

ASHP Therapeutic Position Statement on the Appropriate Use of Medications in the Treatment of Attention-Deficit/Hyperactivity Disorder in Pediatric Patients

Statement of Position

The American Society of Health-System Pharmacists (ASHP) encourages a thorough assessment by a qualified clinician to establish the diagnosis of attention-deficit/hyperactivity disorder (ADHD) and detect all comorbid medical and neuropsychiatric conditions before initiating medication therapy.

ASHP believes that it is important to develop a systematic approach to medication therapy and supports the use of evidence-based therapy for patients with ADHD whose symptoms impair their daily activities. Diagnosing ADHD in preschoolers is difficult and medications should only rarely be used to treat preschoolers suspected of having ADHD. It is important to document usual sleeping and eating patterns before initiating medication therapy in this patient population.

The treatment of ADHD is often suboptimal and the overprescribing and underprescribing of medications to treat ADHD have been documented.¹⁻³ Barriers to appropriate medication use include fear of social stigma, concern about the risk of substance abuse, and lack of education about ADHD and its appropriate treatment. In addition, parents or guardians may have difficulty initiating or continuing therapy if they lack health insurance coverage or coordinated care with teachers and extended family.

Strong evidence from controlled clinical trials in elementary-school-age children supports the superior efficacy of stimulant therapy over nonpharmacologic interventions.⁴⁻⁷ Atomoxetine and bupropion are considered second-line agents and have been effective in placebo-controlled trials. Their use may be appropriate in select patients.^{8,9} Tricyclic antidepressants are considered second- or third-line agents because of the risk of toxicity, while clonidine and guanfacine should be considered fourth-line or as adjunctive treatment because of the lack of established efficacy as monotherapy.^{9,10}

Background

Diagnosis of ADHD. Considerable data gathered from neuroimaging studies, the long-term developmental course of ADHD, cross-national studies, and familial aggregation of ADHD (which may be genetic or environmental) and heritability have demonstrated that ADHD is a legitimate brain disorder and a valid clinical diagnosis.^{4,11} An accurate diagnosis requires information from the patient, parents, teachers, and caregivers.^{4,5,12} The diagnosis of ADHD should be made by a qualified health care professional.^{4,5} Functional impairment (i.e., inattention, hyperactivity, or impulsivity) must be present in at least two settings over six months before a diagnosis can be confirmed.^{4,13}

Examples of functional impairment include making careless mistakes, losing things necessary for tasks, and interrupting or intruding on others, all of which make social and academic success unattainable. Clinicians may need two or three visits with a child or teen, as well as assessments from multiple informants, before a diagnosis can be clearly established. ADHD-specific rating scales (e.g., Connor's rating scale) are useful clinical options for documenting and quantifying ADHD symptoms before initiation of medication therapy and during follow-up assessments.^{5,12} However, these scales were specifically designed and validated under ideal conditions, and their use in clinical practice as diagnostic tools remains unclear.^{4,5,12}

Certain patients with ADHD may suffer from inattention, such as not listening or making careless mistakes in schoolwork or on the job.¹³ Others may have hyperactivity or impulsivity. For example, they may be overly active or have great difficulty waiting their turn.¹³ It is more challenging to diagnose ADHD in girls than it is in boys, because girls are less likely to exhibit hyperactivity and aggression. Inattention and impulsiveness are the most common ADHD symptoms in girls.¹⁴

Symptoms of ADHD must be present before a child is seven years old to establish a diagnosis.¹³ Inattention, hyperactivity, and impulsivity that develop in late childhood, adolescence, or adulthood may be related to another condition (e.g., mood disorder or substance abuse).^{4,5,13} Symptoms must have been present and significant during childhood to establish a diagnosis of ADHD in adolescence or adults.¹³

It may be difficult to differentiate symptoms of ADHD from bipolar disorder, conduct disorder, or an anxiety disorder.¹⁵ In fact, patients with ADHD may have multiple coexisting conditions, including anxiety, major depression, conduct disorders, oppositional defiant disorder, Tourette's syndrome, or a learning disability.^{4,5,10,13,15} Clinicians with experience in diagnostic assessment of ADHD are best able to make an accurate and comprehensive differential diagnosis.^{4,5}

Prevalence, Course, and Health Outcomes of ADHD.

