBT skills to new situations. A consideration for neuropsychological assessment, intelligence, and academic achievement testing should be high in children with OCD who are struggling at school, especially if the difficulties are chronic and not specifically associated with OCD.

## Recommendation 5. When possible, CBT is the first line treatment for mild to moderate cases of OCD in children. [CS]

Perhaps the greatest progress in the previous decade pertains to well-conducted systematic trials of CBT applied to children with OCD. Since the publication of a CBT treatment manual that operationalized and systematized this method,<sup>38</sup> numerous studies have consistently shown its acceptability and efficacy.<sup>39</sup> "Unlike other psychotherapies that have been applied, usually unsuccessfully, to OCD, cognitive behavioral treatment presents a logically consistent and compelling relationship between the disorder, the treatment, and the specified outcome."38 However, a recent survey of clinicians involved in the treatment of pediatric OCD found that only one third regularly used exposure techniques, one third "sometimes" used them, and the remaining third reported "rarely or never using" them. The protocol used by March et al. in the National Institute of Mental Health Pediatric Obsessive-Compulsive Disorder Treatment Study (POTS)<sup>40[rct]</sup> consists of 14 visits over 12 weeks spread across five phases: psychoeducation, cognitive training, mapping OCD, exposure and

response prevention (E/RP), and relapse prevention and generalization training. Except for weeks 1 and 2, when patients come twice weekly, all visits are administered once per week, last 1 hour, and include one between-visit 10-minute telephone contact scheduled during weeks 3 through 12. Each session includes a statement of goals, a review of the preceding week, a provision of new information, therapist-assisted practice, homework for the coming week, and monitoring procedures. Not infrequently, several limitations may preclude delivery of CBT as a first-line treatment option, as discussed in more detail under Recommendation 6.

Exposure and response prevention (E/RP) relies on the fact that anxiety usually attenuates after a sufficient duration of contact with a feared stimulus. 41 Repeated exposure is associated with a decreased anxiety across exposure trials, with the decrease in anxiety largely specific to the domain of exposure, until the child no longer fears contact with specifically targeted phobic stimuli. 42[ut] Adequate exposure depends on blocking the negative reinforcement effect of rituals or avoidance behavior, a process termed "response prevention". For example, a child with germ worries must not only touch "germy things" but also refrain from ritualized washing until his or her anxiety diminishes substantially. E/RP is typically implemented in a gradual fashion (sometimes termed "graded exposure"), with exposure targets under a patient's or, less desirably, a therapist's control. Different cognitive interventions have been used to provide the child with a "tool kit" to facilitate compliance with E/RP. The goals of cognitive therapy typically include increasing a sense of personal efficacy, predictability, controllability, and self-attributed likelihood of a positive outcome within E/RP tasks. Each must be individualized and must mesh with the child's cognitive abilities and developmental stage. Modeling, whether overt (the child understands that the therapist is demonstrating more appropriate or adaptive coping behaviors) or covert (the therapist informally models a behavior), may help improve compliance with in-session E/RP and generalization to between-session E/RP homework. Modeling may decrease anticipatory anxiety and provide an opportunity for practicing constructive self-talk before and during E/RP. Clinically, positive reinforcement (rewards) seems not to directly alter OCD symptoms, but rather helps to encourage exposure and so produces a noticeable, if indirect, clinical benefit. In contrast, punishment is unhelpful in the treatment of OCD.

Most CBT programs use liberal positive reinforcement for E/RP and proscribe contingency management procedures unless targeting disruptive behavior outside the domain of OCD.

Excellent CBT manuals and self-help books are available for therapists and families interested in developing mastery of these techniques, such as Talking Back to OCD: The Program that Helps Kids and Teens Say "No Way" and Parents Say "Way to Go" by John March, M.D.; Obsessive Compulsive Disorders: A Complete Guide to Getting Well and Staying Well by Fred Penzell, Ph.D.; Freeing Your Child from Obsessive Compulsive Disorder by Tamar Chansky, Ph.D.; and What to do When your Child has Obsessive Compulsive Disorder: Strategies and Solutions, by Aureen Pinto Wagner, Ph.D. These may be found on the OCD Foundation Web site resource section at www.ocfoundation.org.

In a recent meta-analysis of five randomized controlled trials of CBT (N = 161) in children with OCD, Watson and Rees<sup>39</sup> found a large mean pooled effect size of 1.45 (95% confidence interval [CI] 0.68–2.22), albeit with less precision and greater heterogeneity in CBT studies compared with pharmacotherapy trials. Several variations in delivering CBT have been studied and reported including those that use family-based approaches. 43[rct] Without question, families must be involved in the treatment of younger children with OCD, where parents control many contingencies of their daily activity.44 Another variation shown to be helpful is CBT delivered in group settings, 45[ut] where the positive elements of group therapy and CBT are combined. Intensive CBT approaches work well for children who subscribe in advance to this approach 46[ut] and may be especially useful for treatment-resistant OCD or for patients who desire a very rapid response.

### Recommendation 6. For moderate to severe OCD, medication is indicated in addition to CBT. [CS]

Although CBT is the first line of treatment in mild to moderate and, depending on the patient's and doctor's preference, even severe cases of OCD, more severe symptoms are an indication for medication, preferably added to CBT. Scores higher than 23 on the CY-BOCS or Clinical Global Impression Severity Scale of marked to severe impairment based on time occupied, subjective distress, and functional limitations provide a threshold for the consideration of drug intervention. In addition, any situation that could impede the successful delivery

