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PRACTICE PARAMETER FOR THE ASSESSMENT AND TREATMENT OF CHILDREN AND ADOLESCENTS WITH OBSESSIVE COMPULSIVE DISORDER

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ABSTRACT

Research in etiology, neurobiology, genetics, clinical correlates and evidence-based treatments in children and adolescents with Obsessive Compulsive Disorder (OCD) indicate a need for revision of practice parameters for assessment and treatment of pediatric OCD first published a decade ago. Here we highlight clinical assessment with attention to symptomatology, insight, family involvement, comorbid psychopathology and impairment as well as review and summarize the evidence base for treatment. Based on this evidence, we provide specific recommendations for assessment, cognitive behavioral therapy, pharmacotherapy, combined treatment and other interventions. **Key Words:** practice parameter, guideline, obsessive-compulsive disorder, child, adolescent, evaluation, treatment

ATTRIBUTION

AACAP practice parameters are developed by the AACAP Work Group on Quality Issues (WGQI) in accordance with American Medical Association policy. Parameter development is an iterative process between the primary author(s), the WGQI, topic experts, and representatives from multiple constituent groups, including the AACAP membership, relevant AACAP components, the AACAP Assembly of Regional Organizations, and the AACAP Council. Responsibility for parameter content and review rests with the author(s), the WGQI, the WGQI 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 treatment

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1 practices. Recommendations are based on empirical evidence (when available) and clinical
2 consensus (when not), and are graded according to the strength of the empirical and clinical
3 support. Clinician-oriented parameters provide clinicians with the information (stated as
4 principles) needed to develop practice-based skills. Although empirical evidence may be
5 available to support certain principles, principles are primarily based on expert opinion derived
6 from clinical experience. This parameter is a patient-oriented parameter.

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30 **INTRODUCTION**

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1 Obsessive Compulsive Disorder (OCD) is one of the most prevalent psychiatric disorders
2 affecting children and adolescents and is projected to be among the ten leading causes of global
3 disability by the World Health Organization.¹ Although categorized among the anxiety disorders
4 in the DSM IV-TR,² a variety of affects may drive the symptoms of OCD, which are frequently
5 hidden or poorly articulated, especially in younger children. Therefore, OCD may be
6 underdiagnosed and undertreated. In the last decade our knowledge base of pediatric OCD has
7 increased with: large scale genetic and family studies; the emergence of research on immune-
8 based neuropsychiatric causes (PANDAS); elaboration of phenotypic dimensions; understanding
9 of comorbid disorders and their moderating effects on treatment response and outcome;
10 publication of randomized controlled trials of selective serotonin reuptake inhibitors (SSRIs) in
11 children; concern and scrutiny regarding safety of these SSRIs in children; the first large scale
12 randomized controlled trials of cognitive behavioral therapy (CBT); and, new approaches in
13 behavior therapy including intensive in- and outpatient treatment, family-based treatment, group
14 therapy and behavioral intervention for very young children with OCD. As such, this revision of
15 the practice parameters is intended to incorporate recent research and empirical clinical wisdom
16 in order to guide child and adolescent psychiatrists who treat children with OCD as well as other
17 medical and mental health providers involved in their care.

18

19 **METHODOLOGY**

20 Information and recommendations used in this parameter were obtained from literature
21 searches using *Medline*, *PubMed*, *PsychINFO* and *Cochrane Library* databases and by iterative
22 bibliographic exploration of articles and reviews beginning with more inclusive and sensitive
23 searches employing the search term *Obsessive Compulsive Disorder*, multiple free text and
24 relevant medical subject headings (MeSH terms), and an initial time period from 1980 to current
25 (749 citations). We narrowed our search by using delimiters and filters such as *age 0-18 years*,
26 *English language only*, *human studies*, *published in the last 10 years*, and using the Boolean
27 operator ‘AND’ *Clinical Trial*, ‘OR’ *Meta-Analysis*, *Practice Guideline*, *Randomized Controlled*
28 *Trial*, *Review*, *Classical Article*, to reduce citations to 322. Using similar strategies we also
29 searched *Obsessive Compulsive Disorder AND randomized controlled trial* to yield 353 citations
30 including 11 reviews. For this practice parameter we selected 99 publications for careful

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1 examination based on their weight in the hierarchy of evidence attending to the quality of
2 individual studies, relevance to clinical practice and the strength of the entire body of evidence.

3

4 **HISTORICAL REVIEW**

5 The high prevalence of OCD in children was not generally recognized until the first
6 epidemiological study just over 20 years ago.³ In that study, most subjects identified through
7 screening who were later diagnosed with OCD had been previously undiagnosed, leading to the
8 notion of pediatric OCD as a “hidden epidemic.” The secretive nature of OCD symptoms and
9 isolated and idiosyncratic functional deficits that may be severe, but domain specific and
10 variable, contribute to the finding that OCD was underrecognized and underdiagnosed in youth.
11 However, because the clinical syndrome of OCD is distinct, remains fairly similar across the life
12 span, and does not suffer the ambiguities of several other psychiatric disorders affecting youth,
13 advances in clinical, translational and genetic research has been rapid. Most randomized
14 controlled SSRI trials for children with OCD have been published only in the last ten years
15 including sertraline (1998), fluoxetine (2001), fluvoxamine (2001), and paroxetine (2004). Large
16 scale and methodologically rigorous cognitive behavior therapy and comparative treatment trials
17 are even more recent (2004). Clinical phenotypes have been described in some detail using both
18 comorbid psychiatric diagnoses and dimensional approaches to “deconstruct” OCD. Underlying
19 neurological pathways have been mapped with a fair degree of the serotonin hypothesis remains
20 essentially unchallenged (but with increasing recent attention to the role of glutamate) and are
21 supported by pharmacogenomic research. Genetic and familial risk studies consistently show
22 genetic etiology and genome wide association studies are underway to identify specific regions
23 of the genome associated with the disorder.

