Chapter 17
Pediatric Psychopharmacological and Combined Interventions

Some pediatric disorders with biogenetic or neuropsychological causes may require medical treatments (Wilens, 2001). Most of these disorders, however, require multimodal treatments, where medication is used in combination with other psychosocial and behavioral interventions or therapies. A select list of common medications will be reviewed, including those designed to control ADHD, major depressive disorders, bipolar disorders, psychotic disorders, Tourette syndrome, and seizure disorders. The National Institute of Mental Health (NIMH) has funded a number of studies to investigate the safety and efficacy of medications and psychosocial interventions for common childhood and adolescent disorders. These will be briefly reviewed.

Specific Classes of Medication

Medications are typically classified as stimulants, antipsychotics, antidepressants (i.e., tricylics, serotonin reuptake inhibitors, atypical antidepressants, and monoamine oxidase inhibitors); antioxiolytics, and antiepileptic medications, depending on their behavioral effects on the CNS (Wilens, 2001). Table 17.1 lists medications currently used to treat children and adolescent disorders. Potential benefits and possible adverse effects are also summarized. Psychopharmacological agents may affect more than one neurotransmitter, and specific neurotransmitters may be implicated in more than one neuropsychiatric disorder (Pliszka, 2003). CNS affects will be discussed in the following sections.

Stimulant Medications

Stimulant medications are the most common psychotropic drugs to treat ADHD in preschool children (Greenhill et al., 2006), school-aged (Barkley, 2006), adolescents (Connor, 2006), and more recently adults (Barkley, Murphy, & Fischer, 2006). Although a majority of children with ADHD respond positively to stimulant medications, approximately 25–30 percent do not (Connor, 2006). Schaughency and Hynd (1989) suggest that "perhaps there are correlated, parallel, or even orthogonal neurotransmitter systems implicated in ADD that account for these differences in response rates" (p. 436). Further, Hunt, Mandl, Lau, and Hughes (1991) propose that "multiple neurotransmitter systems may be involved in integrated cognitive/behavioral functioning," and "the relative balance of these transmitters and these neurofunctional systems determines the modulation of behavior" (p. 272). Thus, individual response rates may be a function of the child's primary dysfunction in cognitive/perceptual systems, arousal systems, or inhibitory systems. This perspective still needs further study in controlled studies. Others suggest that the presence of comorbid disorders may affect the variability in individual response rates (Pliszka, Carlson, & Swanson, 1999). Specifically, Ghurman et al. (2007) did find that preschool children with AD/HD with three or more disorders did not respond to stimulant medications. While demographics were not predictive of medication responses, children with high comorbidity had higher rates of family/environmental risk factors (i.e., lower socioeconomic status, lower

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Table 17.1 Common uses, benefits, and side effects of medications for neuropsychiatric disorders of childhood

Drugs	Common use	Manifestations	Side effects
Stimulants			
Methylphenidate (Ritalin)	ADHD	75% children responders Decreased motor activity, impulsivity, and disruptive behaviors Increased attention Improved socialization Improved ratings (teacher, physician, parent) Increased work completion and accuracy Improved test scores (mazes, PIQ, and visual memory)	Insomnia, appetite loss, nausea, vomiting, abdominal pains, thirst, headaches Tachycardia, change in blood pressure Irritability, moodiness Rebound effects Growth suppression (can be monitored) Lower seizure threshold Exacerbate preexisting tics
Dextroamphetamine (D-amphetamine)	ADHD	Similar to methylphenidate Subdued emotional response Increased reflectivity and ability to monitor self Increased interest level Improved school performance Improved parent ratings (conduct, impulsivity, immaturity, antisocial, and hyperactivity)	Similar to methylphenidate Hallucinations, seizures, and drug-induced psychosis (rare occurrences)
Magnesium pemoline (Cylert)	ADHD	Similar to methylphenidate Improved teacher ratings (defiance, inattention, and hyperactivity) Improved parent ratings (conduct, impulsivity, and antisocial behaviors) Improved test scores (mazes, PIQ, visual memory)	Similar to methylphenidate
Antipsychotics			
Haloperidol (Haldol)	Psychosis Tourette Autism PDD ADD with CD	Reduces aggression, hostility, negativity, and hyperactivity Reduces psychotic symptoms Reduces Tourette symptoms Reduces fixations, withdrawal stereotypes, anger, and fidgetiness in autism Increases social responsivity and reality testing in PDD	Behavioral toxicity with pre-existing disorders Dystonia, loss of tone in tongue and trunk) Parkinsonian symptoms (tremors, mask face, and drooling) Dyskinesis (mouth, tongue, and jaw) Dose reduction decreases motor side effects Intellectual dulling and disorganized thoughts
Chlorpromazine (Thorazine)	Psychosis Severe aggression, explosiveness, and hyperexcitability in MR children	Reduces hyperactivity Reduces tantrums, aggression, self-injury Not effective for young autistic	Similar to haloperidol Dermatological problems Cardiovascular problems Lowers seizure threshold Endocrinological problems Ophthalmological problems Hematological problems
Thioridazine (Mellaril)	Psychosis Severe behavior disorders (extreme)	Reduces hyperactivity Improves schizophrenic symptoms Similar to Thorazine	Similar to haldol Sedation, cognitive dulling, and impaired arousal
Thiothixene (Navane)	Psychosis	Similar to Mellaril	Less sedating than Mellaril

Table 17.1 (continued)