of CBT should be cause for an earlier consideration of medication treatment. For example, a child may be too ill or may refuse to engage in CBT. Concurrent psychopathology, including multiple anxiety disorders, major mood disturbance and disruptive behavioral disorders, including ADHD, may decrease the acceptance of, or adherence to, CBT and may require medication in its own right. For example, a depressed adolescent with a mood-congruent anhedonic view of the future may see little point in making the effort to tolerate E/RP, and therefore major depression may mediate a poor response to CBT, leaving pharmacotherapy as the best option.<sup>36[ut]</sup> Individual and family factors also are important considerations. Poor insight into the irrational nature of the obsession and/or compulsion can lead to resistance to CBT. The need for close family involvement will make successful implementation of CBT more difficult in chaotic or nonintact families. There is a dire shortage of skilled CBT practitioners with the training to deliver the best standard of CBT in many areas, so that combined treatment or medication only may be the default treatment of first choice, even for cases with lower scalar scores and lesser degrees of impairment. Site-specific differences in CBT outcomes in the POTS<sup>40[rct]</sup> have suggested variability in the outcomes for CBT and medication alone compared with combined treatment, which is immune to said variation. This implies that, in the absence of expert CBT, the choice of combined treatment is also favored because outcomes are better even in the absence of expert CBT. In this context, informed consent is not fully "informed" without a discussion of CBT specifically and not just talk therapy, for the simple reason that outcomes with CBT alone or CBT plus medication are superior to medication alone.

Recommendation 7. SRIs are the first-line medications recommended for OCD in children and should be used according to AACAP guidelines to monitor response, tolerability, and safety. [CS]

Efficacy. The previous decade has seen rapid advances in the knowledge of the pharmacotherapy of OCD affecting children and adolescents. Clomipramine, the first agent approved for use in pediatric populations with OCD, <sup>47</sup>[rct] did not gain approval from the U.S. Food and Drug Administration (FDA) until 1989. Subsequent industry-sponsored multisite randomized controlled trials have demonstrated significant efficacy of

the SSRIs compared with placebo, including sertraline, <sup>48[rct]</sup> fluvoxamine, <sup>49[rct]</sup> fluoxetine, <sup>50[rct]</sup> and paroxetine. <sup>51[rct]</sup> Unfortunately, no comparative treatment studies have yet been performed and there is little to guide clinicians in their choice of SSRIs.

The cumulative data accrued from randomized controlled trials of pediatric OCD over the previous 10 years, involving more than 1,000 youth, are sufficient to examine the overall effect of medication treatment. A meta-analysis of all published randomized controlled medication trials in children and adolescents with OCD found an effect size (expressed as a pooled standardized mean difference for results of all studies) of 0.46 (95% CI 0.37-0.55) and showed a statistically significant difference between drug and placebo treatments (z = 9.87, p < .001).<sup>52</sup> Differences in absolute response rate (defined as ≥25% decrease in CY-BOCS scores after treatment) between an SSRI and placebo have ranged from 16% (sertraline and fluvoxamine) to 24% (fluoxetine), yielding a number needed to treat of 4 to 6. However, a multivariate regression of drug effect controlled for other variables showed that clomipramine (a nonselective SRI) was significantly superior to each of the SSRIs, whereas SSRIs were comparably effective.<sup>52</sup> In the absence of head-to-head trials, it is not clear if clomipramine is truly superior to SSRIs or, as is more likely, if the meta-analytic findings reflect the order in which the trials were done along with their methodologic rigor. Superior or not, clomipramine is generally not used as the drug of first choice for children because of its frequent adverse event profile<sup>47[rct]</sup> and concerns of monitoring potential arrhythmogenic effects.<sup>53</sup> Although highly significant statistically, the overall effect sizes of medication were modest. These statistics translate into an improved CY-BOCS score of about 6 points of drug over placebo. It is also possible that placebo response rates in OCD are lower than in other anxiety disorders. Since then, the POTS<sup>40[rct]</sup> confirmed these findings, with an effect size of 0.66 (95% CI 0.12-1.2) for sertraline, whereas a recent meta-analysis of 10 randomized controlled trials<sup>39</sup> showed an overall drug effect size of 0.48 (95% CI 0.36-0.61) and a clomipramine an effect size of 0.85 (95% CI 0.32–1.39). Although the effect size for CBT appears larger than that for medication, metaanalysis cannot determine which treatment is more effective because differences in design (e.g.,

**TABLE 1** Dosing Guidelines

	Starting Dose (mg)		
Drug	Preadolescent	Adolescent	Typical Dose Range (mg) (Mean Dose)a
Clomipramine <sup>b,c</sup>	6.25–25	25	50–200
Fluoxetine <sup>b,d</sup>	2.5–10	10–20	10–80 (25)
Sertraline <sup>b,d</sup>	12.5–25	25-50	50–200 (178)
Fluvoxamine <sup>b,c</sup>	12.5–25	25-50	50–300 (165)
Paroxetine <sup>e</sup>	2.5–10	10	10–60 (32)
Citalopram <sup>d</sup>	2.5–10	10–20	10–60

Note: aMean daily doses used in randomized controlled trials.

placebo-control versus wait-list condition) and study population, rather than differences in efficacy of interventions, could account for differences in observed effect sizes. In the POTS, <sup>40[rct]</sup> CBT alone did not differ statistically from sertraline alone on scalar outcomes but was superior for the remission rate; CBT and sertraline were better than placebo. Long-term studies are fewer but have suggested a cumulative benefit over longer periods of drug exposure with gradually decreasing scalar scores and increasing remission rates for sertraline<sup>54[ut]</sup> up to periods of 1 year.

Safety and Tolerability. In general, SSRI medications are well-tolerated medications and safer than their predecessor, the tricyclic antidepressants, especially in the setting of misuse or overdose. Titration schedules should be conservative, with modest increases from the initial dose each 3 weeks or so to allow for an improvement to manifest before aggressively increasing doses (Table 1). Patience is key to successful outcomes because it may take 12 weeks for substantial benefits to occur. Treatment is generally continued for 6 to 12 months after stabilization ("the dose that gets you well is the dose that keeps you well") and then very gradually withdrawn over several months. CBT "booster" sessions may be helpful to address any recurrence of symptoms during or after medication discontinuation and to prolong remission. Two or three relapses of at least moderate severity should lead to a consideration of longer-term treatment (years).