24

25 **EPIDEMIOLOGY**

26 Earlier epidemiological studies were all conducted on adolescent populations and most
27 used school surveys for sample ascertainment. Prevalence rates of pediatric OCD are around 1%-
28 2% in the USA and elsewhere.^{4,5} In the more recent British Child Mental Health Survey of over
29 10,000 five to fifteen year olds, the point prevalence was 0.25% and almost 90% of cases
30 identified had been undetected and untreated. In this study, lower socioeconomic and intelligence
31 quotients were associated with OCD in youth.⁶ There appears to be two peaks of incidence for

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1 OCD across the life span, one occurring in pre-adolescent children⁷ and a later peak in early
2 adult life (mean age 21 years). If all pediatric cases of OCD persisted in adulthood, we would
3 expect an increasing cumulative prevalence of OCD across the life span as more cases are added
4 to the population. Studies show that this anticipated cumulative increase in prevalence is
5 modified by the variable outcome of childhood-onset OCD, with a substantial number becoming
6 subclinical over time.⁸

8 **CLINICAL PRESENTATION**

9 **Phenotype**

10 Despite continuity in the phenotypic presentation of children and adults, issues such as
11 limited insight and evolution of symptom profiles that follow developmental themes over time
12 may differentiate children from adults with OCD.^{9,10} In addition, children with OCD frequently
13 display compulsions without well-defined obsessions and symptoms other than typical washing
14 or checking rituals (e.g., blinking and breathing rituals).¹¹ The majority of children exhibit both
15 multiple obsessions and compulsions (mean number over lifetime reported as 4.0 and 4.8
16 respectively) and compulsions only without obsessions are more common in children than
17 adolescents.¹⁰ Children's limited capacity to articulate obsessional ideation underlying their
18 rituals may partly explain this finding. Neither gender nor age-at-onset has been reported to
19 determine the type, number or severity of OCD symptoms. Children's obsessions often center
20 upon fear of a catastrophic family event (e.g., death of a parent). Contamination, sexual, somatic
21 obsessions and scruples are the most commonly reported obsessions and washing, repeating,
22 checking and ordering are the most commonly reported compulsions. OCD symptoms tend to
23 wax and wane and are persistent in the majority of patients, changing over time so that the
24 presenting symptom constellation is not maintained.¹¹

26 **Gender**

27 Pediatric OCD is characterized by 3:2 male preponderance. In contrast adult samples of
28 OCD subjects report equal representation or a slight female preponderance. Adult gender
29 patterns appear in late adolescence.

31 **Age at onset**

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1 The mean age of onset of OCD ranges from 7.5 to 12.5 years (mean: 10.3 years, SD 2.5
2 years) and the mean age at ascertainment ranges from 12 to 15.2 years (mean: 13.2 years).¹²
3 Studies documented that, on average, age at assessment was 2.5 years after age at onset, a
4 finding of considerable clinical importance and consistent with the secretive nature of the
5 disorder. Although pediatric OCD is increasingly recognized as a putative developmental
6 subtype of the disorder, it remains uncertain as to whether additional sub-typing by age at onset
7 in childhood or adolescence is warranted.

8 9 **Psychiatric comorbidity**

10 OCD in youth is usually accompanied by other psychopathology that may complicate the
11 assessment and treatment of affected children. Even cases derived from epidemiological studies
12 that avoid the referral bias inherent in many clinical studies, demonstrate rates of comorbid
13 psychiatric diagnoses in over 50% of children with OCD.¹³ Irrespective of current age, an earlier
14 age at onset of OCD predicts increased risk for Attention-Deficit/Hyperactivity Disorder
15 (ADHD), Simple Phobia, Agoraphobia and multiple anxiety disorders. Mood and psychotic
16 disorders are associated with increasing chronological age and are more prevalent in older
17 subjects. Tourette's Disorder shows associations with both age at onset (tics are more frequent in
18 earlier onset cases), gender (tics are more prevalent in boys with OCD) and chronological age
19 (tics usually improve or remit in the second decade of life).

20 21 **Neuropsychological findings**

22 Although not part of the core diagnostic symptoms, interest in the neuropsychological
23 “endophenotype” of children with OCD has grown over the last several years out of clinical and
24 anecdotal experience that many children have academic difficulties that are not wholly explained
25 by their primary disorder. Given the potential involvement of frontal-striatal systems in OCD,
26 several aspects of neuropsychological performance have been especially relevant to its study,
27 especially measures of visuospatial integration, short-term memory, attention, and executive
28 functions. Although not yet well-characterized, deficits in visual spatial performance and
29 processing speed are common and clinicians should be aware that from a functional point of
30 view, academic difficulty in affected children is common.

31

1 **ETIOLOGY**

2 **Genetics**

3 The contribution of genetic factors to the development of OCD has been explored in
4 twin, family genetic and segregation analyses studies.^{14,15} Twin studies show that the
5 concordance rates for monozygotic (MZ) twins are significantly higher than for dizygotic twins.
6 While family studies also consistently demonstrate that OCD is familial,¹⁴ the morbid risk of
7 OCD in first-degree relatives appears to be greater for index cases with a childhood onset. For
8 example, in their multi-site family study of OCD, Nestadt et al.¹⁶ found a risk for OCD of around
9 12% in first degree relatives, while relatives of pediatric OCD probands have shown age-
10 corrected morbid risks from 24-26% in more recent studies.^{17,18} A genome-wide linkage scan for
11 obsessive compulsive disorder showed evidence for susceptibility loci on chromosomes 3q, 7p,
12 1q, 15q, and 6q.¹⁹

13

14 **Non genetic factors**

15 While the above studies emphasize genetic factors, they also clearly point to major
16 effects of *non-genetic* influences in the expression of OCD. For example, twin studies show that
17 even among MZ twins, OCD is *not* fully concordant. Clearly then, non-heritable etiological
18 factors are as great or greater than genetic factors for risk of developing OCD. In fact, many if
19 not most cases of OCD arise *without* a positive family history of the disorder – so called
20 “sporadic” cases. Information regarding environmental triggers of the disorder may be especially
21 relevant for the sporadic form because the OCD cannot be explained by the presence of an
22 affected relative. To date, studies have focused on perinatal (intrauterine, birth and postnatal)
23 experiences of affected subjects and immune mediated neuropsychiatric models of illness. The
24 intrauterine environment also includes exposure to potential teratogens such as alcohol and
25 tobacco.