ADHD occurs in 3–10% of children age 6–12 years, and 60–80% continue to have significant symptoms into adolescence.^{4,16,17} Approximately one third to one half of children will have significant symptoms into adulthood.^{4,16} Children with multiple comorbid conditions, particularly conduct disorder or a mood disorder, have a significantly greater risk of ADHD symptom persistence than do children with uncomplicated ADHD.^{15,16} Upward of 50% of children and adolescents with ADHD have at least one comorbid condition.^{10,15,16}

The predominant symptoms of ADHD may change over the patient's life span. For example, children between five and nine years of age are more likely to exhibit motor hyperactivity than do adolescents, who tend to display

impulsivity and inattention.^{4,5,13} The predominant symptoms of adult ADHD include severe disorganization, the inability to complete tasks, and difficulty with professional and interpersonal relationships.^{10,15,16}

Patients with ADHD are more likely to have adverse educational, social, and health outcomes than are patients without the disorder.^{15,17} High-school dropout rates for adolescents with ADHD are approximately 35%, compared with the national average of 5%.¹⁵ Children with ADHD have higher accidental injury rates and three times the risk of accidental poisoning. The rate of sexually transmitted disease was four times higher in ADHD patients versus similar patients without ADHD.¹⁵ Substance abuse is at least twice as likely in patients with ADHD than in those without the disorder.^{15,18} Health care costs for children with ADHD are double the cost for children without the disorder.^{15,19} Increased costs are attributed to more inpatient and outpatient hospital and emergency department visits and the cost of drugs.^{15,19}

Efficacy of Medications. Drug therapy is appropriate for patients with ADHD who suffer moderate to severe functional impairment.^{7,9,10} Evidence from at least 35 randomized controlled trials involving over 3500 children and adolescents yields a large amount of evidence-based support for stimulant therapy.^{7,9} Atomoxetine has been studied in five randomized controlled trials that included a total of 800 children and adolescents.⁸ Its efficacy in treating ADHD has been demonstrated, and it is the only nonstimulant that has received FDA-approved labeling for this indication. At least six studies involving approximately 300 children and adults have shown that tricyclic antidepressants are effective for treatment of ADHD.^{9,10} Bupropion has been evaluated in three randomized controlled studies of approximately 200 children and teens and significantly improved ADHD symptoms in these patients.^{9,10}

Several reviews and meta-analyses document the short-term efficacy of methylphenidate, dexamethylphenidate, dextroamphetamine, mixed amphetamine salts, and pemoline in reducing the core symptoms of inattention, impulsivity, and hyperactivity associated with ADHD (Table 1).^{2,7,20} Sixty to eighty percent of adolescents and adults and at least 80% of children will respond to stimulants when initiated and adjusted appropriately. There is no evidence showing that one stimulant has superior efficacy over another. Because of the risk of hepatotoxicity, pemoline should be used only if all other stimulant and nonstimulant preparations are ineffective.^{7,9,10}

The largest controlled trial in children with ADHD, the multimodal treatment study of ADHD (MTA), included 579 children 7–10 years old who were randomized to receive medication (methylphenidate) alone, medication and behavior management, behavior management alone, or standard community care over 14 months.⁶ Children treated with medication alone or medication and behavior management showed a marked reduction in core ADHD symptoms compared with children in the behavior management alone and community care groups.⁶

A study of 103 children age 7–10 years showed that significant benefits of methylphenidate for ADHD were stable over two years and that multimodal psychosocial treatment, including behavioral interventions and parent training, did not lead to superior functioning or facilitate the

discontinuation of methylphenidate.²¹ Stimulants are effective in reducing the core symptoms of ADHD for a majority of children, even improving short-term social and academic outcomes. However, the long-term (over two years) effects of stimulant therapy for patients with ADHD are unclear, and quality of life outcome studies are needed to determine these effects.^{4,7,15,21}