Clinicians should be aware of behavioral side effects that are more likely in younger children<sup>55</sup> and may be late-onset adverse effects appearing in parallel with a decrease in anxiety. In a study by Martin et al.,<sup>55</sup> peripubertal children exposed to

antidepressants were at higher risk of conversion to mania compared with adolescents and young adults. For children with anxiety disorders or mild depression, the number needed to harm (NNH) was 13 (95% CI 11-15). These side effects are sensitive to dose adjustment and the goal is to find a therapeutic window that provides an adequate clinical response but "acceptable" degrees of behavioral activation. If not achievable, then rotation to another SSRI is indicated. Black box warnings from the FDA about suicide exist for all antidepressant medications in the United States, but it should be noted that no suicides occurred in any of the pediatric randomized controlled trials of SSRIs. In the most comprehensive analysis of the extant data stratified by diagnosis, Bridge et al.56 found no statistically significant increased risk of suicidal thinking or behavior in the pooled pediatric OCD trials. The pooled absolute risk difference between SSRI- and placebo-treated youth with OCD was 0.5%, with an NNH of 200. In contrast to trials of serotonin-norepinephrine reuptake inhibitor and SSRI medications in OCD and anxiety disorders, in which the risk of a suicidal event is small to negligible, the risk of a suicidal event is notably larger in antidepressant trials, particularly for adolescents.

The use of clomipramine mandates an evaluation of the pediatric patient's medical condition and cardiac status in particular. The baseline evaluation should include a systems review and inquiry for a personal or family history of heart disease. A history of nonfebrile seizures should be noted but is not an absolute contraindication. A general pediatric examination to include auscultation of the heart and measurement of pulse and blood pressures is

<sup>&</sup>lt;sup>b</sup>Approved by the Food and Drug Administration for obsessive-compulsive disorder in children and adolescents.

<sup>&</sup>lt;sup>c</sup>Doses lower than 25 mg/day may be administered by compounding 25 mg into a 5-mL suspension.

<sup>&</sup>lt;sup>d</sup>Oral concentrate commercially available.

Oral suspension commercially available.

indicated. A baseline (pretreatment) electrocardiogram should be requested. Guidelines regarding unacceptable electrocardiographic (EKG) indices for the use (or increase) of clomipramine have been recommended by the FDA: a PR interval longer than 200 ms, a QRS interval more than 30% increased over baseline or longer than 120 ms, blood pressure greater than 140 systolic or 90 diastolic and a heart rate faster than 130 beats/min at rest.<sup>53</sup> A prolonged QTc (>450 ms) is associated with an increased risk of ventricular tachyarrhythmias and is a contraindication for clomipramine use (or further increase). Adverse events are common with clomipramine, including anticholinergic, adrenergic, and histaminergic effects (dry mouth, constipation, dizziness, postural hypotension, sweating, and sedation) that occur in up to 60% of children.<sup>47</sup>

It should be noted that very limited knowledge is available of what effects SSRIs have on brain development.<sup>57</sup>

## Recommendation 8. The modality of assigned treatment should be guided by empirical evidence on the moderators and predictors of treatment response. [CS]

Psychiatric comorbidity may have a significant influence on treatment response. One trial of children and adolescents treated with an SSRI for OCD showed that, although the response rate in the overall treated sample was high (71%), patients with comorbid ADHD, tic disorder, or oppositional defiant disorder had response rates significantly lower (56%, 53%, and 39%, respectively) than patients with OCD only (75%). <sup>58[rct]</sup> Further, comorbidity was associated with a higher rate of relapse after treatment in the total patient population (32% for no comorbidity versus 46% for at least one comorbid disorder, p =.04; 56% for at least two comorbid disorders, p <.05). More recent work has confirmed these findings. March et al.<sup>59</sup> conducted a post hoc analysis of data from the POTS<sup>40[rct]</sup> comparative treatment trial and found that those with a comorbid tic disorder failed to respond to sertraline alone and did not differ statistically from placebotreated patients, whereas the response in youth with OCD without tics replicated previously published intent-to-treat outcomes. In children with comorbid tics, sertraline was helpful only when combined with CBT, whereas CBT alone without medications remained effective. Therefore, children with comorbid tics should be assigned to CBT or combined CBT with medication as a first option.

In contrast, children with a positive first-degree family history of OCD responded far less well to CBT only compared with those without such a history and are good candidates for initial combined treatment. Although the reasons are not clear, high levels of parental accommodation may inadvertently lead to treatment resistance. However, it is difficult to disentangle behavioral factors from greater genetic loading that may manifest as a more familial form of OCD and more severe and treatment resistant illness.

In the comparative POTS, youth with lower severity scale scores, less OCD-related impairment, fewer comorbid externalizing symptoms, better insight, and lower levels of family accommodation showed greater improvement across treatment conditions (predictors of positive response) and are therefore good candidates for CBT as a first line of treatment.<sup>60[rct]</sup>

## Recommendation 9. Multimodal treatment is recommended if CBT fails to achieve a clinical response after several months or in more severe cases. [CS]

For greatest efficacy, the combination of CBT and medication is the treatment of choice and should be considered the default option for firstline treatment in moderate to severe OCD. Recommendations from the comparative treatment trial were to start treatment with CBT alone or combined CBT plus medication treatment. 40[rct] Combined treatment showed the greatest decrease in symptom scores and remission rate, with an effect size that was more or less the arithmetic sum of the component treatments (CBT = 0.97, sertraline = 0.67, combined = 1.4).Fifty-four percent of children receiving the combined treatment achieved a complete remission (defined by CY-BOCS score ≤10) and an unadjusted mean decrease of at least 10 points on the CY-BOCS. Note that this recommendation does not call for switching to medication treatment if CBT alone is unsuccessful, but rather the addition of medication to concurrent CBT. It is possible that one of the greatest benefits of medicine is to mediate better outcomes of CBT by decreasing anxiety and improving a child's ability to tolerate E/RP. Although sertraline was the medication used in the POTS, it is reasonable to extrapolate the POTS findings to other medications that have independently shown efficacy for OCD in children. Strategies for combining CBT with pharmacotherapy are outlined in the POTS method article <sup>61</sup> and the article by Storch et al. <sup>36[ut]</sup>