Drugs	Common use	Manifestations	Side effects
Loxapine Succinate (Loxitane)	Psychosis	Similar to haldol	Similar to haldol
Fluphenazine Hydrochloride (Prolixin, Permitil)	Psychosis		
Pimozide (Orap)	Psychosis Tourette (resistant type)	Clinical improvement	High doses-death and seizures
Clozapine (Clozaril)	Severe psychosis (resistant type)	Clinical improvement	Life-threatening Hypertension, tachycardia, and EEG change Seizures
Tricyclic antidepressants			
Imipramine hydrochloride (Tofranil)	Depression Enuresis ADHD School phobia	Improves depression (not severe) Reduces hyperactivity Reduces separation anxiety Improves sleep disorders)	Potentially life-threatening cardiovascular problems Inhibits bladder muscles CNS symptoms (EEG changes, confusion, lowers seizure threshold, incoordination, drowsiness, delusions, and psychosis) Blurred vision, dry mouth, and constipation
Nortriptyline hydrochloride (Pamelor)	Depression	Low rate of clinical improvement in children and adolescents	Withdrawal symptoms
Desipramine hydrochloride (Norpramine)	ADHD ADHD with Tics	Improved ratings (parents and teachers Conners) Clinical improvement	Dry mouth, decreased appetite, tiredness, dizziness, insomnia EEG changes at high doses
Clomipramine hydrochloride (Anafranil)	Obsessive-compulsive disorders Severe ADHD Enuresis School phobia	Reduces obsessions Reduces school phobia/ anxiety Reduces aggression, impulsivity, and depressive/affective symptoms	Withdrawal symptoms Seizures Somnolence, tremors, dizziness, headaches, sweating, sleep disorder, gastrointestinal problems, cardiovascular effects, anorexia, and fatigue
Monoamine osidase inhibitors			
Fluoxetine hydrochloride (Prozac)	Depression Obsessive-compulsive	Effective for adults Clinical improvement for OCD	Nausea, weight loss, anxiety, nervousness, sweating, sleep disorders
Bupropion hydrochloride (Wellbutrin)	Depression ADHD	Adolescents 18+ improve Improved global ratings Not Conners	Seizures, agitation, dry mouth, insomnia, nausea, constipation, tremors
Anxiolytics			
Chlordiazepoxide (Librium)	Anxiety with hyperactivity and irritability School phobia	Clinical improvement Reduced hyperactivity, fears, enuresis, truancy, bizarreness Decreases emotional overload	Drowsiness, fatigue, muscle weakness, ataxia, anxiety, and depression with high doses
Diazepam (Valium)	Mixed psychiatric DX Anxiety and sleep	Improved global ratings Better results for adolescents	Relatively low toxicity
Alprazolam (Xanax)	Anxiety Panic attacks Separation anxiety	Clinical improvements Responders (premorbid, personality were shy, inhibited, nervous)	Mild drowsiness

Table 17.1 (continued)

Drugs	Common use	Manifestations	Side effects
Anticonvulsants			
Phenobarbital	Seizure disorders	Reduces seizures	Lethal at high doses Cognitive impairment, rigidity, and depression
Diphenylhydantoin sodium (Phenytoin)	Seizure disorders	Reduces tonic-clonic seizures	Cognitive impairment Drug toxicity
Carbamazepine	Seizure disorders Manic-depression	Reduces generalized and tonic-clonic seizures Psychotropic effects	Fewer adverse side effects than other drugs Less cognitive dulling, motoric and affective
Sodium valporate	Seizure disorders	Reduces seizures Petit mal + tonic-clonic	Low cognitive symptoms Relatively nontoxic in adults Rare but potentially fatal hepatoxicity in children

Note: Data taken from Green (1991), Neppe and Tucker (1992), and Dubovsky (1992).

parental education and unemployment, and lived in single-parent families).

Disturbances in dopamine (DA) and norepinephrine (NE) levels have been carefully studied in ADHD children, including how stimulants affect these catecholamines for the ultimate control of attention and movement (Barkley, 2006; Pliszka, 2003). DA systems are involved in a variety of cognitive and perceptual functions, including attentional gaiting, sustaining focus, short-term memory, and allocation of memory, while NE systems are involved in cortical arousal, filtering of incoming stimuli, excessive arousal, restlessness, and hyperactivity (Pliszka, 2003).

Serotonin has also been implicated in ADHD, particularly as it relates to cortical inhibition, direction of motor activity, control of impulses and aggression, and complex judgment (Pliszka, 2003). Increased levels of serotonin produce obsessional thoughts, while decreased levels result in increased impulsivity, violent antisocial behavior, criminality, and suicide attempts. Comings (1990) suggests that serotonin may be less important in understanding ADHD, but may be more useful for children with conduct disorders and aggression.

Although biochemical research is difficult to conduct because of developmental changes in neurotransmitters systems, the use of peripheral measures, and the complexity of neurotransmitter action (Zametkin & Rapoport, 1986; Zametkin & Liotta, 1998), several hypotheses have been generated to explain how stimulant medications affect various neurotransmitters.

1. Stimulants bind to the presynapse, thereby increasing the concentrations of catecholamines at the postsynapse (Pliszka, 2003; Volkow et al., 2002).
2. Stimulants increase the release of DA presence at the presynapse and block the reuptake of DA at the postsynapse (see Barkley, 2006).

These various actions facilitate neural transmission by either increasing the amount of neurotransmitters or prolonging the amount of time transmitters are active at the synapse (Pliszka, 2003). Pliszka (2003) suggests that "it is necessary to affect both NE and DA to fully attenuate the symptoms of ADHD" (p.154).

Stimulant Preparations/ Delivery Systems

Stimulant medications can be delivered in a variety of ways, including immediate release, intermediate, extended release, and transdermal patches (Connor, 2006; Wilens, 2001). The main differences in the preparation of stimulants have to do with how quickly the medication is absorbed by the central nervous system, the rate of absorption, or peak plasma or brain concentration of the stimulant (Connor, 2006). Immediate release preparations include: methylphenidate (brand name, Ritalin, Methylin, Metadate), dextromethylphenidate (brand name Focalin), dextroamphetamine (brand name Dextrostat, Dexedrine), and mixed amphetamine salts (brand name Adderall). Intermediate release preparations include: methylphenidate (brand name, Ritalin SR, Methylin ER, Metadate ER), and

dextromethylphenidate (brand name Dexedrine spansule). Methylphenidate can also be delivered in quicker acting, longer lasting intermediate formulas (brand name Metadate CD, Ritalin LA). Extended release formulas include methylphenidate (brand name Concerta) and mixed amphetamine salts (brand name Adderall XR). See Connor (2006) for additional information about onset of action, peak clinical effect, serum half-life, duration of behavioral effects and required number of daily doses. Transdermal patches have also been developed that deliver methylphenidate for up to 12-hour periods.

Standard dosing practices have changed over the years, from BID (morning and noon administration) to TID which includes a three-times-per-day dosing with a late afternoon dose (Corkum, Panton, Ironside, MacPherson, & Williams, 2008). Long acting or sustained release formulas also treat symptoms after school. Research to date shows that the long acting delivery systems appear to be effective (Biederman, Faraone, Monuteaux, & Grossbard, 2004).

The transdermal methylphenidate system also seems effective for improving core symptoms of ADHD, academic performance, and deportment based on teacher and parent ratings (McGough et al., 2006). The patch was well tolerated and, when present, adverse effects were mild to moderate in intensity. The adverse effects were similar to those reported in oral delivery systems, and abated after several weeks.