26 Since its original description,²⁰ perhaps no issue in OCD remains as controversial as the
27 debate around Pediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcus
28 (PANDAS). The central hypothesis of PANDAS derives from observations of neurobehavioral
29 disturbance accompanying Sydenham’s chorea, a sequela of rheumatic fever. An immune
30 response to group A beta-hemolytic streptococcus (GABHS) infections purportedly leads to
31 cross reactivity with and inflammation of basal ganglia with a distinct neurobehavioral syndrome

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1 that includes OCD and tics. Diagnostic criteria laid out by Swedo et al.²¹ have been used in a
2 variety of antibiotic prophylaxis²² and immune modulating therapies such as plasmapheresis, but
3 detractors argue that GABHS is but one of many non-specific physiological stressors that can
4 trigger an increase in tics or OCD.²³ The weight of evidence at this time supports the belief that a
5 subset of children with OCD and Tourette’s syndrome have both onset and clinical exacerbations
6 linked to GABHS.

NEUROBIOLOGY

7
8
9 Several cortico-striatal-thalamic circuits have been implicated in the pathophysiology of
10 OCD and several neurotransmitter systems modulate this feedback loop including the excitatory
11 amine glutamate as well as dopamine and serotonin-containing neurons.²⁴ Pediatric imaging
12 studies appear similar to those in adults, detecting structural abnormalities in the cingulate
13 cortex, basal ganglia, and thalami of pediatric OCD patients.

CLINICAL COURSE AND OUTCOME

14
15
16 Precipitating psychosocial events are described in several reports indicating that these are
17 occasionally associated with the onset of OCD, sometimes dramatically. However, the majority
18 of pediatric OCD cases does not give a history of clear precipitating stressors and have a
19 subclinical onset. The long-term prognosis for pediatric OCD is better than originally conceived.
20 Many children will remit entirely or become clinically subthreshold over time.²⁵ Earlier age of
21 OCD onset, increased duration of OCD, inpatient treatment and perhaps specific symptom
22 subtypes such as sexual, religious or hoarding obsessions predict greater persistence. Comorbid
23 psychiatric illness and poor initial treatment response are adverse prognostic factors. In contrast,
24 gender, age at assessment, length of follow-up and year of publication are not reported as
25 predictors of remission or persistence. Psychosocial function is frequently compromised. Studies
26 report high levels of social/peer problems (55-100%), isolation, unemployment (45%) and
27 difficulties sustaining a job (20%). However, subjects are fairly well educated overall,
28 demonstrating no difference from controls, with 30-70% having attended college.

DIFFERENTIAL DIAGNOSES

Normal development

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

6 Other psychiatric disorders

7 Perhaps the most difficult differential diagnosis occurs in the context of a more pervasive
8 developmental disorder (PDD or “spectrum” disorder). Core symptoms of these disorders
9 include stereotypic and repetitive behaviors and a restricted and narrow range of interests and
10 activities that may be confused with OCD, especially in young children. A small number of
11 children with OCD (5-7%) may also meet criteria for Asperger’s syndrome or PDD. The most
12 helpful criterion for clinicians to differentiate PDD from OCD is whether symptoms are ego-
13 dystonic and are associated with anxiety-driven obsessional fears. Children with PDD engage in
14 stereotypic behaviors with apparent gratification and will only become upset when their
15 preferred activities are interrupted. While younger children with OCD may not be able to
16 articulate their concerns, evidence of anxiety is usually discernable. Another helpful
17 discriminating factor is whether symptoms are typical of OCD (such as washing, cleaning or
18 checking) from which one can infer some obsessional concern.

19 Another diagnostic dilemma occurs in the context of poor insight around obsessional
20 ideas that merges into overvalued ideation and even delusional thinking suggesting psychosis. In
21 fact insight in children with OCD is not static but varies with anxiety levels and is best assessed
22 when anxiety is at a minimum. While obsessive compulsive (OC) symptoms may herald a
23 psychotic or schizophreniform disorder in youth, especially in adolescents, other positive or
24 negative symptoms of psychosis will usually be present or emerge to assist in differential
25 diagnosis, and the nature of obsessional ideation in these patients is often atypical (for example,
26 a fear that their parent has been replaced by an alien).

28 EVIDENCE BASE FOR PRACTICE PARAMETERS

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

31

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- 1 • Minimal Standard [MS] is applied to recommendations that are based on rigorous
2 empirical evidence (such as randomized, controlled trials) and/or overwhelming clinical
3 consensus. Minimal standards apply more than 95% of the time; i.e., in almost all cases.
- 4 • Clinical Guideline [CG] is applied to recommendations that are based on strong empirical
5 evidence (such as non-randomized controlled trials) and/or strong clinical consensus.
6 Clinical guidelines apply approximately 75% of the time; i.e., in most cases.
- 7 • Option [OP] is applied to recommendations that are acceptable based on emerging
8 empirical evidence (such as uncontrolled trials or case series/reports) or clinical opinion,
9 but lack strong empirical evidence and/or strong clinical consensus.
- 10 • Not Endorsed [NE] is applied to practices that are known to be ineffective or
11 contraindicated.

12

13 The strength of the empirical evidence is rated in descending order as follows:

- 14 • Randomized, controlled trial (rct) is applied to studies where subjects are randomly
15 assigned to two or more treatment conditions
- 16 • Controlled trial (ct) is applied to studies where subjects are non-randomly assigned to two
17 or more treatment conditions
- 18 • Uncontrolled trial (ut) is applied to studies where subjects are assigned to one treatment
19 condition
- 20 • Case series/report (cs) is applied to a case series or a case report

21

22 **RECOMMENDATIONS/PRINCIPLES**

23 **Recommendation 1.** *The psychiatric assessment of children and adolescents should*
24 *routinely screen for the presence of obsessions and/or compulsions or repetitive behaviors.*

25 **[CG]**

26 Several factors indicate a need to screen for OCD even when it is not part of the
27 presenting complaint. Symptoms may be of mild to moderate severity, wax and wane over time,
28 be prominent in one setting and not another, and be kept secret from others (including family)
29 due to insight that obsessional thoughts or rituals will be considered odd or unacceptable. The
30 simplest probes are those that derive from the diagnostic criteria of the DSM IV: “Do you ever
31 have repetitive, intrusive or unwanted thoughts, ideas, images or urges that upset you or make

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1 you anxious and that you cannot suppress?” For younger children the question might be phrased,
2 “Do you have worries that just won’t go away?” It is reasonable to offer some examples at this
3 time such as “worries about things not being clean,” or “worrying that something bad might
4 happen to you or a loved one.”

