Rebound

ADHD and the Impact of Stimulant Medication

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According to recent data from the Centers for Disease Control and Prevention, a staggering 12 percent of all boys in the U.S. are diagnosed with ADHD. This number may rise with DSM-5 guidelines increasing the age from which symptoms must be present from 9 to 12 years (American Psychiatric Association, 2013). With ADHD diagnosis at such endemic proportions, the importance of understanding the impact of stimulant medication on behavior, and by extension on family function, cannot be over-stated.

Many children prescribed stimulant medication experience improvements in self-regulation, in turn leading to gains in social and family function, and improved self-esteem (Coghill, 2010). But stimulant medications are broad-acting, often producing side-effects that both complicate treatment decisions and raise more fundamental, quality of life questions regarding both treatment outcome and medication compliance (Spitzer et al., 1995).

Parents whose children take stimulants are often all too aware that ADHD symptoms appear to return with renewed vigor each morning before children take their medication, and later, as medication wears off. This "re-bound effect" is often exacerbated by the appetite suppressing side effects of stimulant medication (Efron, Jarman, & Barker, 1997). ADHD children habitually forfeit their midday meals, and often experience their appetite return with a fuel-deprived intensity, leading to poor food choices and binge eating. Significantly, for parents and their mental health providers, the dramatic return of ADHD symptoms is often seen as a confirmation of the need for medication itself and further, the justification for additional medication.

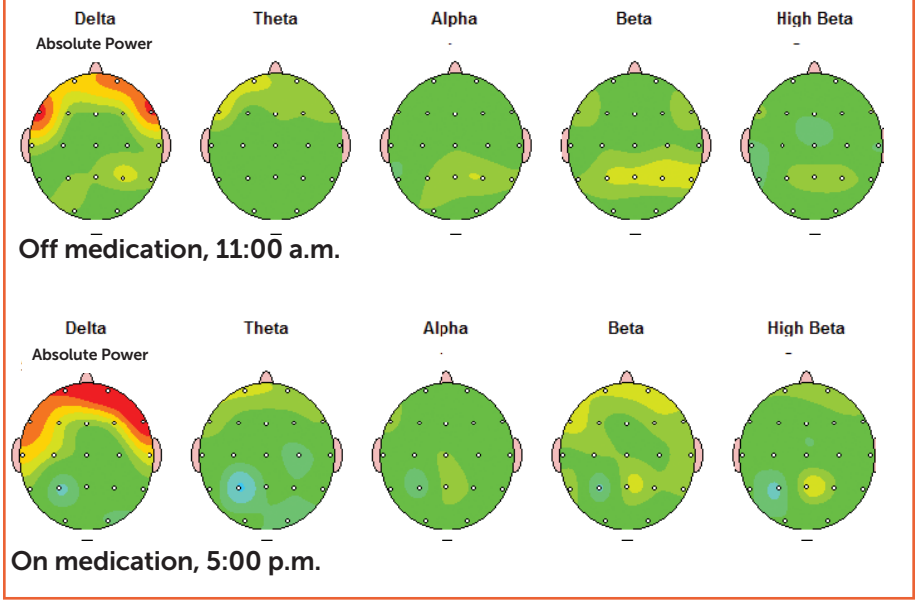
Since many children are medicated, at least in part, due to their emotional disregulation at home, the ability to differentiate between behaviors that are primary to the child, and those that are a consequence of their stimulant medication, should be a vital component of clinical decision-making. Such questions are hard to answer empirically, since we measure behavior observationally in a constantly changing environment. However the use of brain mapping technology offers the intriguing possibility of linking neuroscience with family therapy; allowing behaviors observed within the family to be correlated with measured cortical activity, and for treatment decisions to be made with greater accuracy. In the following case of a boy named "Nick," this approach was contrary to the prevailing push towards increasing medication, but nevertheless improved treatment outcomes at every level. More importantly, this case raises broader, systemic questions regarding stimulant medications that are rarely asked.