Benefits, Potential Side-Effects and Medication Management

The potential benefits of stimulant medications are well documented and include enhanced performance on the major symptoms of ADHD, including impulse control, motor coordination, and vigilance (Connor, 2006; Pliszka, 2003); improved cognitive functioning (Barkley, DuPaul, & McMurray, 1991); increased academic productivity and accuracy (Balthazor, Wagner, & Pelham, 1991); decreased off-task behaviors (Barkley & Cunningham, 1979); decreased aggression (Hinshaw, Henker, Whalen, Erhardt, & Dunnington, 1989); improved peer relations (Hinshaw, 1991); fewer negative commands from teachers (Barkley, 2006), and improved interactions with parents (Barkley, 2006).

In general stimulant medications produce improvement in compliance, impulsive aggression, social interactions and academic performance (Wilens & Spencer, 2000). Many of the improvements in interpersonal domains occur not only because the child's behaviors improve, the behaviors of the adults supervising the child are indirectly affected (e.g., fewer negative interactions, less hostility) (Connor, 2006). Social skills, per se, do not necessarily improve without specific skill development and contingencies to support these new skills.

Despite positive results, individual response remains highly variable (DuPaul, Barkley, & McMurphy, 1991), and children should be carefully monitored for adverse side effects (Connor, 2006; Wilens, 2001). Also, a number of environmental factors may affect a child's positive response to stimulant medication. For example, Barkley and Cunningham (1980) found that the better the mother-child relationship, the greater the positive response rate in the child.

Potential Adverse Side effects

Stimulant medications are considered to be well tolerated by most individuals (Connor, 2006). In large clinical trials, common side effects include: abdominal pain, headache, anorexia, vomiting, insomnia, and nervousness. Reduced prosocial behaviors have been reported in children, particularly when given high doses (Jacobvitz, Sroufe, Stewart, & Leffert, 1990), while a reduction in total sleep time and sleep onset (Corkum et al., 2008) have been found in some children. Young children taking stimulant medications for ADHD also appear to have higher rates of adverse effects, particularly in preschool children. Crying, irritability and temper outbursts have been documented in young children (Connor, 2006).

NIMH sponsored a large scale study of stimulant medication efficacy in preschool children with ADHD (PATS). Children were considered for medication trials only after 10 weeks of behavioral treatment. Greenhill et al. (2006) reported that 89 percent of the study children 3–5 years of age responded positively to immediate release methylphenidate, while 11 percent discontinued medication because of intolerable side effects. These adverse effects included insomnia, loss of appetite, moodiness, nervousness, worry, and skin picking. Swanson et al. (2006) also found that 95 percent of preschool

children who remained on medication for 12 months grew 20.3 percent less than expected in height and had a 55.2 percent reduction in weight. The study authors concluded that preschoolers can benefit from stimulant treatment if carefully monitored and if medication benefits are balanced against negative side effects. Other studies have reported chronic height and weight effects in older children. See the discussion of the MTA study in the next section.

Other rare, acute side effects include: motor and vocal tics; sudden death in children with "silent cardiac abnormalities," and psychosis in children with underlying psychotic disorders [see (Connor, 2006) for a review]. While more serious cardiovascular events have been reported in rare cases, cardiovascular effects are minimal in healthy children (Wilens & Spencer, 2000). Recently, the American Heart Association (AHA) recommended that all children on stimulant medications receive an electrocardiogram (ECG) (Vetter et al., 2008). In contrast, the American Academy of Pediatrics (AAP) argues that ECGs be administered only to children with known heart risks. The AHA and AAP later released a clarification of the recommendation stating that physicians may consider an ECG when prescribing stimulant medications, but these are not mandatory. Medical history of the child and family (including sudden deaths) and physician judgment should guide the need for an ECG. The clarification statement indicated that treatment for ADHD should not be withheld if an ECG is not done.

Combined Pharmacological and Psychosocial Interventions

Pharmacotherapy is rarely advised in isolation. Earlier reviews have shown that most childhood and adolescent disorders are complex and affect multiple facets of the child's cognitive, academic, and psychosocial adjustment. Medications also have their limitations and may not uniformly improve all areas of the child's functioning; thus, most physicians combine pharmacological interventions with psychosocial interventions. Psychosocial interventions may include behavioral treatments (e.g., contingency management, home-school notes), individual or group therapy for the child or adolescent, parent training, and family therapy. Combined therapeutic interventions have been more thoroughly researched with ADHD than with other childhood disorders. Early studies showed that medication combined with parent training and behavior management was more effective than either medication or behavior management alone for "normalizing" children with ADHD (Pelham et al., 1988). Low doses of medication (methylphenidate) were considerably enhanced with combined behavioral interventions. Pelham (1993) suggests that "an important result of combined treatments may be that maximal improvement in behavior may be reached without resorting to high dosages of stimulant medication," which may lower adverse medication effects (p. 220). Further, combined behavioral-medication interventions for children with ADHD appear to complement the shortcomings of either treatment alone (Carlson, Pelham, Milich, & Dixon, 1992), and add incremental effects that do not occur with either intervention alone (Pelham, 1993).

The extent to which similar effects will be shown for combined pharmacological-psychosocial-behavioral interventions with other childhood disorders needs further investigation. Research investigating combined interventions is needed to determine the short-term and long-term effects of psychopharmacotherapy and individual responsivity to various aspects of the other behavioral, academic, and psychosocial interventions.

Multimodal Treatment with Stimulant Medications for ADHD

Multimodal treatment generally includes parent and child education about ADHD, stimulant medication, behavioral therapy, and educational interventions for psychosocial and academic difficulties. The Multimodal Treatment Study of Children with ADHD (MTA Cooperative Group, 2004a, b) investigated the efficacy of medication alone (MedMgt), behavioral and psychosocial treatments alone (Beh), combined medication with behavioral interventions (Comb), and a control group who received treatment in a community setting (CC) or treatment as usual. The study is complex in design and is ongoing, following study participants from childhood into early adulthood.

Initial findings after 14 months of treatment showed that all four groups improved (MTA Cooperative Group, 2008a). However, there were some significant findings that are summarized: (1) MedMgt produced larger benefits than behavior therapy; (2) Comb treatment did not significantly increase the overall benefits of MedMgt alone; (3) participants in the Comb treatment group had 20 a percent lower dose of medication with similar results as those children in the MedMgt group who had higher doses; (4) Comb treatment was superior to other treatments for children in families on public assistance; (5) Comb treatment was superior to other treatments for children with ADHD and comorbid anxiety, and (6) families with higher rates of attendance at monthly clinic visits had better treatment outcomes. Medication as prescribed in the MTA study produced clinical improvement in the core symptoms of ADHD – hyperactivity, inattention and impulsivity. Comb treatment (medication and behavioral therapy) was relatively superior to Beh therapy alone and to CC. Even though some children in CC care received medication (38%), they fared less well compared to those in the MedMgt group. These differences may be the result of lower medication doses prescribed in CC compared to those receiving the MTA MedMgt algorithm. The Beh therapy was also found to be more effective than CC – treatment as usual.