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

10 Sometimes adults are left to infer obsessions from observing behaviors in their children
11 when they are not articulated or even acknowledged by the child. Examples include avoidance
12 behaviors that imply concerns about some normal and expected activity such as entering a room
13 or handling an object. If screening questions suggest that OC symptoms are present, clinicians
14 should follow with more in-depth assessment. More formalized screening scales may also be
15 employed to provide a broad anamnesis of psychological symptoms that includes OCD, such as
16 those to screen for common psychiatric disorders or the commonly employed parent report
17 CBCL²⁶ that includes 8 items derived from factor analysis shown to have good sensitivity and
18 specificity as a screen for OCD in children,^{27,28} but even simple positive item scores using item 9
19 (“obsessions”), item 66 (“compulsions *f*”) and item 112 (“worries”) were equally useful. The
20 take home message for clinicians is that screening for OCD is straightforward and that simple
21 probes will reveal the great majority of cases.

22

23 **Recommendation 2.** *If screening suggests obsessive compulsive symptoms may be*
24 *present, clinicians should fully evaluate the child using the DSM IV-TR criteria and scalar*
25 *assessment.* [CG]

26 The diagnostic criteria of 1) time occupied by OC symptoms, 2) level of subjective
27 distress and 3) functional impairment, as well as a standardized inventory of symptoms and
28 scalar assessment of severity, are best captured by a reliable instrument such as the Children’s
29 Yale-Brown Obsessive Compulsive Scale (CY-BOCS). The CY-BOCS is a 10-item anchored
30 ordinal scale (0-4) that rates the clinical severity of the disorder by scoring the time occupied (0
31 = no time, 4 = more than 8 hours per day), degree of life interference (0 = none, 4 = extreme),

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1 subjective distress (0 = none, 4 = extreme), internal resistance (0 = always, 4 = none), and degree
2 of control (0 = excellent, 4 = none) for both obsessions and compulsions. It has been validated
3 for use with pediatric subjects.²⁹ The CY-BOCS also includes a symptom checklist of over sixty
4 symptoms of obsessions and compulsions categorized by the predominant theme involved, such
5 as contamination, hoarding, washing, checking, etc. The CY-BOCS differs only slightly from the
6 adult Y-BOCS in symptoms recorded, language used, and not at all in the scoring method.
7 Scores of 8-15 represent mild illness, 16-23 moderate illness and scores ≥ 24 severe illness.
8 Equally important are quantitative measures of avoidance, insight, indecisiveness, pathological
9 responsibility, doubt and slowness. The CY-BOCS is a *clinician-administered* instrument that is
10 most informative when given to both children *and* their parents, where a “worst report”
11 algorithm is likely to be most accurate.

12 While the CY-BOCS is the current standard assessment tool for pediatric OCD, there are
13 several important limitations to this scale. The first is that the avoidance rating is not included in
14 the quantitative score of the scale (though it is assigned an ordinal score from 0-4), and thus may
15 underestimate severity when avoidance is a large part of the presenting behavior. Secondly, the
16 scale is not linear. Between three to eight hours of obsessions *or* compulsions rates an ordinal
17 score of 3 while 12 hours scores a 4 (the maximum) on the scale. It is for this reason that a 25%-
18 40% reduction in CY-BOCS scale scores is considered a clinically significant response. Finally,
19 the heterogeneous nature of OCD is such that some atypical symptoms may not be captured by
20 the CY-BOCS symptom checklist. Examples include behaviors driven by sensory discomfort,
21 fear of transformation into other people or of acquiring an unwanted character trait from another
22 (an uncommon form of contamination), and obsessional fixation on acquiring an object with
23 accompanying compulsive behaviors aimed at that goal. The mean CY-BOCS score at
24 ascertainment of child and adolescent OCD subjects in several studies was 23 (± 6.5 SD)
25 indicating moderate to severe illness.³⁰

26 As the phenotype of OCD is so variable, further efforts have been made to parse
27 heterogeneous symptoms occurring in multiple domains into a few consistent and temporally
28 stable symptom *dimensions* using factor or cluster analytic methods. The Dimensional Yale-
29 Brown Obsessive-Compulsive Scale (DY-BOCS)³¹ measures the presence and severity of OC
30 symptoms within six distinct dimensions that combine thematically related obsessions and
31 compulsions. The DY-BOCS shows excellent internal consistency of each symptom dimension

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1 and the global severity score, excellent inter-rater agreement for all component scores and high
2 correlation between self-report and expert ratings.

3
4 **Recommendation 3. *A complete psychiatric evaluation to include standard elements of***
5 ***history and a mental state exam with attention to the presence of commonly occurring***
6 ***comorbid psychiatric disorders is required to lay the groundwork for best clinical practice.***
7 ***Best estimate diagnostic formulations should include information from ALL available sources***
8 ***including the child, parents and other caregivers, teachers and other clinicians. [MS]***

9 Psychiatric comorbidity is the rule in youth with OCD, seen in both clinically referred
10 and epidemiological samples and specialty and non-specialty child psychiatry settings.³² Careful
11 consideration of the presence of comorbid psychiatric symptoms should be given in the
12 assessment and management of OCD subjects at all ages.