The onset of therapeutic efficacy of a stimulant occurs during the absorptive phase, within 30 minutes to an hour of an effective dose of an immediate-release preparation and within one to two hours of an extended-release formulation. If one stimulant is ineffective, another stimulant may be effective and should be used as long as tolerability is good.^{7,10} Dosing of stimulants is based on clinical response and the patient's weight. For example, the starting dose of methylphenidate is 5 mg twice daily (after breakfast and lunch), while dextroamphetamine or mixed amphetamine salts may be initiated at 2.5 mg every morning with a second daily dose added midday, if needed, to cover symptoms. The MTA demonstrated added therapeutic benefit when children were given a third dose of methylphenidate (usually half of the morning dose) around 3 p.m.⁶ Dosage adjustment is recommended, keeping in mind that children weighing less than 25 kg should not exceed half of the recommended maximum daily dose of a stimulant.^{5-7,20}

Newer, long-acting preparations of methylphenidate and mixed amphetamine salts are convenient formulations because they cover symptoms throughout the day after a single morning dose. These preparations are comparable in efficacy to immediate-release formulations for most individuals, although they are significantly more expensive.^{7,10,20}

Stimulants should be considered first-line therapy for ADHD unless the patient has a history of substance abuse, bipolar disorder, or an active psychotic disorder. Alternative therapies have therapeutic advantages in these patients because stimulants can worsen psychosis^{7,22} and have greater abuse potential in patients with an active substance abuse disorder.⁷ In addition to stimulants, current evidence supports the use of three other medications for ADHD: atomoxetine, bupropion, and tricyclic antidepressants (Table 2).⁸⁻¹⁰ Atomoxetine and bupropion are appropriate therapies if substance abuse is ongoing, a mood stabilizer is appropriate if bipolar disorder is present, and an atypical antipsychotic may be appropriate if the patient has a psychotic disorder or exhibits severe aggression.^{9,22,23} Atomoxetine is a selective norepinephrine-reuptake inhibitor with FDA-approved labeling for the treatment of ADHD in children, adolescents, and adults. Several placebo-controlled, short-term trials (6–12 weeks) have shown that atomoxetine is effective in reducing ADHD symptoms.⁸ It is not clear whether atomoxetine is as effective as stimulants, although one preliminary open-label study suggested comparable efficacy with methylphenidate.⁸ Controlled trials of tricyclic antidepressants versus stimulants in children have shown either no difference in response or slightly better results with stimulant therapy.^{4,9,10} Bupropion has demonstrated efficacy in placebo-controlled trials and in one small controlled trial versus methylphenidate.^{10,23} Both bupropion and tricyclic antidepressants should be initiated in divided doses, starting in the morning, and adjusted as necessary.^{9,10} Atomoxetine may be administered once or twice daily, with dosage adjustments as necessary.⁸ Symptoms of ADHD usu-

Table 1.
Stimulants Used in the Treatment of Attention-Deficit/Hyperactivity Disorder for Children Age Six Years or Older^{7,10,21,a}

