

# **The Case Against Drugs - and For Neurofeedback, The Superior Alternative for Attention Deficit/Hyperactivity Disorder (ADHD)**

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The majority of practitioners treating ADHD use one or more drugs to treat it. This preference is based on their belief that the drugs are effective and safe in the treatment of ADHD. I will review the abundant evidence that often drugs are ineffective, that they often have unfavorable side effects, and that there are long-term problems associated with the use of stimulants. At best, they do not eliminate ADHD, but temporarily suppress the symptoms. Complications may be quite severe with their long-term use.

In contrast, neurofeedback is usually effective in eliminating ADHD altogether, usually without side effects or long-term complications. It is also useful in eliminating comorbidities associated with ADHD, such as learning difficulties, anxiety, depression, oppositional defiant disorder, sleep disturbances, and anger control issues. There is no problem with withdrawal or drug interactions. It offers a permanent solution in the majority of cases. In the long run it is less expensive than prolonged drug usage.

The alerting and euphoric effects of amphetamines were discovered in the 1920s (1). College students in the 1930s recognized they could be used to relieve fatigue and increase focus when cramming for final exams. Benedrine inhalers were banned in 1959 because of an epidemic of amphetamine abuse. Methylphenidate (Ritalin®) was synthesized in 1944. It was found to decrease ADHD symptoms in institutionalized children first, and then in school children. With special interest group support (the learning disability industry, the creation of DSM-III and its revisions, and pharmaceutical industry front groups like CHADD and NAMI), stimulants such as dexedrine and methylphenidate were approved for management of ADHD in children and adults. Pemoline is no longer available because of the risk of liver toxicity. Atomoxetine (Strattera®) has recently also been associated with liver toxicity in some cases.

Animal research indicates that chronic administration of stimulants leads to many adaptive changes in the brain, including a persistent reduction of dopamine transporters in the striatum, a decrease in dopamine D2 receptors, alterations in gene expression (C-fos), and morphological changes, including dendritic lengthening, branching, and increases in cell density in the nucleus accumbens and frontal cortex (key areas in learning and addiction). These changes may result in interference with the acquisition of new skills and behaviors. They may be the bases of craving and addiction and/or psychotic features which occur in some individuals during periods of extended drug abstinence. PET studies have confirmed a reduction of D2 receptors and dopamine transporters in the striatum of human children. Chronic Ritalin® use, followed by drug withdrawal, may induce a significant worsening of the original condition.

Many children develop tics during active treatment. Another long-term effect of stimulant therapy is heart disease, including occasional sudden death. Anxiety and irritability are common side effects, and severe depression may be a consequence. Appetite suppression (1 in 4), insomnia (1 in 7), and stomach ache (1 in 9) occur in significant numbers of children. Most trials of stimulants have been short-term, acute studies. Only a few long term studies have been done. The best known of these is the NIMH Multimodal Treatment Study (MTA). Subjects remaining on treatment experienced a slight deterioration of medication effect. Only 68% of subjects remained on medication at 24 months. There was a significant worsening of symptoms when the drug (Ritalin®) was stopped. Both parents and teachers rated behavioral therapy as superior to medication. Studies have shown that treatment with stimulant drugs does not reduce the incidence of conduct disorder and antisocial personality disorder seen in many ADHD children as they grow older. A study in Australia found growth retardation, with progressive declines in both height and weight in 86% of the subjects on Ritalin®. In a Yale study of children on Ritalin® for 2 years or more without interruption, 76% of the males and 90% of the females experienced significant height suppression after 3 years of therapy, with an overall height deficit of 3-4 cm (1/2 – 1 1/2 inches). There was an average weight loss of 1.25 kg (2.75 pounds) per year. Unmedicated patients demonstrated above average growth rates when the Ritalin was discontinued.

Most long-term studies have shown a waning of the beneficial cognitive effects of stimulants over time. Seven per cent of stimulant-treated children in one Canadian practice developed clear symptoms of psychosis after the initiation of therapy. The psychosis resolved on discontinuation of therapy in most of these patients. Many ADHD children on stimulants experience withdrawal symptoms (moodiness, excitability, insomnia, and excessive talk). This may result in the child being labeled as “bipolar,” when in fact the problem is iatrogenic. The psychiatric literature has consistently minimized the addictive potential of psychostimulants. The reinforcing effects of intravenous Ritalin® are identical to those of intravenous cocaine. In a high dose (80 mg.), oral Ritalin® may produce levels of dopamine transporter blockade as quickly as intravenous doses.

Animal studies have documented that stimulant medications sensitize the brain to cocaine. In a study of 492 children begun in 1974, stimulant-treated subjects developed higher rates of cocaine dependence than non-medicated peers diagnosed with either ADHD or behavioral (conduct) disorders. The abuse potential and sensitizing effects of Ritalin® are supported by over 60 studies of nonhuman and human subjects.

In the large MTA study quoted above, medicated and non-medicated children were given behavioral therapy, including classroom training, sports, social skills training, daily group sessions, tasks to promote cooperation, parent training with reinforcement systems, and daily report cards. After eight weeks of the therapeutic camp the two groups showed no differences in overall improvement ratings. Other non-pharmacologic approaches have documented similar benefit, including exercise (martial arts, massage therapy, music therapy, and cognitive therapy). Direct comparisons of Ritalin® and neurofeedback have shown equivalent efficacies (2, 3). The effects of neurofeedback persist when the training is completed. When the drug is stopped, the child (or adult) returns to his/her baseline state.

Neurofeedback offers the additional advantage of remediating the comorbidities of ADHD—anxiety, depression, learning disabilities, oppositional defiant disorder, conduct disorder, sleep disturbances, and anger control. QEEG-guided neurofeedback is very helpful in remediating these associated disorders (4, 5). Adverse effects of neurofeedback are extremely rare and no long-lasting complications have been reported (6). There appear to be no withdrawal effects. Dependence has not been reported. There have been no studies reported of the incidence of drug abuse or criminal behavior in ADHD children remediated with neurofeedback, but we know that proper neurofeedback can be used to reduce drug-seeking behavior and aggression in incarcerated individuals with ADHD (7). I suspect that we will see a decrease in drug-seeking behavior and criminal behavior in neurofeedback-treated children with ADHD as they grow older. We need to collect data in this regard.

The studies reviewed here indicate that neurofeedback treatment is a superior alternative to stimulant therapy in ADHD. It is usually curative of ADHD. It is extremely safe. Over the course of a lifetime it is less expensive than the drugs. In truth, it is a revolutionary approach to managing ADHD. We urgently need to educate our neurological, psychiatric, and pediatric colleagues about the advantages of neurofeedback.

#### List of References

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