13 The influence of psychiatric comorbidity on response and relapse rates in children and
14 adolescents treated with paroxetine for OCD³³ showed that while the response rate to paroxetine
15 in the overall treated sample was high (71%), the response rates in patients with comorbid
16 ADHD, tic disorder, or oppositional defiant disorder (56%, 53%, and 39%, respectively) were
17 significantly less than in patients with OCD only (75%). Further, comorbidity was associated
18 with a greater rate of relapse following treatment in the total patient population (46% for ≥ 1
19 comorbid disorder ($p=0.04$) and 56% for ≥ 2 comorbid disorders ($p<0.05$) vs. 32% for no
20 comorbidity). More recent work has confirmed these findings. March et al.³⁴ conducted a post-
21 hoc analysis of data from the NIMH-funded Pediatric OCD Treatment Study (POTS)³⁵
22 comparative treatment trial and found that those with a comorbid tic disorder *failed* to respond to
23 sertraline and did not separate statistically from placebo-treated patients, while response in youth
24 with OCD but without tics replicated previously published intent-to-treat outcomes. In children
25 with tics, sertraline was *only* helpful when combined with CBT, while CBT alone without
26 medications remained effective.

27 The presence of disruptive behavior disorders in particular may represent a therapeutic
28 challenge for clinicians, especially cognitive behavioral clinicians. Storch et al.³⁶ found that 74%
29 of their youth with OCD met criteria for at least one comorbid diagnosis and those children with
30 one or more comorbid diagnoses had lower treatment response and remission rates with CBT
31 relative to those without a comorbid diagnosis. It appears clear that certain comorbid disorders

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1 may adversely impact the outcome of both CBT and medication management of pediatric OCD
2 and that assessment and treatment of other psychiatric disorders prior to and concurrent with
3 treatment of OCD may improve final outcome.

4 Less confidence can be applied to the assessment and treatment of OCD in children that
5 overlaps with several other clinically challenging disorders. Some children present clinical
6 pictures that overlap with pervasive developmental disorders. Assessment of these children may
7 be facilitated by a new scale developed by Scahill et al.³⁷, the Children's Yale-Brown Obsessive
8 Compulsive Scale modified for pervasive developmental disorders. Comorbid eating disorders
9 are quite infrequent in pre-adolescent children with OCD but become more prevalent through
10 adolescence.³⁸ In these children, medical considerations outweigh other concerns of
11 psychopathology (except suicidality) and must be addressed and stabilized to permit mental
12 health interventions.

13

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

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

28 Medical inquiry should focus on the central nervous system during systems review with
29 attention to trauma (e.g. concussion) and neurological symptoms (e.g. choreiform movements).
30 Recently, attention to infection with group A beta-hemolytic streptococcus as a potential
31 precipitant for a PANDAS-associated OCD has increased. Such an inquiry is indicated in acute

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1 and dramatic onsets or exacerbations in preadolescent patients or when a child in remission
2 suddenly relapses. Neurological signs such as chorea are evidence of rheumatic fever but may
3 not occur for many months after infection. Softer neurological signs such as tremor, coordination
4 difficulties and soft motor abnormalities on exam are one criterion of the PANDAS diagnosis.³⁹
5 When suspicious, anti-streptococcal antibodies such as Anti Streptolysin O (ASO) and Anti
6 DN’ase B titers can be helpful. Intercurrent titers are also helpful in that subsequent
7 exacerbations can be assayed to detect any sudden increase in antibody levels.

8 School and educational history provides an ecologically valid and important measure of
9 function and of illness severity. OC symptoms that spill into the school setting imply more
10 anxiety, stronger compulsions, less insight and less resistance and control. Therefore educational
11 impairment denoted by falling grades, the need for extra help or special class placement indicate
12 more urgency for treatment and could justify more aggressive interventions, including
13 medications. Beyond this, there is increasing interest in a specific neuropsychological pattern of
14 dysfunction that is (endo)phenotypic for pediatric OCD, evidenced by impairments in visual
15 memory, visual organization and processing speed. Consideration for neuropsychological
16 assessment, intelligence and academic achievement testing should be high in children with OCD
17 who are struggling at school, especially if declines are recent.

18

19 **Recommendation 5. *Cognitive behavioral therapy (CBT) is the first line treatment for***
20 ***mild to moderate cases of OCD in children.* [MS]**

21 Perhaps the greatest progress in the last decade pertains to well conducted systematic
22 trials of CBT applied to children with OCD. Since the publication of a CBT treatment manual
23 that operationalized and systematized this method,⁴⁰ numerous studies have consistently shown
24 its acceptability and efficacy.⁴¹ “Unlike other psychotherapies that have been applied usually
25 unsuccessfully to OCD, cognitive behavioral treatment (CBT) presents a logically consistent and
26 compelling relationship between the disorder, the treatment, and the specified outcome.”⁴⁰

27 Despite this, a recent survey of clinicians involved in the treatment of pediatric OCD found that
28 only one third regularly used exposure techniques, one third “sometimes” used them, and the
29 remaining third reported “rarely or never using” them. The protocol used by March and Foa in
30 the NIMH POTS study,³⁵ consists of 14 visits over 12 weeks spread across five phases: (a)
31 psychoeducation, (b) cognitive training, (c) mapping OCD, (d) exposure and response

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1 prevention, and (e) relapse prevention and generalization training. Except for Weeks 1 and 2,
2 where patients come twice weekly, all visits are administered on a once per week basis, last one
3 hour, and include one between-visit 10 minute telephone contact scheduled during Weeks 3
4 through 12. Each session includes a statement of goals, review of the preceding week, provision
5 of new information, therapist-assisted practice, homework for the coming week, and monitoring
6 procedures.

7 8 **Exposure and response prevention (E/RP)**

9 This principle relies on the fact that anxiety usually attenuates after sufficient duration of
10 contact with a feared stimulus.⁴² Repeated exposure is associated with decreased anxiety across
11 exposure trials, with anxiety reduction largely specific to the domain of exposure, until the child
12 no longer fears contact with specifically targeted phobic stimuli.⁴³ Adequate exposure depends
13 on blocking the negative reinforcement effect by rituals or avoidance behavior, a process termed
14 response prevention. For example, a child with germ worries must not only touch "germy things"
15 but also must refrain from ritualized washing until his or her anxiety diminishes substantially.
16 E/RP is typically implemented in a gradual fashion (sometimes termed graded exposure), with
17 exposure targets under patient or, less desirably, therapist control.