Drug	Brand Name(s)	Duration of Effect (hr)	Usual Initial Dosage	Usual Dosage Range	Maximum Daily Dose	Adverse Effects
Methylphenidate Immediate release	Ritalin, Methylin	3–5	5 mg b.i.d., ^{b,c} increase by 5–10 mg per dose at weekly intervals	5–20 mg b.i.d.–t.i.d. ^b	60 mg	Nausea, stomach pain, anorexia, increased pulse, insomnia, growth effects, emergence of tics and zombie-like or robotic behavior, psychosis Same as above
Extended release	Ritalin SR, Metadate ER, Methylin ER	3–8	Doses should correspond to the immediate-release dose required over 8 hr	20–40 mg daily or 40 mg every a.m. and 20 mg in early p.m.	60 mg	Same as above
Long acting	Ritalin LA, Metadate CD	8–12	20 mg every a.m., increase by 10 mg at weekly intervals	20–40 mg every a.m.	60 mg	Same as above
	Concerta	8–12	18 mg every a.m., increase by 18 mg at weekly intervals	18–54 mg every a.m.	72 mg	Same as above, risk of obstruction in pts. with GI tract narrowing because Concerta is a nondeformable tablet; pts. and families should be told that tablet may appear in stool
Dexamethylphenidate	Focalin	3–5	2.5 mg b.i.d., ^b increase by 2.5–5 mg per dose at weekly intervals	5–10 mg b.i.d.	20 mg	Same as for methylphenidate, no clinical advantage and causes fewer headaches but more stomach pain compared with methylphenidate
Dextroamphetamine Short acting	Dexedrine, DextroStat	4–6	2.5 mg b.i.d., ^b increase by 2.5–5 mg per dose at weekly intervals	5–15 mg b.i.d. or 5–10 mg t.i.d.	40 mg	Similar to methylphenidate, may cause more GI adverse effects or insomnia in some children
Intermediate acting	Dexedrine Spansule	5–8	5 mg every a.m. to b.i.d., ^d increase by 5 mg per dose at weekly intervals	5–30 mg daily or 5–15 mg b.i.d.	40 mg	Same as for short-acting dextroamphetamine
Mixed amphetamine salts Intermediate acting	Adderall	5–7	2.5 mg every a.m. ^e to b.i.d., increase by 2.5–5 mg per dose at weekly intervals	5–30 mg daily or 5–15 mg b.i.d.	40 mg	Similar to methylphenidate and dextroamphetamine
Long acting	Adderall XR	8–12	10 mg every a.m., increase by 5–10 mg at weekly intervals	10–30 mg daily	30 mg	Similar to methylphenidate and dextroamphetamine
Pemoline	Cylert	12–24	37.5 mg every a.m., increase by 18.75–37.5 mg at weekly intervals	56.25–75 mg daily	112.5 mg	Last-resort stimulant, risk of hepatotoxicity, biweekly liver function monitoring required during use

^aAlthough stimulant dosing is not based on weight, the following usual therapeutic dosage ranges for children are useful for assessing the appropriateness of the initial dose: methylphenidate 5 mg daily, b.i.d., or t.i.d. for patients ≤ 25 kg and 5–10 mg daily, b.i.d., or t.i.d. for patients > 25 kg; dexamethylphenidate, dextroamphetamine, and mixed amphetamine salts 2.5 mg daily or b.i.d. for patients < 25 kg. GI = gastrointestinal.

^bDoses are usually taken with breakfast and lunch.

^cA third dose can be given in the evening at the clinician's discretion.

^dSpansule may be sprinkled on food.

^eTablets can be cut in half.

Table 2.
Nonstimulants Used in the Treatment of Attention-Deficit/Hyperactivity Disorder in Children⁸⁻¹⁰