Youth receiving medication in CC group had better outcomes than the non-medicated CC youth, and outcomes were similar to those in the Beh group. However, children in the MedMgt had better outcomes than those receiving treatment as usual in the CC group. It is likely that the manner in which medications are administered and monitored in the community affects long-term outcomes.

The MTA did a 24-month follow-up to measure the long-term outcome of treatment (MTA Cooperative Group, 2008a). The major outcomes include the following. (1) There was a persistent relative superiority of MedMgt and Comb groups over the Beh and CC treatment groups, although effect size was lower than 14-month analyses. (2) While participants taking medication at 24 months had better outcomes than those who were not medicated, there was a partial loss of the relative benefits of medication compared to the data from the 14-month interval. (3) Youths who were continually medicated had slower growth gains compared to non-medicated youths (1 cm/year reduction; 1.2 kg/year in weight gain), and stimulant growth suppression may continue when medication treatment is maintained. (4) The compliance with assigned medication doses dropped at the 24-month follow-up, while the percentage of children in the Beh group increased their use of medication. This may have affected the results between the treatment groups (see #1 above). (5) Children who stopped medication at the 24-month follow-up had greater overall deterioration in outcomes, while those children starting medication after the initial study phase improved during the 10-month interval.

The 36-month follow-up of the MTA study revealed interesting and sometimes confusing results at the 2nd follow-up (MTA Cooperative Group, 2008a, 2008b). The relative superiority of Comb and MedMgt over the Beh and CC was completely lost at 36 months. The relative superiority of the MTA medication algorithm was lost, and continued medication was a marker of deterioration in some children. There were three different outcome trajectories that are important. Two groups (66% of sample) showed large initial improvement on medication. Class 1 (52%) had a large initial improvement that was maintained over time, while another Class 3 (14%) had large initial improvement that was not maintained at 36 months. In fact, Class 2 showed deterioration at the 36-month follow-up. Family demographics showed that this group had higher rates of adversity than the other groups. Further, Class 2 (34% of sample), had initial modest improvement on medication that gradually increased over time, and was significantly better than those who were not medicated. The first two groups (66%) of the sample had a large and significant improvement with medication over time, and for Class 1 the magnitude of improvement was even greater at 36 months.

The MTA Cooperative Group (2008b) also investigated moderator variables to determine how comorbidity factors impact outcomes. Children with comorbid anxiety had improved outcomes when given Beh therapy, and with outcomes similar to both the MedMgt and the Comb therapies. The Beh, Comb, and MedMgt were superior to treatment as usual (CC therapy) for children with anxiety. Children with disruptive disorder, ADHD and anxiety also fared better with Comb treatment compared to the other therapies. Further, Comb,

but not MedMgt, therapies reduced the persistence of oppositional defiant disorder (ODD) and mood disorders.

Family demographics appeared to impact the effectiveness of various treatment modalities. Children from families with income challenges (receiving public assistance versus those who were not) had better outcomes in the Comb therapy compared to other treatments (MTA Cooperative Group, 2008b). Significant reductions in negative/ineffective discipline practices only occurred in the Comb treatment group. In general, families with socioeconomic disadvantage responded most favorably to Beh treatment. In addition, children receiving Comb and Beh therapies had some protection against substance experimentation and delinquency compared to MedMgt and CC groups that were not protected.

While the MTA study has been the most comprehensive of its kind, there are complexities to the data and analyses that have created controversies and confusion. The study is ongoing, and eight-year follow-up data were presented at the MTA Research Symposium (2007), 10-year data analyses have been completed, and 12-year follow-up is in progress (MTA Cooperative Group, 2008b). To date the study has shown long-term benefits of stimulants over a one-year period. These findings are consistent with those reported by Abikoff et al. (2004) which documented benefits of stimulant medications over a two-year period when medications are properly monitored and adjusted. At the 24-month follow-up, the MTA data found that the relative benefits of stimulants were gradually reduced when children return to community care. In the three-year follow-up, treatment benefits dissipated completely for some children, while others continued to improve. In the future, the MTA study will address important questions about quality care for ADHD, the ultimate effects on height and weight for children who show stimulant-related growth suppression, and long-term functional outcomes of treatment (MTA Cooperative Group, 2008b).

Non-Stimulant Medications for ADHD

A non-stimulant medication, atomoxetine hydrochloride (brand name Strattera) was recently approved and has shown to be effective for reducing the core symptoms of ADHD (Kelsey et al., 2004). Comparison studies are somewhat inconsistent, with some reports showing greater improvements on Adderall (extended release mixed amphetamine salts) compared to Strattera (Faraone, Wigal, & Hodgins, 2007), while others show that Strattera is comparable to that of methylphenidate (Kratochvil et al., 2002). Barkley, Anderson, and Kruesi (2007) also found that Strattera improved self-ratings of ADHD symptoms in adults with ADHD and self-evaluations in a driving simulator, although these improvements were not noted for examiner-rated driving performance. Bohnstedt et al. (2005) also found that parent and teacher ratings of ADHD showed significant improvement for children on atomoxetine compared to placebo. Parent ratings appeared to detect "a larger effect and accounted for more unique variance in the prediction of treatment type, independent of teacher-based ratings" (p. 158).

Other medications used for children who are considered non-responders to stimulant medications include imipramine (Connor, 2006), and MAOI (Zametkin, Rapoport, Murphy, Linnoila, & Ismond, 1985). Desipramine selectively blocks the uptake of NE, and imipramine may block the uptake of serotonin. While antidepressants may increase NE availability at the synapse (Hunt et al., 1991), these are not the frontline medications of choice for most children with ADHD.

Wood, Crager, Delap, and Heiskell (2007) reviewed non-stimulant medications that have been used to treat ADHD. Tricyclic antidepressants have been used to treat individuals with ADHD, particularly those with comorbid disorders including mood, anxiety, oppositional and tic disorders. Selective serotonin reuptake inhibitors (SSRIs) have a more cautioned tale. In 2004, the FDA issued a "black box" warning about the risk for suicide in youths taking SSRIs. "The implementation of an SSRI in treating ADHD should only be considered when there is a dual diagnosis of ADHD with depression or anxiety" (Wood et al., 2007, p. 344). Other side effects for SSRIs (increased mania, agitation, insomnia, akathisia, and sleep difficulties) should be carefully monitored. Other antidepressants (i.e., bupropion, venlafaxine) block the reuptake of serotonin and norepinephrine, but children on these medications require regular electroencephalogram (EEG) monitoring to assess the risk for seizure activity in a very small percentage of individuals.