Recommendation 10. Medication augmentation strategies are reserved for treatment-resistant cases in which impairments are deemed moderate in at least one important domain of function despite adequate monotherapy. [OP]

Treatment Resistance. As a general principle, treatment resistant refers to a patient who has not responded to interventions known to be effective for the specific condition being treated. Applied to children with OCD, this indicates persistent and substantial OCD symptomatology in the face of adequate treatment known to be effective in childhood OCD. Experience supports at least two SRI trials as a necessary precondition to declare adequate medication therapy. Therefore, failure of adequate trials of at least two SSRIs or one SSRI and a clomipramine trial and a failure of adequately delivered CBT would constitute treatment resistance. Children should have a minimum of 10 weeks of each SSRI or clomipramine at maximum recommended or maximum tolerated doses, with no change in dose for the preceding 3 weeks. CBT nonresponders of adequate CBT would include a child who has not shown any improvement after 8 to 10 total sessions (or six to eight sessions of E/RP) or has substantial residual OC psychopathology after completing standard CBT, as detailed earlier. To summarize, the failure of at least two monotherapies and CBT is required before labeling a child as treatment resistant.

Most children, however, are not nonresponders, but rather partial responders. To meet the definition of partial response, children must have had at least 3 weeks of stable and persistent moderate (or worse) OCD symptoms at an SSRI dose equal to the maximal dose, or shown a flat dose-response curve for one-dose increment above the minimum expected starting dose, or experienced adverse effects as a result of dosage increase. Before rotating SSRI medications or implementing any of the augmentation strategies listed below, clinicians should ask themselves the following questions: Has the child received an adequate trial at or above the minimum starting dose? Has the child reached the maximum dose? Has the child been unable to tolerate a dose above his or her current dose? Has the child been

stable at his or her current dose for 3 weeks? Has the child had at least 10 weeks of treatment?

Hospitalization is infrequently indicated for OCD alone. Some children, however, require inpatient care for comorbid conditions such as severe mood instability or suicidal ideation. Typical inpatient psychiatric units and staff are not well equipped to deal with youth with OCD, whose avoidance or rituals may be misconstrued as oppositional behavior, leading to unhelpful behavioral interventions. Few highly specialized inpatient units exist to treat children with treatment-resistant OCD, where the milieu and highly trained staff provide an opportunity for intensive CBT.

Medication Augmentation Strategies. Adding clomipramine to an SSRI may be helpful. The rationale is to combine the serotonergic effects of each while minimizing adverse events across different drug classes. Fluvoxamine is the SSRI with the most synergistic effect when added to clomipramine, because of its ability to inhibit the conversion of clomipramine to desmethylclomipramine and increase the ratio in favor of the serotonergic parent compound. Even low-dose augmentation (25–75 mg/day) may be useful, but care must be taken when combining clomipramine with fluvoxamine and with CYP-450 2D6 inhibitors such as fluoxetine or paroxetine owing to potentially toxic increases in serum clomipramine levels, which must be monitored in addition to EKG indices. Other approaches for treatment resistance in pediatric OCD that are not supported by randomized controlled evidence but derive from expert opinion include the use of venlafaxine and duloxetine, which possess similar combined monoamine uptake inhibition properties to clomipramine but with fewer potential cardiovascular adverse effects.

Clonazepam has also been used in combination with SSRIs in several small open trials but should be used with caution in younger children. 62[ut] By far the most common drug augmentation strategies have employed (atypical) neuroleptics. High-quality randomized controlled trials using atypicals have been performed in adults with OCD and are summarized in a comprehensive meta-analysis by Bloch et al.,63 but no controlled data exist in children and only case reports and open trials have been reported. However, expert consensus has suggested that some children with treatment-resistant OCD may benefit from judicious neuroleptic augparticularly children with mentation,

disorders, 64[rct] poor insight, pervasive developmental disorder symptoms, and mood instability. In the adult studies, an absolute response rate difference of 21% was found in pooled data (number needed to treat [NNT] = 4.5), 63 with risperidone and haloperidol showing significant advantage over placebo and an even better response for those with a comorbid tic disorder (NNT = 2.3). Adverse events reported included sedation (NNH = 1.5-3) and weight gain (NNH not computed). This meta-analysis also suggested that at least 12 weeks of SSRI treatment was required before atypical augmentation was effective. Clinical experience indicates a minimum of two different adequate SSRI trials or an SSRI and clomipramine before atypical augmentation. To repeat, no controlled data exist for the use of atypical antipsychotics in children with OCD. In view of the great responsibility involved in prescribing atypical antipsychotic agents to minors, diligence is required in assessing efficacy and accurate safety data by practicing clinicians. Because there is a lack of a well-defined "standard of care," the dictum non nocere ("do no harm") is especially relevant. At a minimum, regular weight and adverse event monitoring should occur with baseline and follow-up assays of fasting lipid profile and serum glucose.