By the time Nick, 11 years old, was referred to us for brain mapping and neurofeedback, his parents were at their wits' end. His family was dominated by the strain of parenting him, and typically for parents of ADHD children (Wymbs, Pelham, Gnagy, Molina, & Greenhouse, 2008), their marriage was under significant strain. Nick's parents described wild and irrational rants during which he was verbally and physically violent, especially at home. As Nick grew bigger and stronger, there were increasing concerns for his mother's physical safety. Unsurprisingly, Nick's psychologist was considering adding oppositional

Figure 1

May 27, 2013 (Standard Deviations from the norm)		June 14, 2013 (Standard Deviations from the norm)	
left pre-frontal lobe	1.81 SD's	left pre-frontal lobe	2.66 SD's
left frontal lobe	2.69 SD's	left frontal lobe	2.39 SD's
right pre-frontal lobe	2.18 SD's	right pre-frontal lobe	3.18 SD's
right frontal lobe	2.58 SD's	right frontal lobe	3.06 SD's

Figure 2



What we actually recorded was a "rebound effect" as Nick's brain struggled to adapt to stimulant withdrawal.

defiant disorder (ODD) to his existing ADHD diagnosis—and from his parents' description of his behavior, the decision seemed entirely justified. Like many parents in their position, Nick's parents were asking what medications might stabilize his volatility.

We decided to map Nick's brain twice: the first time when he'd been off stimulant medication for a number of days, and the second time when he was on medication, in order to assess its effectiveness. Our findings at first seemed counter-intuitive, but later proved to be important.

The first mapping took place at 11:00 a.m. on May 27, 2013. Nick had been medication free for 72 hours, but was calm and appropriate. Four-minute EEG recordings were taken from 19 standard scalp locations, in both eyes open and eyes closed conditions. The results were compared with a normative database of individuals of similar age, sex and handedness, and with no history of neurological or behavioral disorders. (A similar process was approved by the FDA in July 2013 for the diagnosis of ADHD, in which the ratio of slow brain waves to faster "beta" brain waves was found to be a diagnostically valid predictor of ADHD.) The results showed a relative excess of slower brain activity (delta waves) in Nick's frontal lobes consistent with his original ADHD diagnosis.

The second mapping took place at 5:00 p.m. on June 14, 2013, and was quite different. Nick had taken his stimulant that morning, but typically for days when he took his medication, he hadn't touched his lunch, and had hardly eaten his breakfast. At 5:00 p.m., our session was dominated by his ravenous hunger. More importantly, the frontal slowing we measured when he was off medication was considerably worse on the afternoon that he'd taken his medication (see figures 1 and 2).

Why would Nick's brain look worse on stimulant medication than it had without it? The answer appears to lie in the time of day the second recordings took place, whereby what we actually recorded was a "rebound effect" as Nick's brain struggled to adapt to stimulant withdrawal.

For Nick and his family, these findings were profound. Like many children diagnosed and medicated for ADHD, Nick's issue was not his academic performance (which was good), but rather his impulsivity and emotional disregulation. However, the implications of the brain mapping were that Nick's medication appeared to be most active at school (where it was needed least), but actually detrimental in the environment in which it was needed most—the home. Furthermore, it seemed highly likely that Nick's irritability was exacerbated by low blood sugar caused by his loss of appetite.

We recommended that Nick take a break from his medication to assess the impact on his mood volatility. To date, his father has reported no major outbursts, and that the family is functioning much better.

To be fair, it's important to recognize that Nick still meets the criteria for

ADHD, as reflected by his original diagnosis and first ("off medication") brain map. It's also important to recognize that Nick is one case, and we cannot infer that all children taking stimulants are functionally worse off when their medication wears off than if they had taken none at all. But it does raise broader questions regarding stimulant medication and family function.

If the "rebound effect" does exist (and we believe it does), how frequently do parents mistake stimulant withdrawal, and its impact on the family, as evidence for stimulant need? To what degree does appetite suppression caused by stimulant medication contribute to the impulsivity and erratic mood that often characterize ongoing ADHD diagnoses? Are parents given a complete explanation of the effects of stimulant medication before their children embark on its long-term use?

This case speaks to a societal view on how we approach our children—on what is, and is not, considered acceptable in our children, and what we are prepared to tolerate. It seems that while medication can and does help many children, there are clearly times when our unquestioning faith in the medical model is not just misplaced, but counterproductive.

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