Studies of antihypertensive medications show that clonidine (CLN) reduces ADHD symptoms (Wilens & Spencer, 1999). Clonidine appears to successfully reduce aggression, sleep disturbances, and tics [see (Wood et al., 2007) for a review]. While improvement in conduct-related problems has been shown when CLN is used in combination with stimulants, serious side effects have been reported. In rare instances, sudden death in children has been reported when stimulants and CLN are combined, but evidence for the linkage is not well established (Taylor et al., 2004). Although promising, less research has been conducted testing the efficacy of guanfacine (GFN).

A number of atypical antipsychotic drugs have been used for children experiencing high levels of behavioral problems, including aggression and disruptiveness. Risperidone (RPD) and aripiprazole (ARP) seem to improve severe behaviors, but are less helpful for core ADHD symptoms (hyperactivity and cognition) (Wood et al., 2007). Serious side effects reduce the widespread use of these medications, except for children with extreme behavioral problems. Carbamazepine (CBZ), a seizure medication, appears to be safe and effective for children, but serious side effects (i.e., dizziness, headaches, drowsiness, ataxia, blurred vision, nausea, vomiting, rash, heptic abnormalities) limit their widespread use as an alternative medication for ADHD (Silva, Munoz, Alpert, 1996; Pellock, 1987). Wood et al. (2007) conclude "Despite their variability as alternatives, each type of medication has certain limitations, side effects, and varying amounts of research available to substantiate its use for the treatment of ADHD. These medications offer several potentially successful options to those who fail to respond to stimulants or those who find the side effects of stimulants bothersome" (p. 341).

Antidepressant Medications

There are three main classes of antidepressants, including: Tricyclic antidepressants (TCAs), selective serotonin reuptake inhibitors (SSRIs), atypical antidepressants, and monoamine oxidase inhibitors (MAOIs) (Wilens, 2001). Tricyclic antidepressants (e.g., imipramine, desipramine, nortriptyline, and clomipramine) act on DA and selectively block the reuptake of NE and serotonin (Pliszka, 2003). TCAs have been used to treat a variety of childhood and adolescent disorders, including (1) imipramine for depression, enuresis, school phobia, and sleep disorders; (2) desipramine for ADHD and ADHD with tics; (3) nortriptyline for major depressive disorder, and (4) clomipramine for obsessive-compulsive disorders (Phelps et al., 2002; Wilens, 2001). Plasma levels should be monitored to identify toxic effects, including affective (mood, concentration, lethargy, social withdrawal), motor (i.e., tremor, ataxia, seizures), psychotic (thought disorders, hallucinations, delusions), and organic (disorientation, memory loss, agitation, confusion) symptoms, or to identify subtherapeutic levels of medication (Phelps et al., 2002). Bostic, Prince, Frazier, DeJong, and Wilens (2003) found that TCAs were less effective for young children with depression, while studies with teens show more promise (Brent et al., 2008).

SSRIs, newer agents that inhibit the reuptake of serotonin, include fluxotine (Prozac), sertraline (Zoloft), fluvoxamine (Luvox), citalopram (Celexa), sertraline (Zoloft), and paroxetine (Paxil) (Pliszka et al., 1999; Wilens, 2001). Fluoxetine (Prozac) is effective for the treatment of depression and obsessive-compulsive disorders (Phelps et al., 2002). Side effects include nausea, decreased appetite, and insomnia, and are usually mild and transient in nature (Phelps et al., 2002). While SSRIs are considered to be frontline treatments for children and adolescents with depression (Birmaher & Brent, 2003), Safer (2006) reports that SSRIs are not more effective than placebo for treating preadolescent youths and they produce increased adverse effects in children.

Atypical antidepressants have been used to treat children with depression including Wellbutrin, Effexor, Remeron, and trazodone (Wilens, 2001). Wellbutrin (bupropion), similar to amphetamines, works on dopamine and has also been found to be useful for smoking cessation (Wilens, 2001). Wellbutrin is frequently used for children with comorbid depression and ADHD, and/or depression and serious mood swings. The major side effects are irritability, appetite suppression, insomnia, tics and, when given in high doses, Wellbutrin can produce drug-related seizures (Wilens, 2001). In rare cases, self-injury and manic episodes have been reported in children on SSRIs (Pliszka et al., 1999).

MAOIs have recently been investigated for treating childhood disorders including depression, anxiety, panic attacks, and ADHD (Wilens, 2001). The most common MAOIs are phenelzine (Nardil) and tranylcypromine (Parnate). Because of their potential for hepatotoxicity, the need for food restrictions, and questionable effectiveness MAOIs were not commonly administered to children, although this trend has recently been reversed (Wilens, 2001). MAOI-Type A deactivates NE and serotonin, while MAOI-Type B deactivates DA and phenylethylamine (Zametkin & Rapoport, 1987). Food restrictions include aged foods particularly cheeses, certain drugs (cocaine), and cold medicines due to the possibility of increased blood pressure. Other side effects that have been reported include, blood pressure changes, weight gain, drowsiness, and dizziness [see (Wilens, 2001) for more details]. Green (1991) suggests that MAOIs should be tried before starting a trial of Wellbutrin. Like other antidepressants, MAOIs also require careful plasma level monitoring.

In 2004, the U.S. Food and Drug Administration (FDA) announced a "black box warning" on the administration of antidepressants due to an increased risk for suicidal thoughts and behaviors (Bhatia et al., 2008). Studies investigating the risk for suicide in medicated children and adolescents report mixed findings, with some indicating an increased risk (Olfson, Marcus, & Shaffer, 2005; Simon, Savarino, Operskalski, & Wang, 2006), while others suggest the benefits of antidepressants outweigh the risks in pediatric populations (Bridge et al., 2007; Gibbons, Hur, Bhaumik, & Mann, 2006). In a meta-analysis of studies investigated the safety and efficacy of SSRIs, nefazodone, venlafaxine, mirtazapine in children younger than 19. Bridge et al. (2007) indicate that antidepressants are efficacious for pediatric patients with major mood disorders, obsessive-compulsive disorders (OCD), and non-OCD anxiety, while Olfson, Marcus, and Shaffer (2006) support careful clinical monitoring in depressed children (6–18 years of age) due to increased suicide attempts and deaths.

NIMH has funded a number of large-scale investigations to study the effects on antidepressants alone and in combination with cognitive behavioral therapy for reducing depression in young children. These studies provide evidence for the efficacy of antidepressants in teens, while others call for caution when using SSRIs with children and adolescents.