Novel augmentation trials also have been reported for stimulants, gabapentin, sumatriptan, pindolol, inositol, opiates, St. John's wort, and, more recently, N-acetyl cysteine and the glutamate antagonists memantine and riluzole, but none of these meet minimal standards that permit recommendation for their routine use. Putative PANDAS cases of OCD have also attracted novel and experimental treatment interventions. Antibiotic prophylaxis with penicillin failed to prevent streptococcal infections in one study but was effective in a subsequent study, with decreases in infections and OCD symptoms in the year of prophylaxis compared with the previous baseline year. 65[rct] Extant data are insufficient to meet minimal standards to recommend routine antibiotic prophylaxis for children with OCD, even when PANDAS is suspected as an etiology. Instead, standard treatments for OCD and streptococcal infections are recommended. Therapeutic plasma exchange and intravenous immunoglobulin remain experimental interventions with substantial risk and potential morbidity. D-cycloserine augmentation of CBT remains unproved in children, but a meta-analysis in adults suggests efficacy.66

## Recommendation 11. Empirically validated medication and psychosocial treatments for comorbid disorders should be considered. [CG]

Because CBT interventions for OCD are focused and time limited, additional CBT protocols that have been empirically validated for the treatment of disorders that are frequently comorbid with OCD, such as oppositional-defiant disorder and major depressive disorder, or family-based therapy for comorbid eating disorder symptoms may be incorporated into the treatment of the child to enhance outcome. Insight-oriented psychotherapy, whether delivered individually or in the family setting, has not been shown effective in remitting OCD symptoms in children and adolescents. Some children who have experienced decreased function in some important domain of life, for example, in school grades or an ability to maintain friendships or a loss of self-esteem or marked conflict at home that has disrupted primary relationships as a result of their OCD symptoms, may well benefit from supportive psychotherapy. Family therapy for conflict or dysfunction that impedes treatments aimed at the primary symptoms of OCD or for high parental levels of accommodation to the child's rituals and demands may lead to better outcomes.

Pharmacotherapy for common comorbid disorders is frequently needed. Almost no systematic data are available to guide clinicians in the management of complex cases. When present, ADHD is best addressed after the OCD has been treated, because stimulants may exacerbate anxiety and obsessions in some children. Some measurement of inattention can often be attributed to OCD symptoms and may improve as a result of treatment. Similarly, oppositional behavior may ameliorate markedly with a decrease in anxiety. However, the behavioral adverse effects of SSRIs, especially in younger children, may mimic the hyperactive impulsive symptoms of ADHD. Atomoxetine may be a useful medication in such situations, as may clomipramine, whose metabolite exerts a secondary amine noradrenergic effect. Although many children with chronic tic and Tourette's disorder require no pharmacological treatment, anxiolytic treatment aimed at anxiety and obsessional symptoms frequently ameliorates tics. Standard anti-tic medications including the  $\alpha$ agonists clonidine and guanfacine may be combined with antiobsessional medication, with blood pressure, heart rate, and EKG surveillance. The atypical antipsychotics may be especially helpful in OCD comorbid with tics, but great care is required, especially in children. Treatment of mood disorders is also often required. Medication for major depressive disorder aligns with antiobsessional treatment, but pediatric OCD that is comorbid with bipolar disorder represents one of the greatest treatment challenges in child psychiatry, because SSRIs may exacerbate manic symptoms, even at low doses. In these cases, mood stabilization is usually required before OCD can be addressed.

#### PARAMETER LIMITATIONS

AACAP Practice Parameters are developed to assist clinicians in psychiatric decision making. These Parameters are not intended to define the sole standard of care. As such, the Parameters should not be deemed inclusive of all proper methods of care nor exclusive of other methods of care directed at obtaining the desired results. The ultimate judgment regarding the care of a particular patient must be made by the clinician in light of all of the circumstances presented by the patient and his or her family, the diagnostic and treatment options available, and the available resources. &

This Parameter was developed by Daniel A. Geller, M.B.B.S., John March, M.D., and the AACAP Committee on Quality Issues (CQI): Heather J. Walter, M.D., M.P.H., and Oscar G. Bukstein, M.D., M.P.H., Co-Chairs, and R. Scott Benson, M.D., Allan Chrisman, M.D., Tiffany R. Farchione, M.D., John Hamilton, M.D., Helene Keable, M.D., Joan Kinlan, M.D., Ulrich Schoettle, M.D., Matthew Siegel, M.D., and Saundra Stock, M.D. AACAP liaison: Jennifer Medicus.

The American Academy of Child and Adolescent Psychiatry (AACAP) Practice Parameters are developed by the AACAP CQI in accordance with American Medical Association policy. Parameter development is an iterative process between the primary author(s), the CQI, topic experts, and representatives from multiple constituent groups, including the AACAP membership, relevant AACAP Committees, the AACAP Assembly of Regional Organizations, and the AACAP Council. Details of the Parameter development process can be accessed on the AACAP Web site. Responsibility for Parameter content and review rests with the author(s), the CQI, the CQI Consensus Group, and the AACAP Council.

The AACAP develops both patient-oriented and clinician-oriented Practice Parameters. Patient-oriented Parameters provide recommendations to guide clinicians toward best assessment and treatment practices.

#### **REFERENCES**

References marked with an asterisk are particularly recommended.

- American Academy of Child and Adolescent Psychiatry. Practice parameter for the assessment and treatment of children and adolescents with obsessive-compulsive disorder. J Am Acad Child Adolesc Psychiatry. 1998;37(suppl):27S-45S.
- Flament M, Whitaker A, Rapoport J, et al. Obsessive compulsive disorder in adolescence: An epidemiological study. J Am Acad Child Adolesc Psychiatry. 1988;27:764-771.

Recommendations are based on the critical appraisal of empirical evidence (when available) and clinical consensus (when not), and are graded according to the strength of the empirical and clinical support. Clinician-oriented Parameters provide clinicians with the information (stated as principles) needed to develop practice-based skills. Although empirical evidence may be available to support certain principles, principles are based primarily on clinical consensus. This Parameter is a patient-oriented Parameter.

The primary intended audience for the AACAP Practice Parameters is child and adolescent psychiatrists; however, the information contained therein may also be useful for other mental health clinicians.

