

Attention-Deficit/Hyperactivity Disorder

in Infancy and in the Preschool-aged Child

By Leon Strauss, MTechHom (TWR)

The diagnosis and treatment of attention-deficit/hyperactivity disorder (ADHD) has been focused on school-aged children, with recent attention drawn to adults with ADHD. Chronic treatment programs from childhood through late adulthood have become part of modern ADHD management programs, identifying and treating key areas of need within each age group.

Attention deficits are generally thought to be mostly associated with children; however, it has been recognized that attention problems not only continue into adulthood but also reveal themselves in more adults as they get older.¹ The onset of ADHD typically occurs before the age of 3 years, with parents of children with ADHD commonly reporting excessive motor activity in toddlers. Peak presentation to health care professionals occurs between the ages of 7 and 10 years.²

This article will concentrate on the early identification and management of ADHD in infants and preschool-aged children.* Early treatment of ADHD can prepare children for the academic years, as well as improve relationships within the family, which are often strained.³ Numerous family studies have suggested a familial pattern to ADHD.⁴ These studies suggest that there is a higher

prevalence of mood and anxiety, learning, substance-related, and antisocial personality disorders in family members of individuals with ADHD.⁵

The chemical messengers

Neurotransmitters include chemicals classed as peptides, nitric oxides, neurotrophic factors, and cytokines. More than 300 substances that control our internal neural world and directly influence our interaction with others have been identified.

Catecholamines, such as dopamine, and amines, such as serotonin, play an important role in the evolution of ADHD; these imbalances can be identified in the preschool-aged child with ADHD.⁶ Dopamine plays a critical role in motivation, reward-seeking behavior, and attentional processes.⁷ Imbalances of dopamine in limbic regions have been linked to ADHD,⁸ schizophrenia, and sub-

cortical neuropsychiatric disorders, including Tourette syndrome and possibly autism.⁹ Dopamine levels are preferentially reduced in the frontal brain regions of adult patients with ADHD. Furthermore, genetic abnormalities related to dopamine transporter proteins have been reported in patients with ADHD,¹⁰ supporting the concept that ADHD has strong genetic ties and is a disorder that begins early in life and changes form through the teenage years and adulthood.

Serotonin is essential in neurobehavioral processes, including mood and anxiety.¹⁰ Serotonergic imbalances are related to mood disorders, anxiety syndromes (including obsessive-compulsive disorder,¹¹ posttraumatic stress disorder, and panic disorder), autism, and insomnia.⁹ Low platelet serotonin concentrations were identified in children with ADHD more than 20 years ago; increasing serotonin levels to within the normal range repeatedly lessens ADHD symptoms in children with low serotonin levels.⁵

Circulating serotonin and dopamine levels and receptor site activity can be adversely affected by genetic and environmental factors.¹⁰ Functional polymorphisms of the serotonin transporter genes have been associated with depression and autism.¹² Environmental toxins, such as 1-methyl-4-phenyl-1,2,3,6-tetrahy-

* Future articles will address ADHD and its management in school-aged children and hyperactivity in adults.

dropridine (MPTP), can produce a permanent hypodopaminergic state indistinguishable from Parkinson disease by killing neurons in the substantia nigra of the brain.¹⁰ Toxins from the environment include gases (e.g., carbon monoxide), metals (e.g., mercury), liquids (e.g., ethanol), and numerous solids. Prenatal exposure to lead can result in mental retardation and cerebral palsy. As many as 1 in 10 women are at risk of bearing children with learning disabilities and other neurological problems because of mercury exposure. Mercury affects both prenatal and postnatal brain development; it specifically damages or kills neurons in utero. The consumption of fish is the most common source of exposure, although airborne mercury contamination is becoming more of a concern. Exposure to neurotoxins

in pregnancy is associated with disordered cognitive development, lowered IQ scores, impairments of memory and attention, and coordination deficits.¹³

Identifying ADHD in the preschool-aged child

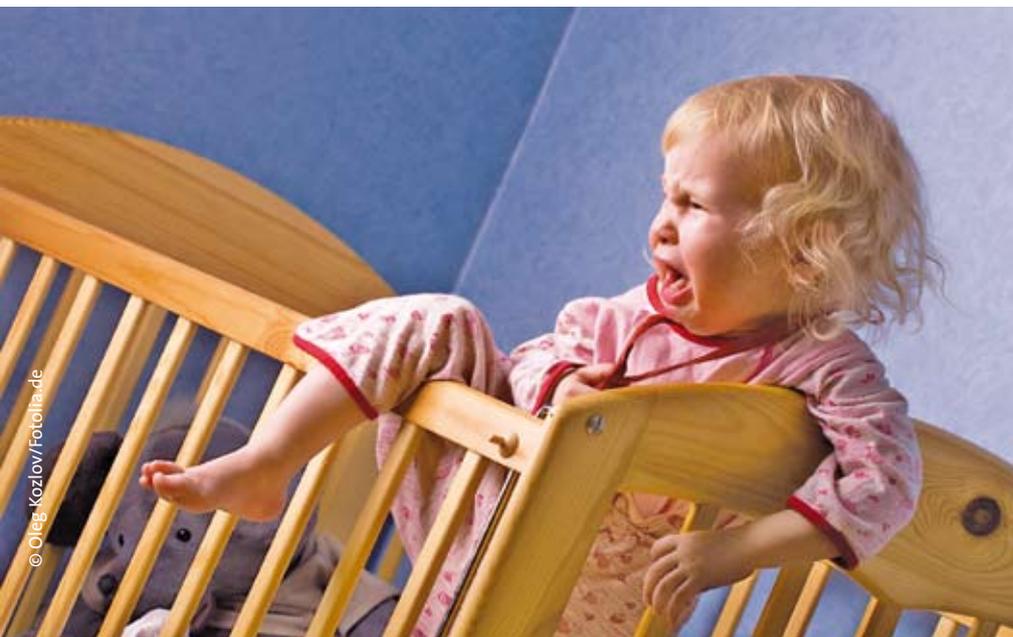
In early childhood, it may be difficult to distinguish symptoms of ADHD from age-appropriate behavior in active children. Family histories and environmental factors may be more valuable in determining whether treatment should be recommended in children with potential ADHD.