Treatment for Adolescents with Depression Study (TADS)

NIMH funded a large scale, multi-site study of the treatment of depression in teens (TADS, 2004). Treatment conditions were medication (fluoxetine) alone; cognitive behavioral therapy (CBT) alone; combined treatment (fluoxetine plus CBT) and placebo. The study found that antidepressant medication was effective for treating youths with depression, but the combined treatment produced the greatest improvement. CBT alone was less effective than medication alone, although both treatments were better than the placebo condition.

The combined treatment significantly reduced depressive symptoms, and was superior to medication and CBT alone (TADS, 2004). Teens in the study were carefully monitored for adverse side effects including gastrointestinal track events, sedation, and insomnia. Medication doses were adjusted to reduce these effects.

While 30 percent of teens had suicidal ideation at the start of the study, suicidal ideation was reduced in all groups. Decreases in suicidal ideation were greatest for teens in the combined treatment condition, while medication alone was not as protective. Based on this study, CBT appears to be an effective treatment for depression and protects teens with SI with carefully monitored medication (prozac).

STAR*D Sequenced Treatment Alternatives to Relieve Depression

The National Institute of Mental Health (NIMH) funded the nation's largest study investigating 18–75-year-olds with treatment-resistant depression (Rush et al., 2004; Weissman et al., 2006). STAR*D data show that about half of all individuals with difficult to treat depression reached remission with additional treatment, but the odds were reduced with each new trial of medication (Rush et al., 2006). Approximately one-third of participants reached remission at Level 1 treatment when given Celexa

(citalopram), an antidepressant. CBT was used either as a switch or add-on treatment during Level 2 of the study, while at Levels 3 and 4 new medications were either switched or added on. Drop-out of the study also increased when additional treatments were not effective. It is important to note that patients who have not responded positively to two prior antidepressants and then switch to a different class of antidepressants have only a minimal chance at remission by taking the new medication (Rush et al., 2004, 2006). Additional research is being conducted on the STAR*D sample to determine the efficacy of either adding on or switching to CBT, and to determine who responds to what treatment sequence.

In another phase of the STAR*D study, Zisook et al. (2007) investigated the impact of early versus late onset of depression to determine medication response across five age groups: childhood onset (ages <12), adolescent onset (ages 12–17), early adult onset (ages 18–44), middle adult onset (ages 45–59), and late adult onset (ages $\geq$ 60). "No group clearly stood out as distinct from the others. Rather, the authors observed an apparent gradient, with earlier ages at onset associated with never being married, more impaired social and occupational function, poorer quality of life, greater medical and psychiatric comorbidity, a more negative view of life and the self, more lifetime depressive episodes and suicide attempts, and greater symptom severity and suicidal ideation in the index episode compared to those with later ages at onset of major depressive disorder" (Zisook et al., 2007, p. 1539). Thus, age of onset was not associated with a difference in treatment response to the initial trial of citalopram.

Treatment of Resistant Depression in Adolescents, TORDIA

In a study of 12–18 year olds who were non-responders to one SSRI, Brent et al. (2008) investigated other antidepressants, with and without CBT. Teens in the TORDIA study were exposed to the following treatments: (1) switch to a 2nd SSRI (i.e., paroxetine, citalopram, or fluxotine); (2) switch to different antidepressant medication plus CBT; (3) switch to venlafaxine, and (4) switch to venlafaxine plus CBT. Those teens who had switch to 2nd SSRI and also received CBT showed the most improvement. Treatment with venlafaxine (compared to another SSRI) was just as efficacious with fewer adverse effects than the other SSRIs.

In summary, continued research on the safety and efficacy of all antidepressants in children and adolescents is needed. While research has shown that some antidepressants can effectively treat depression in teens, especially when combined with cognitive behavioral therapy, the findings for young children are more equivocal. Regardless of the study, clinicians are advised to carefully monitor the adverse events that accompany antidepressants, particularly suicidal ideation and/or behaviors.

Antipsychotic Medications

Antipsychotic medications have been classified as typical (1st generation) and atypical (2nd generation), and are used to treat a variety of neuropsychiatric disorders in children and adolescents, including schizophrenia (Wilens, 2001); pervasive developmental delays (Joshi, Cappozzoli, & Coyle, 1988); chronic motor tics and Tourette syndrome (Comings, 1990); severe aggression and conduct disorders (Green, 1991); cognitive retardation with psychotic symptoms (Gadow & Poling, 1988), and excessive or severe hyperactivity, low frustration tolerance, and poor attention (Wood et al., 2007). While the first generation antipsychotics were effective, the adverse side effects were often intolerable, thus the need to find other safe alternatives.

The typical antipsychotics include butyrophenones (e.g., haldol), phenthiazines (e.g., Thorazine), and thioxanthenes (i.e., navane), while the atypical antipsychotics include clozaril, resperidal, ziprasidone, seroquel, and geodone (NIMH, 2007). The major differences between the classes of medications are the side effects, doses, and potency. While both classes of antipsychotics have receptor blocking properties in the dopamine (DA) and serotonin (5-HT) systems, the medications have different actions in the frontal -hippocampal systems [see (Pliszka, 2003) for a more detailed discussion]. Pliszka (2003) indicates that a new class of drugs are being developed (aripiprazole) that have minimal effects on the 5-HT, but stabilizes the DA

system (e.g., increases DA in the cortex while reducing activity in the mesolimbic system).

Weizman et al. (1984) indicate that neuroleptics, in combination with stimulants, might be effective for a small number of ADHD children who do not respond to either medication alone. Apparently, when these drugs are used in combination, the stimulants increase the release of DA while the neuroleptics block DA, thereby suggesting synergetic effects between the two agents (Green, 1991). Zametkin and Rapoport (1987) indicate that antipsychotics are not as effective as stimulants, but these medications do seem to decrease motoric activity and inattention.

Because of the serious side effects associated with antipsychotics, these medications require careful monitoring. Green (1991) suggests that cognitive dulling, sedation, and irreversible tardive dyskinesia (abnormal involuntary movements) are of particular concern when treating children and adolescents. Children and adolescents are prone to exhibit acute dystonic reactions (e.g., neck spasms, mouth and tongue contractions, eyes rolling upward) within the first five hours of ingestion, and are more at risk when taking high potency, low dose antipsychotics, versus low potency, high dose regimens.

Antioxiolytic Medications

Antioxiolytics, specifically benzodiazepines (BZDs), are typically administered to control of severe anxiety, sleep disorders (e.g., insomnia, sleep terrors, and/or sleep walking), and over-inhibition disorders (Wilens, 2001). Relatively little research has been conducted on these medications with children and adolescents, although the American Psychiatric Association Task Force on Benzodiazepines reported that these drugs have low toxicity and abuse potential (Salzman, 1990).