The authors wish to acknowledge the following experts for their contributions to this Parameter: Marco Grados, M.D., Gregory Hanna M.D., Aude Henin Ph.D., Isobel Heyman, M.B.B.S., James Leckman, M.D., Gobriella Masi, M.D., Euripides Miguel, M.D., Janardhan Reddy, M.D., D.P.M., Maria Conceição do Rosario, M.D., Aline Sampaio, M.D., S. Evelyn Stewart, M.D., Eric Storch Ph.D., and Karen Wagner, M.D.

This Parameter was reviewed at the Member Forum at the AACAP in October 2008.

From September 2010 to May 2011, this Parameter was reviewed by a Consensus Group convened by the CQI. Consensus Group members and their constituent groups were as follows: Heather J. Walter, M.D., M.P.H., chair, Ulrich Schoettle, M.D., shepherd, and Christopher Bellonci, M.D. and Saundra Stock, M.D., members (CQI); Marco Grados, M.D., M.P.H., and Greg Hanna, M.D. (topic experts); Efrain Bleiberg, M.D., (AACAP Psychotherapy Committee); Johnny Lops, D.O., and Catherine Anne Steele, M.D. (AACAP Assembly of Regional Organizations); and Christopher Kratochvil, M.D., and Paramijit Joshi, M.D. (AACAP Council).

This Practice Parameter was approved by the AACAP Council on August 11, 2011.

This Practice Parameter supersedes the Practice Parameters for the Assessment and Treatment of Children and Adolescents With Obsessive Compulsive Disorder, published in the Journal of the American Academy of Child and Adolescent Psychiatry, 1998;37(suppl):27S-45S.

This Practice Parameter is available on the Internet (www.aacap.org).

Disclosures: Dr. Geller receives or has received research funding from Otsuka and Boehringer Inglheim and has served as a consultant for and received honoraria from Eli Lilly and Co. Dr. March receives or has received industry support from MedAvante, Pfizer, Eli Lilly and Co., Bristol-Myers Squibb, and Johnson and Johnson; royalties from Multihealth Systems, Guilford Press, and Oxford University Press; is a scientific advisor for Pfizer, Eli Lilly and Co., Scion, and Psymetrix; and has federal affiliation with the Treatment for Adolescents with Depression Study, the Child/Adolescent Anxiety Multimodal Study, the Pediatric Obsessive-Compulsive Disorder Treatment Study I and II and Junior, the Research Units on Pediatric Psychopharmacology and Psychosocial Interventions, the Child and Adolescent Psychiatry Trials Network, and K24. Dr. Walter has no financial relationships to disclose. Dr. Bukstein receives or has received research support, acted as a consultant, and/or served on a speaker's bureau for McNeil Pediatrics and Novartis Pharmaceuticals Corporation. Disclosures of potential conflicts of interest for all other individuals named earlier are provided on the AACAP Web site on the Practice Parameters page.

Correspondence to the AACAP Communications Department, 3615 Wisconsin Avenue, NW, Washington, D.C. 20016.

0890.8567/\$36.00/@2012 American Academy of Child and Adolescent Psychiatry

DOI: 10.1016/j.jaac.2011.09.019

- Apter A, Fallon TJ Jr, King RA, et al. Obsessive-compulsive characteristics: from symptoms to syndrome. J Am Acad Child Adolesc Psychiatry. 1996;35:907-912.
- Heyman I, Fombonne E, Simmons H, Ford T, Meltzer H, Goodman R. Prevalence of obsessive-compulsive disorder in the British nationwide survey of child mental health. Int Rev Psychiatry. 2003;15:178-184.
- 5. Geller D, Biederman J, Jones J, *et al.* Is juvenile obsessive compulsive disorder a developmental subtype of the disorder? A review