18 19 **Cognitive therapy**

20 A variety of cognitive interventions have been used to provide the child with a "tool kit"
21 to facilitate compliance with E/RP. The goals of cognitive therapy (CT) typically include
22 increasing a sense of personal efficacy, predictability, controllability, and self-attributed
23 likelihood of a positive outcome within E/RP tasks. Each must be individualized to match the
24 specific OCD symptoms that afflict the child, and they must mesh with the child's cognitive
25 abilities, developmental stage, and individual preference.

26 27 **Extinction**

28 Because blocking rituals or avoidance behaviors removes the negative reinforcement
29 effect of the rituals or avoidance, response prevention technically is an extinction procedure. By
30 convention, however, extinction is usually defined as the elimination of OCD-related behaviors
31 through removal of parental positive reinforcement for rituals. For example, a therapist may ask

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1 parents to refrain from gratifying the child's reassurance seeking. Extinction frequently produces
2 rapid effects but can be hard to implement when the child's behavior is bizarre or very frequent.
3 In addition, nonconsensual extinction procedures may cause unmanageable distress on the part of
4 the child, disrupt the therapeutic alliance, miss important E/RP targets that are not amenable to
5 extinction procedures, and, most important, fail to help the child internalize a strategy for
6 resisting OCD. Hence, as with E/RP, placing the extinction program under the child's control
7 usually leads to increased compliance and improved outcomes.

8

9 **Modeling and shaping**

10 Modeling, whether overt (the child understands that the therapist is demonstrating more
11 appropriate or adaptive coping behaviors) or covert (the therapist informally models a behavior),
12 may help improve compliance with in-session E/RP and generalization to between-session E/RP
13 homework. Shaping involves positively reinforcing successive approximations to a desired target
14 behavior. Modeling and shaping may reduce anticipatory anxiety and provide an opportunity for
15 practicing constructive self-talk before and during E/RP.

16

17 **Operant procedures**

18 Clinically, positive reinforcement seems not to directly alter OCD symptoms, but rather
19 helps to encourage exposure and so produces a noticeable if indirect clinical benefit. In contrast,
20 punishment (defined as imposition of an aversive event) and response-cost (defined as removal
21 of a positive event) procedures have shown themselves to be unhelpful in the treatment of OCD.
22 Most CBT programs use liberal positive reinforcement for E/RP and proscribe contingency
23 management procedures unless targeting disruptive behavior outside the domain of OCD.

24 In a recent meta-analysis of five randomized controlled trials of CBT (N=161) in children
25 with OCD, Watson and Rees⁴¹ found a large mean pooled effect size of 1.45 (95% CI = .68 to
26 2.22) albeit with less precision and greater heterogeneity in CBT studies compared with
27 pharmacotherapy trials. Site-specific differences in CBT outcomes in the POTS study³⁵ suggest
28 that expert training will improve response rates to CBT.

29 Increasingly, the central role of family (both for maintenance of pathology as well as
30 therapeutic agents) in children affected with OCD has been recognized and is reflected in both
31 broader assessment of family function and new models of treatment. Several variations in

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1 delivering CBT have been studied and reported including those that utilize family based
2 approaches.^{44,45} Without question, families must be involved in treatment of younger children
3 with OCD, where parents control many contingencies of their daily activity.⁴⁶

4 Another variation shown to be helpful is CBT delivered in group settings⁴⁷ where
5 positive elements of both group therapy and CBT are combined. Intensive CBT approaches work
6 well for children who subscribe in advance to this approach.⁴⁸ Intensive approaches may be
7 especially useful for treatment-resistant OCD or for patients who desire a very rapid response
8 and this can now be found in a few specialized intensive outpatient or residential treatment
9 centers in the USA.

10

11 **Recommendation 6. *For moderate to severe OCD, medication is indicated.* [MS]**

12 While CBT is the first line of treatment in mild to moderate cases of OCD in youth, more
13 severe symptoms are an indication for medication trials. Scores of >23 on the CY-BOCS or
14 clinical global impression severity scale (CGI-S) scores of *marked to severe impairment* based
15 on time occupied, subjective distress and functional limitations, provide a threshold for
16 consideration of drug intervention. In addition, any situation that could impede the successful
17 delivery of CBT should be cause for earlier consideration of medication treatment. Concurrent
18 psychopathology including multiple anxiety disorders, major mood disturbance and disruptive
19 behavioral disorders may reduce acceptance of, or compliance with, CBT and may require
20 medication in their own right. For example, a depressed adolescent with a mood-congruent
21 anhedonic view of the future may see little point in making the effort to tolerate E/RP and in this
22 way major depression may mediate a poor response to CBT leaving pharmacotherapy as the best
23 option. Individual and family factors also are important considerations. Poor insight into the
24 irrational nature of the obsession and/or compulsion can lead to resistance of CBT. The need for
25 close family involvement will make successful implementation of CBT more difficult in chaotic
26 or non-intact families. Finally, there is a dire shortage of skilled CBT practitioners with the
27 training to deliver the best standard of CBT.

28

29 **Recommendation 7. *SSRIs are the first line medications recommended for OCD in***
30 ***children and should be used following AACAP guidelines to monitor response, tolerability and***
31 ***safety.* [MS]**

1 **Efficacy**

2 The past decade has seen rapid advances in our knowledge of the pharmacotherapy of
3 OCD affecting children and adolescents. Clomipramine, the first agent approved for use in
4 pediatric populations with OCD, did not gain FDA approval until 1989. Subsequent industry-
5 sponsored multi-site randomized controlled trials (RCTs) have demonstrated significant efficacy
6 of the selective serotonin reuptake inhibitors (SSRIs) compared with placebo, including
7 sertraline,⁴⁹ fluvoxamine,⁵⁰ fluoxetine,⁵¹ and paroxetine.⁵² Unfortunately, no comparative
8 treatment studies have yet been performed and there is little to guide clinicians in their choice of
9 SSRIs.