The most common medication of benzodiazepines include Valium (diazepam), Librium (chlordiazepoxide), and Klonopin (clonazepam; Wilens, 2001). Other antioxiolytics medications are antihistamines (i.e., Benadryl, Vistaril, and Chlor-Trimeton), and atypical antioxiolytics (Buspar).

Benzodiazepines appear to affect GABA receptors, which in turn enhance chloride channels to produce hyperpolarization of neurons (Pliszka, 2003). This neurochemical (BZD-GABA) process has inhibitory affects in arousal and affective brain centers, thus reducing anxiety. In addition, benzodiazepines are also effective antiepileptic medications. For example, Valium enhances GABA's inhibitory action to terminate seizures. Potential side effects (e.g., sedation, muscle relaxation, and elevated seizure threshold) appear related to the effects BZD receptors have on cortical, pyramidal, and spinal neurons throughout the brain (Wilens, 2001).

Withdrawal symptoms (e.g., dysphoria, anxiety, heightened sensitivity to light and sound, headaches, sweating, tremors, insomnia, nightmares, delirium, and paranoia) have been reported with BZDs and are similar to the effects of withdrawal from other CNS depressants. Further, long-term use of the BZDs may result in tolerance to the medications, thereby reducing the benefits (Wilens, 2001). Care must be taken when discontinuing BZDs, particularly at high doses. Withdrawal symptoms are common if the medication is stopped too quickly, including mental confusion, increased blood pressure, and, in some cases, seizure activity.

Buspar, a newer antioxiolytic medication, has been used to treat children with severe aggression (Wilens, 2001). Buspar works differently than the BZDs and does not produce anticonvulsant, sedative or muscle relaxing effects. Buspar apparently works on serotonin, but may not be as effective as the typical BZDs. However, side effects for Buspar are not as adverse as the BZDs. Buspar has lower abuse potential, and does not require blood monitoring (Wilens, 2001).

Antiepileptic Medications

Antiepileptic medication is the major form of therapeutic intervention for children and adolescents with nonfebrile seizure disorders. Phenobarbital and phenytoin both have adverse affects on academic work, due to their sedative affects. Phenobarbital has been known to decrease memory in some children and contribute to disturbed behaviors in other children (Wilens, 2001). However, when the children are given other antiepileptic medications, these behavioral and cognitive side effects improve. Carbamazepine also has adverse side effects, but these seem to be less severe than those of the other two agents. All three medications are commonly used, either in combination or as single agents, and

require careful blood level monitoring. Antiepileptic medications act as enzyme-inducing agents in the liver, which in turn appears to reduce the "bioavailability of almost all psychotropic agents" (Neppe & Tucker, 1992, p. 4 17). Antiepileptic medications appear to modulate DA, serotonin, and GABA receptor sites.

There has been a trend to use the antiepileptic medications to treat other childhood psychiatric disorders, especially valporic acid/divalproex sodium, carbamazepine, and oxcarbazepine (Handen & Gilchrist, 2006). Valproate has been used to treat children with cognitive retardation who also have aggression, but adverse effects should be carefully monitored, including hepatic failure and hemorrhagic pancreatic which can be life threatening.

Mood Stabilizers: Medications for Bipolar Disorders

Early reports from the STEP-BD study indicate that participants with early onset bipolar disorder (BD) have higher rates of comorbid disorders (i.e., anxiety disorders and substance abuse), higher and shorter periods of euthymia, higher rates of suicide attempts, and increased mood episodes upon entering the study. Thus, bipolar disorders are more complex, severe forms of childhood psychopathology. Mood stabilizers are used for bipolar disorders, seizures, aggression, and self-injurious behaviors (Handen & Gilchrist, 2006). Mood stabilizers include: lithium (Lithobid), oxcarbazepine (Trileptal), valporic acid (Depakene), carbamazine (Tegretol), lamotrigine (Lamictal), and topirmate (Topamax). Divalproex and carbamazepine were first developed as anticonvulsants, while other mood stabilizers are also "atypical" antipsychotics. Adverse side effects have been reported and are often difficult to tolerate, including dizziness, drowsiness, cognitive sedation, vomiting, diarrhea, insomnia, loss of appetite, and extrapyramidal effects (Phelps et al., 2002).

While TCAs and SSRIs have been effective treatments for adults with bipolar disorder, these antidepressants are less effective for treating pediatric populations (Birmaher, 1998). Scheffer, Kowatch, Carmody, and Rush (2005) investigated treatments for youths between the ages of 6–17 years with bipolar disorder with comorbid ADHD. Using a placebo-controlled crossover design, Scheffer et al. (2005) found that mixed amphetamine salts were significantly more effective than placebo for ADHD symptoms. In addition, there were no significant adverse effects and manic symptoms did not increase. In this study, manic symptoms were first controlled with divalproex sodium, which did not effectively reduce ADHD.

NIMH has funded three major studies investigating the treatment options for children and adolescents with bipolar disorders, including the Systematic Treatment Enhancement Program for Bipolar Disorder (STEP-BD), Treatment of early Age Mania (6–15-year-olds), and Effectiveness of Family-focused Therapy (13–17-year-olds). These studies are ongoing and will no doubt shed needed light onto the most effective treatment options for pediatric bipolar disorder.

Regardless of the prescribed medication, there is agreement that benefits must be carefully weighed against adverse effects. Clinicians must strategically monitor medication effects and determine the need for medication, appropriate doses and the need for other combined treatments.

Monitoring Medication Efficacy

A key question prior to selecting pharmacological intervention is whether medication is warranted. This decision typically requires a comprehensive assessment of the problem and a careful review of the child's medical, educational, and psychosocial history (DuPaul et al., 2003). It is important to determine the exact nature and severity of the disorder prior to medicating and, in some cases, to determine if other psychosocial or behavioral interventions have been attempted. Information concerning previous non-medical interventions is particularly important for such pediatric disorders as ADHD, depression, anxiety, and conduct disorders.

When non-medical interventions are not successful in ameliorating the child's problems, then a controlled trial of medication may be considered. Physicians usually obtain baseline data prior to medication trials, which may include electrocardiogram (ECG); electroencephalogram (EEG); urinalysis; liver, thyroid, and renal function tests; blood pressure, and

serum blood levels when administering antipsychotics, antiepileptics, and antidepressants (Green, 1991). Other baseline behavioral data (rating scales, questionnaires, etc.) are also collected in order to measure the effects of medication. Once psychopharmacotherapy is initiated, objective measures of medication effects are needed to determine individual response rates and to assess the side effects of various medications (Barkley, 2006; DuPaul & Stoner, 2003; Wilens, 2001). A number of rating scales are available to measure classroom behaviors and side effects for ADHD (see Barkley, 2006; DuPaul & Stoner, 2003), but fewer scales are available for other childhood disorders.