- of the pediatric literature. J Am Acad Child Adolesc Psychiatry. 1998;37:420-427.
- Stewart SE, Geller DA, Jenike M, et al. Long term outcome of pediatric obsessive compulsive disorder: a meta-analysis and qualitative review of the literature. Acta Psychiatr Scand. 2004; 110:4-13.
- Pauls D, Alsobrook J II, Goodman W, Rasmussen S, Leckman J. A family study of obsessive-compulsive disorder. Am J Psychiatry. 1995:152:76-84.
- Hanna G, Himle JA, Curtis GC, Gillespie B. A family study of obsessive-compulsive disorder with pediatric probands. Am J Med Genet. 2005;134:13-19.
- 9. Shugart YY, Samuels J, Willour VL, *et al.* Genomewide linkage scan for obsessive-compulsive disorder: evidence for susceptibility loci on chromosomes 3q, 7p, 1q, 15q, and 6q. Mol Psychiatry. 2006;11:763-770.
- Nestadt G, Samuels J, Bienvenu JO, et al. A family study of obsessive compulsive disorder. Arch Gen Psychiatry. 2000;57:358-363.
- \*Do Rosario-Campos MC, Leckman JF, Curi M, et al. A family study of early-onset obsessive-compulsive disorder. Am J Med Genet B Neuropsychiatr Genet. 2005;136B:92-97.
- Hanna GL, Veenstra-Vanderweele J, Cox NJ, et al. Evidence for a susceptibility locus on chromosome 10p15 in early-onset obsessive-compulsive disorder. Biol Psychiatry. 2007;62:856-862.
- Geller D, Wieland N, Carey K, et al. Perinatal factors affecting expression of obsessive compulsive disorder in children and adolescents. J Child Adolesc Psychopharmacol. 2008;18:373-379.
- \*Swedo SE, Leonard HL, Garvey M, et al. Pediatric autoimmune neuropsychiatric disorders associated with streptococcal infections: clinical description of the first 50 cases. Am J Psychiatry. 1998;155:264-271.
- 15. Kurlan R, Johnson D, Kaplan EL; Tourette Syndrome Study Group. Streptococcal infection and exacerbations of childhood tics and obsessive-compulsive symptoms: a prospective blinded cohort study. Pediatrics. 2008;121:1188-1197.
- Leckman JF, King RA, Gilbert DL, et al. Streptococcal upper respiratory tract infections and exacerbations of tic and obsessivecompulsive symptoms: a prospective longitudinal study. J Am Acad Child Adolesc Psychiatry. 2011;50:108-118.
- Swedo SE, Leonard HL, Rapoport JL. The Pediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcal Infection (PANDAS) subgroup: separating fact from fiction. Pediatrics. 2004;113:907-911.
- Leslie DL, Kozma L, Martin A, et al. Neuropsychiatric disorders associated with streptococcal infection: a case-control study among privately insured children. J Am Acad Child Adolesc Psychiatry. 2008;47:1166-1172.
- Geller D, Biederman J, Agranat A, et al. Developmental aspects of obsessive compulsive disorder: findings in children, adolescents and adults. J Nerv Ment Dis. 2001;189:471-477.
- Rettew DC, Swedo SE, Leonard HL, Lenane MC, Rapoport JL.
   Obsessions and compulsions across time in 79 children and adolescents with obsessive-compulsive disorder. J Am Acad Child Adolesc Psychiatry. 1992;31:1050-1056.
- Rosario-Campos C, Miguel EC, Quatrano S, et al. The Dimensional Yale-Brown Obsessive-Compulsive Scale (DY-BOCS): an instrument for assessing obsessive-compulsive symptom dimensions. Mol Psychiatry. 2006;11:495-504.
- Rosario-Campos MC, Leckman JF, Mercadante MT, et al. Adults with early-onset obsessive-compulsive disorder. Am J Psychiatry. 2001;158:1899-1903.
- 23. Geller D, Biederman J, Faraone SV, Bellorde CA, Kim GS, Hagermoser LM. Disentangling chronological age from age of onset in children and adolescents with obsessive compulsive disorder. Int J Neuropsychopharmacol. 2001;4:169-178.
- Andres S, Boget T, Lazaro L, et al. Neuropsychological performance in children and adolescents with obsessive-compulsive disorder and influence of clinical variables. Biol Psychiatry. 2007;61:946-951.
- 25. Lafleur DL, Petty C, Mancuso E, *et al.* Traumatic events and obsessive compulsive disorder in children and adolescents: is there a link? J Anxiety Disord. 2011;25:513-519.

- Achenbach TM. Manual for the Child Behavior Checklist 4–18 and 1991 Profile. Burlington, VT: University of Vermont Department of Psychiatry; 1991.
- Nelson EC, Hanna GL, Hudziak JJ, Botteron KN, Heath AC, Todd RD. Obsessive-Compulsive Scale of the Child Behavior Checklist: specificity, sensitivity, and predictive power. Pediatrics. 2001;108: E14.
- Scahill L, Riddle M, McSwiggin-Hardin M, et al. Children's Yale-Brown Obsessive Compulsive Scale: reliability and validity. J Am Acad Child Adolesc Psychiatry. 1997;36:844-852.
- Berg CJ, Rapoport JL, Flament M. The Leyton Obsessional Inventory-Child Version. J Am Acad Child Adolesc Psychiatry. 1986;25:84-91.
- Myers K, Winters NC. Ten-year review of rating scales. II: scales for internalizing disorders. J Am Acad Child Adolesc Psychiatry. 2002;41:634-659.
- Silverman WK, Albano AM. The Anxiety Disorders Interview Schedule for DSM-IV: Child and Parent Versions. San Antonio, TX: Psychological Corporation; 1996.
- The Research Units on Pediatric Psychopharmacology Anxiety Study Group. The Pediatric Anxiety Rating Scale (PARS): development and psychometric properties. J Am Acad Child Adolesc Psychiatry. 2002;41:1061-1069.
- Birmaher B, Khetarpal S, Brent D, et al. The Screen for Child Anxiety Related Emotional Disorders (SCARED): scale construction and psychometric characteristics. J Am Acad Child Adolesc Psychiatry. 1997;36:545-553.
- March JS, Parker JD, Sullivan K, Stallings P, Conners CK. The Multidimensional Anxiety Scale for Children (MASC): factor structure, reliability, and validity. J Am Acad Child Adolesc Psychiatry. 1997;36:554-565.
- Geller D, Biederman J, Faraone SV, et al. Clinical correlates of obsessive compulsive disorder in children and adolescents referred to specialized and non-specialized clinical settings. Depress Anxiety. 2000;11:163-168.
- Storch EA, Merlo LJ, Larson MJ, et al. Impact of comorbidity on cognitive-behavioral therapy response in pediatric obsessivecompulsive disorder. J Am Acad Child Adolesc Psychiatry. 2008;47:583-592.
- Rubenstein CS, Pigott TA, L'Heureux F, Hill JL, Murphy DL. A
  preliminary investigation of the lifetime prevalence of anorexia
  and bulimia nervosa in patients with obsessive compulsive disorder. J Clin Psychiatry. 1992;53:309-314.
- March JS, Mulle K. OCD in Children and Adolescents: A Cognitive-Behavioral Treatment Manual. New York: Guilford Press; 1998.
- Watson HJ, Rees CS. Meta-analysis of randomized, controlled treatment trials for pediatric obsessive-compulsive disorder. J Child Psychol Psychiatry. 2008;49:489-498.
- \*March J, Foa E, Gammon P, et al. Cognitive-behavior therapy, sertraline, and their combination for children and adolescents with obsessive-compulsive disorder: the Pediatric OCD Treatment Study (POTS) randomized controlled trial. JAMA. 2004;292: 1969-1976.
- Foa EB, Kozak MJ. Emotional processing of fear: exposure to corrective information. Psychol Bull. 1986;99:20-35.
- March JS, Mulle K, Herbel B. Behavioral psychotherapy for children and adolescents with obsessive-compulsive disorder: an open trial of a new protocol-driven treatment package. J Am Acad Child Adolesc Psychiatry. 1994;33:333-341.
- Barrett P, Healy-Farrell L, March JS. Cognitive-behavioral family treatment of childhood obsessive-compulsive disorder: a controlled clinical trial. J Am Acad Child Adolesc Psychiatry. 2004; 43:46-62.
- 44. Freeman JB, Choate-Summers ML, Moore PS, et al. Cognitive behavioral treatment for young children with obsessive-compulsive disorder. Biol Psychiatry. 2007;61:337-343.
- Himle JA, Rassi S, Haghighatgou H, Krone KP, Nesse RM, Abelson J. Group behavioral therapy of obsessive-compulsive disorder: seven vs. twelve-week outcomes. Depress Anxiety. 2001;13:161-165.
- Franklin M, Kozak M, Cashman L, Coles M, Rheingold A, Foa E. Cognitive-behavioral treatment of pediatric obsessive-compulsive disorder: an open clinical trial. J Am Acad Child Adolesc Psychiatry. 1998;37:412-419.