10 The cumulative data accrued from RCTs of pediatric OCD over the last ten years,
11 involving over one thousand youth, are now sufficient to examine the overall effect of
12 medication treatment. One meta-analysis of all published randomized controlled medication
13 trials in children and adolescents with OCD found an effect size (ES) (expressed as a pooled
14 Standardized Mean Difference (SMD) for results of all studies) of 0.46 (95% CI=0.37-0.55) and
15 showed a highly significant difference between drug and placebo treatment ($z=9.87$, $p<0.001$).
16 Multivariate regression of drug effect controlled for other variables showed that clomipramine
17 was significantly superior to each of the SSRIs but that the other SSRIs were comparably
18 effective. Although highly significant, overall effect sizes of medication were modest. These
19 statistics translate into an improved CY-BOCS score of about 6 points of drug over placebo. It is
20 also possible that placebo response rates in OCD are lower than in other anxiety disorders. Since
21 then, the POTS study³⁶ confirmed these findings with an effect size of 0.66 (95% CI=0.12-1.2)
22 for sertraline while a recent meta-analysis of 10 RCTs⁴¹ showed an overall drug effect size of
23 0.48 (95% CI=0.36-0.61) and a clomipramine effect size of 0.85 (95% CI=0.32-1.39). Although
24 the effect size for CBT appears larger than that for medication, meta-analysis cannot determine
25 which treatment is more efficacious as differences in design (e.g. placebo control versus wait list
26 condition) and study population, rather than differences in efficacy of interventions could
27 account for differences in observed effect sizes. In the POTS study, CBT alone did not differ
28 from sertraline alone and both were better than placebo.

29

30 **Long term efficacy**

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1 Long-term studies are fewer but suggest that there is a cumulative benefit over longer
2 periods of drug exposure with gradually declining scalar scores and increasing remission rates
3 for sertraline⁵³ up to periods of one year.

4 5 **Safety and tolerability**

6 In general, SSRI medications are well-tolerated medications, and safer than their
7 predecessor TCAs, especially in the setting of misuse or overdose. Clinicians should be aware of
8 behavioral side effects that are more likely in younger children and may be a late-onset adverse
9 effect appearing in parallel with reduction in anxiety. These side effects are sensitive to dose
10 adjustment and the goal is to find a therapeutic window that provides an adequate clinical
11 response but acceptable degrees of behavioral activation. If not achievable, then rotation to
12 another SSRI is indicated. However, the risk of behavioral activation with subsequent SSRI trials
13 is increased. Black box warnings from the FDA exist for all antidepressant medications in the US
14 but it should be noted that no suicides occurred in any of the RCTs of SSRIs. In the most
15 comprehensive analysis of the extant data stratified by diagnosis, Bridge et al.⁵⁴ found no
16 statistically increased risk of suicidal thinking or behavior in the pooled pediatric OCD trials.
17 Rather, the risk appeared to be limited to subjects participating in depression studies.

18 19 **Initiating and maintenance of medication**

20 In the case of a child with OCD, informed consent should also include a discussion on the
21 value and availability of cognitive behavioral treatments that are recommended as first-line
22 interventions and should be documented in the medical record.

23 Titration schedules should be conservative, with modest increases each 3 weeks or so to
24 allow for improvement to manifest before aggressively increasing doses (Table 1).

25 26 Table 1: Dosing Guidelines

27
28 Especially for treatment of OCD, *patience is key to successful outcomes* because it may
29 take a full 12 weeks for benefits to occur. Treatment is generally continued for 6-12 months
30 following stabilization and then gradually withdrawn. CBT “booster” sessions may be helpful to

1 address any recurrence of symptoms. Two or three relapses of at least moderate severity should
2 lead to consideration of longer-term treatment.

3

4 **Recommendation 8. *Multimodal treatment is recommended if CBT fails to achieve***
5 ***clinical response after several months or in more severe cases.*** [MS]

6 For greatest efficacy, the combination of CBT with medication is the treatment of choice.
7 Recommendations from the comparative treatment trial were to start treatment with either CBT
8 alone or combined CBT plus medication treatment.³⁵ In fact, the combined treatment showed the
9 greatest decrease in symptom scores and the greatest remission rate with an effect size that was
10 more or less the arithmetic sum of the component treatments (ES Combined=1.4, CBT=0.97 and
11 sertraline =0.67). Fifty four percent of children receiving combined treatment achieved a
12 remission (defined by CY-BOCS \leq 10) and an unadjusted mean decrease of 10 points on the CY-
13 BOCS. Note that this recommendation does *not* call for switching to medication treatment if
14 CBT alone is unsuccessful, but rather the addition of medication to concurrent CBT. It is
15 possible that one of the greatest benefits of medicine is to mediate better outcomes of CBT by
16 decreasing anxiety and improving a child’s ability to tolerate E/RP. This has not been studied
17 because all RCTs of medication have understandably excluded concurrent CBT during the trial.
18 Although sertraline was the medication used in the POTS study, others have reported similar
19 combination treatment approaches with different drugs including clomipramine and fluvoxamine
20 so that it is reasonable to extrapolate the POTS study findings to other medications that have
21 independently shown efficacy for OCD in children. Strategies for combining CBT with
22 pharmacotherapy are outlined in the POTS method paper⁵⁵ and in Storch et al.³⁶

23

24 **Recommendation 9. *Medication augmentation strategies are reserved only for***
25 ***treatment resistant cases where impairments are deemed moderate in at least one important***
26 ***domain of function.*** [CG-OP]

27 **Treatment resistance**

28 As a general principle, “treatment resistant” refers to a patient who has not responded to
29 interventions known to be effective for the specific condition being treated. Applied to children
30 with OCD, this indicates persistent and *substantial* OCD symptomatology in the face of
31 *adequate* treatment *known to be effective* in childhood OCD. Such a definition requires