Pelham (1993) suggests that when monitoring medication it is advisable to measure ecologically valid behaviors in order to assess the effects of medication on a child's performance in the classroom and in social situations. Pelham (1993) employs daily report cards that target behaviors such as work completion, compliance, and accuracy in order to determine the effects of stimulant medications. Although Pelham (1993) specifically addresses medication monitoring of stimulants for ADHD children, ecologically valid measurements would also seem appropriate for other childhood disorders, including depression, anxiety, and conduct-related problems. To assess whether a particular medication is helping a child, the behaviors of concern (e.g., sadness, panic attacks, or anger outbursts) may need to be defined more explicitly and monitored on a regular basis. Thus, for medication monitoring to be ecologically valid, it should occur in the child's natural setting (home and school) and not solely in the clinic or the doctor's office.

Given the need for assessing medication affects in the child's natural setting, it is important that schools, physicians, and parents work together to produce the most benefits from pharmacological approaches. The following section discusses these partnerships.

Home-School-Physician Partnerships

Home-school-physician partnerships are necessary for several reasons. First, children often receive psychosocial, behavioral, and medical interventions from a number of different professionals, and coordination of these services is required. It is not uncommon for a child with a neuropsychiatric disorder to have a psychiatrist or physician prescribe medication, a clinical psychologist conduct therapy, and a school psychologist and/or counselor address school-related academic and psychosocial problems. These various professionals often target the same behaviors and have similar therapy goals, but they may use different techniques. Therapeutic efforts in one setting should not be counterproductive to the efforts in another. These situations occur when professionals have diametrically opposed theoretical orientations or utilize drastically different approaches for the same behavioral, psychological, and/or academic problem. Parents may pursue the course recommended by one professional, only to hear a completely opposite opinion from another. This not only creates stress and confusion for the parent, it may set a course of action that is completely counterproductive for the child.

Second, because of the concern over high costs of comprehensive assessments and interventions, duplication of services should be avoided whenever possible. Professionals in different settings may utilize similar evaluation procedures (e.g., rating scales, intellectual measures). It is not uncommon for a child to be assessed using the same instruments, for parents to fill out the same rating scales, and for teachers to respond to the same questionnaires for different professionals (e.g., psychiatrist, clinical psychologist, and school psychologist) within a relatively short period of time. Interventions may also be similar across therapeutic settings. Coordinating services and communication between professionals and parents helps to reduce needless redundancy.

Third, a number of children receive medication on a daily basis. Medication monitoring is an important element of pharmacotherapy and is most helpful when conducted in the child's natural environment, the home or the school, where the behaviors of concern can be systematically observed. Physicians need careful and systematic information about how the child is responding to medication, and whether there are side effects at various dosage levels. Properly trained school professionals (e.g., school psychologists) can be extremely helpful in this process. School psychologists may observe the child, collect behavioral data (e.g., work completion rates), and assess

psychosocial adjustment at various dosage levels. These data can be communicated directly to the physician (with parental permission), or to the parent for proper medication monitoring. Information concerning individual responsivity needs to be communicated on a regular basis in order to ascertain the child's progress.

Fourth, when children with various brain-related diseases or disorders (e.g., brain tumors, traumatic brain injury) reenter the school system, the professional staff needs to be knowledgeable about the child's medical, psychosocial, academic and behavioral needs. In order to be knowledgeable about the ramifications of brain-related disorders, educational professionals need to be in regular contact with attending physicians (e.g., neurologists, neurosurgeons) and other medical specialists (e.g., speech and physical therapists). Information in these situations needs to be bidirectional – from the physician to the teacher or school psychologist, and vice versa. Physicians need information from the school about how the child is progressing and if relapses or other secondary problems are emerging. Educational professionals need to understand the nature and course of recovery of the child's injury or disease.

Fifth, parents and family members may need help coping with the demands and stresses of the child's neuropsychiatric disorders, diseases, or trauma. While each professional may play a different role in this process, each may also possess important information that may be useful to the other. Again, communication between the physician and the school is essential.

It is important to remember that when developing home-school-physician partnerships, confidentiality is required. Parental permission is needed before obtaining and sharing information, and sensitive or personal information should be discussed only on a need-to-know basis. That is, teachers and other school personnel may be informed when information directly affects the intervention or treatment plan; otherwise, personal information should be kept confidential. A case illustration may help clarify this point. A child had been severely beaten by his mother's boyfriend and sustained serious brain trauma. When the child reenters school, should the source of the child's injury be shared with school personnel (e.g., child's teacher, school psychologist)? If there is continued concern about the safety of the child or concern about the psychological trauma suffered by the child, then sharing this information with the educational professionals is appropriate. If psychoeducational services are needed, then the school psychologist and other educational professionals may also need to know. If the child has already stabilized (i.e., medically, neuropsychologically, and emotionally), then the cause of the injury may not be all that pertinent. Most often the school administrator would be informed under both conditions.

Most of the reasons discussed here suggest the need for communication and coordination of services across agencies. Many parents feel that they have been placed in the role of services coordinator for their child–a role that parents do not always want to assume. Thus, it is imperative that school and medical professionals discuss these issues and identify an individual who will be responsible for coordinating assessment and intervention plans across the various settings. Regular communication among all parties is needed, and a plan or systematic schedule may be helpful, particularly during the assessment and early intervention stages. Contact may be less frequent once the child stabilizes and shows steady progress in meeting the therapeutic or intervention goals. Regular follow-up at six-, 12-, 18-, and 24-month intervals may be sufficient in later stages when the child has shown adequate recovery or is progressing on target.

Summary and Conclusions

This chapter presented a model for comprehensive, multimethod assessment and intervention for children. Five basic assumptions underlie this model. First, the model assumes that many childhood disorders have a biogenetic basis, such that neuropsychological as well as cognitive, behavioral, and psychosocial factors must be considered for assessment and treatment. Second, a single theoretical paradigm (e.g., behavioral, cognitive, or neuropsychological) is rarely defensible when applied in isolation. One-dimensional explanations for complex, multidimensional conditions are not scientifically founded. Third, developmental disorders of childhood present early in life and respond favorably to early intervention. Neurocognitive paradigms offer

strong theories and methods for addressing childhood disorders within a developmental framework. Fourth, various paradigms make important contributions for different reasons and, when combined, increase the probability of obtaining the best treatment for children with serious disorders. Finally, advancing the science of childhood disorders will not occur in the form of dramatic discoveries from or within a single paradigm, but will occur through patient working and reworking of complex sets of experimental variables, with clinical validation (Doehring, 1968).

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