- Flament MF, Rapoport JL, Berg CJ, et al. Clomipramine treatment of childhood obsessive-compulsive disorder: a double-blind controlled study. Arch Gen Psychiatry. 1985;42:977-983.
- March JS, Biederman J, Wolkow R, et al. Sertraline in children and adolescents with obsessive-compulsive disorder: a multicenter randomized control trial. JAMA. 1998;280:1752-1756.
- Riddle MA, Reeve EA, Yaryura-Tobias JA, et al. Fluvoxamine for children and adolescents with obsessive-compulsive disorder: a randomized, controlled, multicenter trial. J Am Acad Child Adolesc Psychiatry. 2001;40:222-229.
- \*Geller DA, Hoog SL, Heiligenstein JH, et al. Fluoxetine treatment for obsessive-compulsive disorder in children and adolescents: a placebo-controlled clinical trial. J Am Acad Child Adolesc Psychiatry. 2001;40:773-779.
- Geller DA, Wagner KD, Emslie G, et al. Paroxetine treatment in children and adolescents with obsessive-compulsive disorder: a randomized, multicenter, double-blind, placebo-controlled trial. J Am Acad Child Adolesc Psychiatry. 2004;43:1387-1396.
- Geller DA, Biederman J, Stewart ES, et al. Which SSRI? A meta-analysis of pharmacotherapy trials in pediatric obsessive compulsive disorder. Am J Psychiatry. 2003;160:1919-1928.
- Biederman J. Sudden death in children treated with a tricyclic antidepressant: a commentary. J Am Acad Child Adolesc Psychiatry. 1991;30:495-497.
- Wagner KD, Cook EH, Chung H, Messig M. Remission status after long-term sertraline treatment of pediatric obsessivecompulsive disorder. J Child Adolesc Psychopharmacol. 2003; 13(suppl 1):S53-S60.
- Martin A, Young C, Leckman JF, Mukonoweshuro C, Rosenheck R, Douglas L. Age effects of antidepressant-induced manic conversion. Arch Pediatr Adolesc Med. 2004;158:773-780.
- Bridge J, Iyengar S, Salary CB, et al. Clinical Response and risk for reported suicidal ideation and suicide attempts in pediatric antidepressant treatment: a meta-analysis of randomized controlled trials. JAMA. 2007;297:1683-1696.
- 57. Ansorge MS, Morelli E, Gingrich JA. Inhibition of serotonin but not norepinephrine transport during development produces de-

- layed, persistent perturbations of emotional behaviors in mice. J Neurosci. 2008;28:199-207.
- Geller DA, Biederman J, Stewart SE, et al. Impact of comorbidity on treatment response to paroxetine in pediatric obsessive compulsive disorder: is the use of exclusion criteria empirically supported in randomized clinical trials? J Child Adolesc Psychopharmacol. 2003;13(suppl 1):S19-S29.
- March J, Franklin M, Leonard H, et al. Tics moderate treatment outcome with sertraline but not cognitive-behavior therapy in pediatric obsessive-compulsive disorder. Biol Psychiatry. 2007;61: 344-347.
- Garcia AM, Sapyta JJ, Moore PS, et al. Predictors and moderators of treatment outcome in the Pediatric Obsessive Compulsive Treatment Study (POTS I). J Am Acad Child Adolesc Psychiatry. 2010;49:1024-1033.
- Franklin M, Foa E, March JS. The pediatric obsessive-compulsive disorder treatment study: rationale, design, and methods. J Child Adolesc Psychopharmacol. 2003;13(suppl 1):S39-S51.
- Leonard HL, Topol D, Bukstein O, Hindmarsh D, Allen AJ, Swedo SE. Clonazepam as an augmenting agent in the treatment of childhood-onset obsessive-compulsive disorder. J Am Acad Child Adolesc Psychiatry. 1994;33:792-794.
- Bloch MH, Landeros-Weisenberger A, Kelmendi B, Coric V, Bracken MB, Leckman JF. A systematic review: antipsychotic augmentation with treatment refractory obsessive-compulsive disorder. Mol Psychiatry. 2006;11:622-632.
- McDougle C, Epperson C, Pelton G, Wasylink S, Price L. A double-blind placebo-controlled study of risperidone addition in serotonin-reuptake inhibitor-refractory obsessive-compulsive disorder. Arch Gen Psychiatry. 2000;57:794-801.
- Snider LA, Lougee L, Slattery M, Grant P, Swedo SE. Antibiotic prophylaxis with azithromycin or penicillin for childhood-onset neuropsychiatric disorders. Biol Psychiatry. 2005;57:788-792.
- Norberg MM, Krystal JH, Tolin DF. A meta-analysis of Dcycloserine and the facilitation of fear extinction and exposure therapy. Biol Psychiatry. 2008;63:1118-1126.