

Neurofeedback in Application to the ADHD Spectrum

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1.0 Introduction

The ADHD spectrum has been the primary clinical application of neurofeedback for over thirty years. In Chapter 2, review of the early research history established that the traditional SMR-beta protocols of EEG biofeedback were quite effective in managing the canonical symptoms of ADHD. Our own role in that development is covered in detail in two book chapters dating back to 1999.^{1,2} Six comparison studies have now been done that unanimously find an essential equivalence between EEG training in the classical manner and state-of-the art pharmacological management. These comparisons typically relied strongly on the results of continuous performance tests of attention (CPTs), which primarily test for inattention and impulsivity. These tests don't give us a handle on the hyperactivity component or distractibility, for which one needs to rely on the observations of parents, teachers, or trained observers. These have their obvious shortcomings. Nevertheless, the essential findings are no longer in any doubt. Neurofeedback is competitive with standard medical treatment in the management of ADHD.

In recognition of this substantial body of evidence, the American Academy of Pediatrics rated neurofeedback as having Level 1 efficacy in application to ADHD.³ (Under political pressure, the AAP subsequently softened this recognition without further investigation. Research support for the original assignment was never called into question.) In support, recent brain imaging research was cited in addition to the clinical studies.⁴ This research documents the impact of neurofeedback training on the functional connectivity of neuronal networks. More recently, the adjudicating body in German psychiatry has likewise given recognition to neurofeedback in this application. The effect will be the gradual recruitment of neurofeedback into the arsenal of remedies for ADHD around the world.

The existing model of ADHD, however, remains in place essentially without alteration. The more ground-breaking implications of the effectiveness of neurofeedback in application to ADHD have yet to be recognized. The clinical reality, as experienced by neurofeedback practitioners, cannot be accommodated within the prevailing model of ADHD. By and large, the clientele that seeks out neurofeedback for ADHD is not looking merely to replace medication in the therapy. Neurofeedback is sought out because the medications are not resolving the issues that are actually compromising the child's life, or the side effects are not tolerated, or because the impetus is a related symptom such as bed-wetting, nocturnal bruxism, scary dreams, persistent head or stomach pain, or conduct problems. And yet these are clearly ADHD children that have been medically diagnosed.

The case can be made that the very success of the medical remedies has resulted in a co-development of the conception of ADHD as being whatever is successfully treated with the medical arsenal, most typically stimulants. ADHD is what the medications treat. This has resulted in a kind of diagnostic tunnel vision that is unlikely to be called into question because it is working as intended. The synergy between the model and the remedy has led to ever more elaborate efforts to shore up the model rather than to move beyond it. There is a reluctance to recognize ADHD as the complex syndrome that it is. Developmental precursors

are not given the attention they deserve. The complex etiology is not taken into account. The emotional context of attentional deficits has been completely ignored. There is no nexus between the behavioral model and emerging neurophysiological models of brain regulation.

This has not been an issue just for ADHD. With the development of the DSM-5, the initial objective was to seek more congruence with emerging neurophysiological models, as well as to increase reliance on biomarkers for diagnostic specificity. This nobly motivated effort failed, however, because the DSM model was hopelessly paradigm-bound. Plainly, the phenomenological basis of the DSM formalism ‘does not carve nature at its joints.’ The diagnostic partitioning does not line up with any biologically based organizational schema.

The clinical experience of the neurofeedback practitioner mandates a more inclusive conception. This is the *Dysregulation Model*. In this model, ADHD is seen as a disorder of cerebral dysregulation that crosses diagnostic boundaries and affects multiple systems. In this model, attentional dysfunction becomes a mere observable for the core dysregulation, one among many such observables. The issue can be illustrated with the following example. Within the classic DSM framework, Oppositional Defiant Disorder is comorbid with ADHD in some 60% of cases.⁵ Since stimulants do not address this comorbidity, the dichotomous view is likely to be sustained. The more organic view would have it that emotional dysregulation clearly cohabits quite commonly with attentional dysregulation. One clearly impinges on the other. Emotionally-based disorders often lie at the core of attentional disorders, and in such cases should have priority in therapeutic attention. A common mechanism may even underlie both. Alternatively, sensory processing disorders or specific learning disabilities may lie at the root of the apparent attentional deficit. Conversely, the attentional deficits seen in ADHD are ubiquitous in other clinical conditions.