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1 operational criteria for (1) a threshold for persistent symptoms and (2) a definition of adequacy
2 of dose and duration of initial treatment. Persistent symptoms of at least moderate severity (e.g.
3 CY-BOCS or CGI-S of marked or severe impairment) is more useful than a pre-specified
4 percentage reduction in scalar scores such as the 25-40% reduction used in drug RCTs. Data
5 support at least two SRI trials as a necessary precondition to declare *adequate* medication
6 therapy. Therefore, failure of *adequate* trials of at least two SSRIs or one SSRI and a
7 clomipramine trial as well as a failure of *adequately* delivered CBT would constitute treatment
8 resistance. Children should have a minimum of 10 weeks of each SSRI or clomipramine at
9 maximum recommended or maximum tolerated doses, with no change in dose for the preceding
10 3 weeks. In terms of *adequate* CBT dose, if a child has not shown any improvement after 8-10
11 total sessions (or 5-6 sessions of exposure), or has substantial residual OC psychopathology after
12 completing standard CBT treatment as detailed above, they may be considered CBT
13 nonresponders. To summarize, failure of at least two monotherapies as well as CBT is required
14 prior to labeling a child as treatment resistant.

15 Most children however, are not non-responders, but rather are partial responders. To meet
16 the definition of partial response, children must have 1) had at least three weeks of stable and
17 persistent moderate (or worse) OCD symptoms at an SSRI dose equal to the maximal dose OR 2)
18 shown a flat dose-response curve for one dose increment above the minimum expected starting
19 dose OR 3) experienced adverse effects as a result of dosage increase. Before rotating SSRI
20 medications or implementing any augmentation strategies below, clinicians should ask
21 themselves the following questions: 1) has the child received an *adequate* trial at or above the
22 minimum starting dose? 2) has the child reached the maximum dose? 3) has the child been
23 unable to tolerate a dose above his or her current dose? 4) has the child been stable at his or her
24 current dose for 3 weeks? 5) has the child had at least 10 weeks of treatment?

25

26 **Medication augmentation strategies**

27 Adding clomipramine to an SSRI may be helpful. The rationale is to combine
28 serotonergic effects of each while minimizing adverse events across differing drug classes. Even
29 low dose augmentation (25-75 mg/d) may be useful but care must be taken when combining with
30 CYP-450 2 D6 inhibitors such as fluoxetine or paroxetine due to potentially toxic increases in
31 serum CMI levels, which *must* be monitored along with EKG indices. Clonazepam has also been

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1 used in combination with SSRIs in several small open and double blind trials. By far, the
2 commonest drug augmentation strategies have employed atypical neuroleptics. High quality
3 RCTs employing atypicals have been done in adults with OCD and are summarized in a
4 comprehensive meta-analysis by Bloch et al.,⁵⁶ but no controlled data exists in children and only
5 case reports and open trials are reported. However, expert consensus suggests that many children
6 with treatment resistant OCD with benefit from judicious atypical augmentation, particularly
7 children with tic disorders,⁵⁷ poor insight, pervasive developmental symptoms and mood
8 instability.

9 Novel augmentation trials are also reported for stimulants, gabapentin, sumatriptan,
10 pindolol, inositol, St. John's wort, and more recently the glutamate antagonists memantine and
11 riluzole but *none of these meet minimal standards that permit recommendation for their routine*
12 *use*. Putative PANDAS cases of OCD have also attracted novel and experimental treatment
13 interventions. Antibiotic prophylaxis with penicillin failed to prevent streptococcal infections in
14 one study but was effective in a subsequent study with reduction in both infections and OCD
15 symptoms in the year of prophylaxis compared to the previous baseline year.²² Extant data are
16 insufficient to meet minimal standards to recommend antibiotic prophylaxis for children with
17 OCD, even when PANDAS is suspected as an etiology. Instead standard treatments for both
18 OCD and streptococcal infections are recommended. Therapeutic plasma exchange remains an
19 experimental intervention with substantial risk and potential morbidity.

20

21 **Recommendation 10. *While supportive non-behavioral psychotherapy is not indicated***
22 ***for OCD, it may be helpful for the morbid sequelae of symptoms and impairment and should***
23 ***be considered as further treatment, especially when OCD has been longstanding.*** [OP]

24 Insight-oriented psychotherapy, whether delivered individually or in the family setting,
25 has not been proven effective in remitting OCD symptoms in children and adolescents.

26 Nevertheless, children who have experienced decreased function in some important domain of
27 life, for example in school grades or ability to maintain friendships, or a loss of self esteem or
28 marked conflict at home that has disrupted primary relationships as a result of their OCD
29 symptoms, may very well benefit from supportive psychotherapy. Non-CBT supportive and
30 family psychotherapy for comorbid psychopathology that impedes treatments aimed at primary
31 symptoms of OCD may lead to (mediate) better outcomes. Such a decision should be based on

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1 the whole of the child’s situation, rather than being based upon a practitioner’s allegiance to a
2 particular theoretical model of care.

3

4 **PARAMETER LIMITATIONS**

5 AACAP practice parameters are developed to assist clinicians in psychiatric decision-
6 making. These parameters are not intended to define the standard of care; nor should they be
7 deemed inclusive of all proper methods of care or exclusive of other methods of care directed at
8 obtaining the desired results. The ultimate judgment regarding the care of a particular patient
9 must be made by the clinician in light of all the circumstances presented by the patient and
10 his/her family, the diagnostic and treatment options available, and available resources.

11

1
2

Table 1: Dosing Guidelines

Drug	Starting Dose (mg)		Typical Dose Range (mg) (Mean Dose)*
	Pre-Adolescent	Adolescent	
Clomipramine †**	6.25-25	25	50-200
Fluoxetine †***	2.5-10	10-20	10-80 (25)
Sertraline †***	12.5-25	25-50	50-200 (178)
Fluvoxamine †**	12.5-25	25-50	50-300 (165)
Paroxetine ****	2.5-10	10	10-60 (32)
Citalopram ***	2.5-10	10-20	10-60

- 3 *Mean daily doses used in randomized controlled trials.
 4 † FDA approved for OCD in children and adolescents.
 5 **Doses < 25 mg/day may be administered by compounding 25mg into 5ml suspension.
 6 ***Oral concentrate commercially available.
 7 ****Oral suspension commercially available.

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