Drug	Brand Name(s)	Place in Therapy	Usual Onset of Effect (wk)	Dosage Range	Adverse Effects
Atomoxetine	Strattera	Second line (first line in pts. who cannot take a stimulant due to an active substance abuse disorder or prior adverse effect)	2-4	<70 kg: 0.5 mg/kg/day, increase after a minimum of 3 days to 1.2 mg/kg/day; ≥70 kg: 20-40 mg/day, increase after a minimum of 3 days as tolerated and p.r.n. up to 100 mg/day ^a	Reduced appetite, stomach pain, nausea, vomiting, weight loss, sedation, dizziness, insomnia; monitor for increases in blood pressure and pulse
Bupropion	Wellbutrin, Wellbutrin SR, Wellbutrin XL	Second line	2-4	3 mg/kg/day at end of week 1; may increase over 3 wk to 6 mg/kg/day or 300 mg/day, whichever is smaller (divided b.i.d.-t.i.d. for immediate-acting formulation; divided b.i.d. for sustained-release formulation ^b)	Nausea, insomnia, rash, tics, dry mouth, agitation, headache, constipation, tremor, weight gain, increased risk of seizures; maximum doses: 150 mg for immediate release, 200 mg for SR, and 450 mg for XL
Tricyclic antidepressants					
Imipramine	Tofranil	Second or third line	2-4	1 mg/kg/day, increase by 1 mg/kg weekly to a maximum of 4 mg/kg/day	Dry mouth, dizziness, constipation, sedation; ECG ^c monitoring required at baseline and follow-up
Desipramine	Norpramin	Second or third line	2-4	Same as for imipramine	Same as for imipramine; close monitoring if > 3 mg/kg/day
Nortriptyline	Aventyl, Pamelor	Second or third line	2-4	0.5 mg/kg/day, increase by 0.5 mg/kg weekly to a maximum of 2.5 mg/kg/day	Same as for imipramine
Alpha-agonists					
Clonidine	Catapres	Adjunct therapy or fourth-line treatment	2-8	0.05 mg b.i.d.-t.i.d., increase by 0.05 mg weekly to a target range of 0.1-0.4 mg/day; clonidine syrup can be compounded	Sedation, irritability, drop in blood pressure, sleep disturbances, dry mouth, constipation, dizziness, ECG monitoring recommended but controversial, vital sign monitoring recommended
Guanfacine	Tenex	Adjunct therapy or fourth-line treatment	2-8	0.5 mg q.d. or b.i.d., increase by 0.5 mg weekly to a target range of 1-4 mg/day	Same as for clonidine

^aPoor metabolizers may require lower dosages.

^bThe typical starting dosage is 37.5 mg b.i.d. for the immediate-release formulation and 100 mg daily for the sustained-release formulation. The sustained-release formulation can be given once daily or it can be given as 50 mg twice a day.

^cECG = electrocardiogram.

ally improve within one to two weeks of therapy with an effective dosage, although an adequate trial may take up to two months.^{8,23} Antidepressants with predominant serotonergic activity, such as selective serotonin-reuptake inhibitors, are ineffective in treating ADHD.^{9,10,23}

Limited studies have shown greater efficacy of the α_2 -agonists clonidine and guanfacine versus placebo in reducing symptoms of ADHD.^{9,10} However, the data for clonidine and guanfacine are not compelling.^{9,23} Clonidine or guanfacine may be considered as an adjunct to stimulants for a sleep aid or for controlling aggression or if monotherapy trials of two different stimulants, bupropion, atomoxetine, and a tricyclic antidepressant fail.^{24,25}

Evidence of Stimulant Underuse, Overuse, and Risk of Abuse. The literature documents widespread variability in the rates of ADHD diagnosis and stimulant prescription patterns across the United States. One study found that 5.1% of children and teens across four major U.S. cities met the full diagnostic criteria for ADHD, yet only 12.5% of these children received treatment with a stimulant.¹ In contrast, a rural community survey found that 72% of children receiving stimulant therapy did not meet the diagnostic criteria for ADHD. For the majority of children, parents did not report ADHD symptoms.² Suboptimal treatment of ADHD has been documented across several naturalistic, community-based surveys and studies.^{2,3,15} Lack of health care access and insurance coverage for diagnostic assessment and follow-up treatment has been cited as a major reason for less-than-optimal care.^{2,9,15}

Stimulants are the most frequently prescribed psychotropics for patients younger than 18 years, and prescriptions for stimulants have increased dramatically.²⁶ In 1996, more than 10 million prescriptions were written for methylphenidate, compared with approximately 3 million prescriptions for methylphenidate in 1993.⁷ Stimulant prescribing has increased for all age groups.²⁶ For example, the rate of stimulant prescriptions for preschoolers tripled between 1991 and 1995.²⁶

Because of this increase in stimulant prescribing, parents, teachers, health care providers, and the Drug Enforcement Agency have become concerned about the risk of abuse and diversion of stimulant medications.^{8,18} Clinician and parent supervision is required to detect substance abuse and prevent adverse outcomes. A diagnosis of ADHD confers at least a twofold greater risk for substance abuse compared to those without ADHD.^{11,15,18} Comorbid conditions, such as a mood disorder or conduct disorder, increase the likelihood of the development of a substance abuse disorder.^{16–18} Effective treatment of ADHD can facilitate substance abuse recovery according to several reports.^{18,27}