Common symptoms in infancy include the following:

- excessive dribbling
- excessive motor activity
- increased thirst
- head banging

- fits
 - tantrums
 - screaming
 - restlessness
 - needing little sleep
 - being difficult to feed
 - inability to be pacified
 - spurning affection and cuddles
- Common symptoms in young children include the following:

- clumsiness
- impulsiveness
- being accident prone
- erratic disruptive behaviors
- compulsive touching
- constant motion
- nonstop and repetitive talking
- concentration deficits
- loud talking
- restless sleep
- nightmares
- being oversensitive to odors, lights, sound, and cold



An early diagnosis of ADHD is difficult. Symptoms in infants and toddlers include restlessness, frequent crying and fits of anger during which the child cannot be pacified.

Treatment

Prenatal neurotoxin exposure sets the scene for neurochemical imbalances in the newborn and highlights the need for drainage and detoxification, even in young children. Inherent tendencies to the development of illness may be genetic or the result of environmental toxin exposure.

Young children will all benefit from a two- to three-month course of Lymphomyosot (2-5 drops of each given 3 times daily). In more toxic environments (because of environmental or medicinal exposure), deeper detoxification protocols may

be necessary, with ampoule preparations such as Thyroidea compositum and Pulsatilla compositum (1 dose biweekly). Prenatal or postnatal heavy metal exposure is an indication for the use of biocatalysts and the corresponding low-dose metal-containing product (i.e., bioregulatory products that contain mercurius, lead, and arsenic). Lead exposure is an indication for Placenta compositum and Cerebrum compositum. The use of Cerebrum compositum is essential in all cases of potential brain injury (traumatic or toxic) in the young child.

Omega-3 fatty acids exert direct and indirect influences on neurotransmission through modifications at the postsynaptic receptor. They influence signal transduction by inhibiting the hydrolysis of inositol triphosphate (IP3), an effect that closely resembles the activity of lithium. Essential fatty acids also inhibit membrane phospholipase activity¹⁴ and reduce arachidonic acid release from neuronal cell membranes.¹⁵ A deficiency of omega-3 fatty acids has been linked to low dopamine receptors in rats¹⁶; there is a direct correlation between a low plasma or membrane-bound essential fatty

Table: Specific Treatment Protocols for Children With ADHD.†

	Young Children With Low Serotonin Levels	Young Children With Low Dopamine Levels
Presentation	<ul style="list-style-type: none"> • Sleep disturbances • Erratic and changing moods (often crying with screaming and uncontrolled tantrums) • Compulsive repetitive behaviors • A history of depression is often seen in 1 or both of the parents 	<ul style="list-style-type: none"> • Listless behavior and possible delays in reaching milestones • Low metabolic rates may be seen, with a tendency toward constipation • Children rock back and forth to music (because they love rhythmic sounds), with sensitivity to loud and unexpected sounds • There may be a family history of addictive disorders
Treatment	<ul style="list-style-type: none"> • Nervoheel tablets (half a tablet 3 times daily) • Ignatia-Homaccord drops (2-5 drops 3 times daily) • Viburcol suppositories (to be used as needed for tantrums and sleeplessness) • Thalamus compositum ampoules (1 dose every evening at sunset for 1 week for sleep disturbances) 	<ul style="list-style-type: none"> • Neuro-Injeel ampoules (1 dose twice weekly) • Barijodeel tablets (half a tablet 3 times daily) • Lymphomyosot drops (2-5 drops 3 times daily) • Calcoheel tablets (half a tablet 3 times daily) • Bacillinum-Injeel (1 dose weekly if indicated in unresponsive cases)

† The dosage of certain medications may vary depending on the age of the child. Please refer to the respective package insert.

Young children with ADHD find it hard to focus on one thing at a time. Constantly in motion, they are somewhat clumsy and tend to have more accidents than their healthy age peers.



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acid level and depression.¹⁷ Modulation of sodium and calcium neuronal channels and reduction of electrochemically excitable tissue are major neuroprotective mechanisms of omega-3 fatty acids. These fatty acids are essential for neural development in utero, and preschool-aged children need omega-3 fatty acids for all of the reasons specified. Children may show overt signs of fatty acid deficiency, such as allergy development, indicating T-helper cell type 2 dysregulation; and excessive thirst levels and dryness of the mucous membranes, conjunctiva, and skin as the result of cellular membrane phospholipid breakdown.

Concentrations of omega-3 fatty acids, particularly docosahexaenoic acid (DHA), increase 3- to 5-fold during the last 3 months of pregnancy and by the same amount during the first 3 months of life. The accumulation of DHA in the brain continues for at least the first 2 years of postnatal life. The optimal recommended daily intake of omega-3 has not been established and may vary from person to person.

Recommended daily intake:

- Children aged 2 to 4 years: 500 mg of omega-3, with a minimum of 125 mg of DHA and 30 mg of eicosapentaenoic acid (EPA)

- Children aged 4 years and older: 1000 mg of omega-3, with a minimum of 250 mg of DHA and 60 mg EPA

As signs and symptoms progress and differentiation between low serotonin and low dopamine levels in infants and children become apparent, specific treatment protocols can be approached (Table).

More specific protocols for the classic ADHD symptoms in school-aged children and the difficulties they face in learning will be addressed in a future article. ■

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