Efficacy of Nonpharmacologic Interventions. A multimodal approach to treatment is recommended and should include medication and psychosocial interventions.¹⁰ Psychosocial interventions include parent training, coordination of the treatment plan with teachers, and consistent behavioral interventions. Behavioral interventions include creating a structured environment, setting limits, and adopting a contingency management plan (positive rewards for good behavior).^{9,10,28} Schools may provide behavior therapy with teachers in the context of a Rehabilitation Act (section 504) or an individual education plan. Section 504 requires schools

to make classroom adaptations to help children with ADHD function in the classroom setting. Controlled clinical trials have shown that psychosocial and behavioral interventions are consistently less effective than stimulant therapy; however, parent and teacher satisfaction ratings with these nonpharmacologic interventions are high.^{6,21} Widespread community implementation studies of behavioral interventions at home and in the classroom are lacking. Interpersonal psychotherapy, cognitive therapy, and cognitive-behavioral therapy are not effective in managing ADHD symptoms.²⁸

Clinical Monitoring

Clinical monitoring should be aimed at objectively assessing drug therapy efficacy and treatment-emergent adverse effects.^{4,9,10} Clinical monitoring starts with a careful baseline assessment to determine all coexisting medical and neuropsychiatric conditions, in addition to rating the severity of ADHD symptoms.^{9,10} Several validated rating scales and behavioral checklists are available for parents, teachers, and siblings to measure ADHD symptoms before and after treatment initiation.¹² Specific target outcomes (e.g., completion of assignments, less time in detention) should be assessed, as should the risks and benefits of long-term treatment.^{4,5,10,12,15}

A child's weight, height, sleeping and eating habits, and physical complaints should be recorded before medication initiation to determine the effect of therapy.^{7,9,10} Pulse and blood pressure should be measured at baseline, by one week after initiation of medication (or dosage increase), and then monthly or if the child reports symptoms of lethargy or tachycardia.

Adverse Drug Reactions

Stimulants are well tolerated by most patients. Possible short-term adverse effects include anorexia, stomach pain, insomnia, irritability, and tics.^{7,9,10} These adverse effects can usually be managed by administering the medication with food or through dosage adjustments.⁷ If a child is "overfocused" or exhibiting robotic- or zombie-like behavior, the dose of stimulant should be reduced.^{7,9,10} Tics can emerge or worsen during therapy with any stimulant, and lowering the dosage or changing medications can alleviate the problem.⁷ Growth delays are possible with chronic stimulant therapy, although some studies indicate that long-term effects are minimal.^{7,29} High dosages of a stimulant over extended periods may increase the risk of growth delay, although one study showed a decrease in growth velocity with methylphenidate 20–80 mg/day. Drug holidays are not routinely recommended to prevent growth suppression.⁷ An annual drug holiday may be useful in reassessing the dosage and ongoing need for stimulant therapy and to compensate for any possible growth suppression.

Susceptible patients may experience dysphoria, worsening anxiety, or, in rare cases, psychosis related to stimulant therapy.^{7,23} If a significant mood change occurs, the dosage should be lowered or the drug discontinued. If psychotic symptoms emerge, the stimulant should be discontinued.⁷ Reassessment of diagnosis and treatment is indicated when such serious adverse events occur.^{7,9,10} Possible adverse effects of atomoxetine include nausea, stomach pain, anorexia, dizziness, sedation or insomnia, and pulse or blood pressure

changes.⁸ Bupropion may cause jitteriness, rash, insomnia, or stomach pain or increase the risk of seizures.^{9,23} Tricyclic antidepressant therapy may be associated with dry mouth, constipation, tachycardia, dizziness, weight gain, electrocardiogram changes, and, rarely, sudden death.^{9,10,23} Clonidine and guanfacine can cause sedation, dizziness, syncope, pulse and blood pressure changes, cardiac conduction delay, dry mouth, and constipation.^{10,23,25}

When discontinuing clonidine or a tricyclic antidepressant, the dosage should be gradually tapered over one to four weeks, if possible, because children are particularly sensitive to withdrawal effects. Cholinergic rebound may occur if therapy with a tricyclic antidepressant is abruptly discontinued. Rebound hypertension is possible if clonidine or guanfacine therapy is abruptly discontinued.^{23,25}

Special Populations

Children with comorbid conditions require special considerations in drug selection and clinical monitoring. For example, children with seizure disorders should not receive bupropion but may receive stimulant therapy as long as seizures are well controlled on anticonvulsant therapy.³⁰ Children with bipolar disorder and symptoms of ADHD may require mood stabilizers, such as lithium or valproic acid or atypical antipsychotics, to control their symptoms.^{22,31}

Atomoxetine or an antidepressant like bupropion or a tricyclic antidepressant is preferable for patients with an active substance abuse disorder because of the risk of abuse with stimulants. Patients with a history of substance abuse who have been successful in a substance abuse recovery program may receive stimulant therapy with close supervision and drug monitoring.^{18,27}

A systematic approach to medication implementation that targets the predominant disorder with one medication is initially recommended.⁹ Follow-up assessment will determine if adjunctive therapy is needed. For example, if a child has both ADHD and depression but the ADHD symptoms are more severe, stimulant monotherapy should be implemented and the child should be evaluated to determine if depressive symptoms remit. If the depressive symptoms continue, an antidepressant can be added.⁹

Adult ADHD

The adults studied in drug trials are a heterogeneous population of patients who may have been diagnosed with ADHD in childhood, misdiagnosed, or retrospectively diagnosed.^{16,32} Follow-up studies have found that functionally impairing ADHD persists in 10–60% of childhood-onset cases.¹⁷ Evidence from controlled clinical trials supports the use of stimulants, bupropion, atomoxetine, and tricyclic antidepressants for the treatment of adult ADHD.^{8,32} Atomoxetine, methylphenidate, mixed amphetamine salts, and pemoline all have FDA-approved labeling for the treatment of adult ADHD.

Education

MTA provided strong evidence that clinician contact and ongoing psychoeducational groups focusing on parent training improve therapeutic outcomes in children with ADHD. Parents and caregivers should receive information on realistic

expectations of drug therapy outcomes and training on how to implement behavioral interventions.^{10,33,34} In addition, children should receive support and education to help destigmatize the disorder and prevent negative self-perceptions.^{15,33}

The American Academy of Pediatrics provides answers to commonly asked questions about ADHD diagnosis, course, and treatment on its Web site (www.aap.org/healthtopics/adhd.cfm). For more information on ADHD and its treatment, contact the following resources:

- American Academy of Child and Adolescent Psychiatry: 202-966-7300 or www.aacap.org
- American Psychiatric Association: 888-357-7924 or www.psych.org/public_infor/child.cfm
- Children and Adults with Attention Deficit Hyperactivity Disorder: www.chadd.org
- National Institute of Mental Health: 301-443-4513 or www.nimh.nih.gov

Summary

A thorough assessment by a qualified clinician is needed to establish the diagnosis of ADHD and detect all comorbid medical and neuropsychiatric conditions before initiating therapy. Therapy should be started in patients who suffer from moderate to severe functional impairment. The treatment of ADHD is often suboptimal and overprescribing and underprescribing of medication have been documented in the medical literature. Successful and appropriate medication management includes strategies to overcome the social stigma of diagnosis and concerns about the risk of substance abuse, lack of education about ADHD, lack of health insurance coverage, and lack of coordinated care with parents, teachers, and extended family. The results of controlled clinical trials document the superior efficacy of stimulant therapy over nonpharmacologic interventions in elementary-school-aged children with ADHD. Effective nonstimulant therapies include atomoxetine, bupropion, and tricyclic antidepressants.

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