The need clearly exists for us to move beyond a purely behavioral and phenomenological model. Since neurofeedback engages neural network organization directly, the canonical disorders need to be understood in the new framework. That project is aided by having a neurophysiological measure to go along with our neurophysiological remedy. The Continuous Performance Test (CPT) that has been in use in our work since 1990 serves this purpose admirably. Whereas the results of CPTs have historically been interpreted in behavioral terms, they can equally well inform us with respect to behavior at the level of the neuronal networks. Refinements and new developments over the last fifteen years have substantially increased the utility of the test.

At the same time, the clinical decision-making by the neurofeedback practitioner remains based largely on behavioral and phenomenological observations, as these are available in real time. The distinction being drawn here is that a more physiologically grounded model allows the behavioral observations to be appraised differently. A more inclusive perspective is called for, one that accommodates not only the pharmacological model of ADHD but also the much broader implications of infra-low frequency neurofeedback. In essence, a few key failure modes are identified that line up with the basic training protocols. These failure modes exist on a continuum, and at some level they afflict us all at one time or another, in sickness if not in health. Hence, they are no respecter of diagnostic boundaries. These basic failure modes are developmentally grounded. We all start out in life inattentive, distractible and impulsive. Children only become capable of taking the CPT test during their fifth year, by and large. Full maturity is not reached until the twentieth year.

As described in Chapter 4, the clinical model is informed by considerations of functional neuroanatomy and is subject to selection through empirical findings. The results are consistent with current understanding of our intrinsic connectivity networks (ICNs), or resting state networks (RSNs). The quality of brain function is contingent on the steady-state functional connectivity of our ICNs, as well as on their dynamical interaction.⁶ This is discussed further in Chapter 2 and 3.

The study of intra-individual variability of reaction time performance under various challenge conditions has revealed fluctuations in the range of 0.01 to 0.1 Hz.⁷ Another such study found correlations between responses on the timescale of one second.⁸ The study of event-related potentials (ERPs) under such challenges has shown differences on the timescale of 10msec.⁹ Each of these time domains can be targeted with a neuromodulation strategy. However, empirically it has been found that the broadest impact can be achieved by targeting the steady-state behavior of functional connectivity in the infra-low frequency range. This engages the foundations of our regulatory hierarchy.

Developments in the measurement of CPT data have enabled efficient collection of group data. These will be presented to consolidate the case for ILF NF in application to the classic symptoms of ADHD. The case histories that follow are intended to illustrate the breadth of impact of the method, and the particularity of its administration in each case. This supports the view that ADHD should be seen as a syndrome or spectrum. Heavy reliance is placed on CPT data in these case reports.

2.0 Refinements in the analysis of CPT data

All of the CPT data we published in the nineties was acquired with the Test of Variables of Attention (T.O.V.A.®). This pressured choice reaction time (Go/No-Go) test involves two periods of stimulus-sparse challenge, followed by two periods of stimulus-intensive challenge. The inter-stimulus interval is invariant at two seconds in order to maintain uniformly boring conditions throughout. This presents a challenge to the maintenance of vigilance. Data analysis was based on Gaussian distributions, and therefore contingent on norming populations that were in turn dependent on expert judgment.

In 2004 we acted upon the perceived need to make minor modifications in the test design that the T.O.V.A. team was not able to accommodate, and therefore new hardware and software were developed to emulate the T.O.V.A. design. The result was the QIKtest,¹³ developed by bee Medic in Switzerland. The basic features of the T.O.V.A. were preserved in the new design, even to the point that the norms carried over as well. The QIKtest provided for the return to a single period of stimulus-sparse challenge, in order to have an additional state shift to evaluate. Also, the threshold criterion for an anticipatory error was shortened from 200msec to 150msec for adults, and it was age-adjusted for the young.

All subsequent data were stored on a central server, which then facilitated efficient evaluation of large data sets in systematic data mining. Analysis of reaction time data revealed reaction time outliers to be power-law distributed (i.e., $1/t^\alpha$). They would have to be considered separately so as to avoid contaminating the ‘normal’ reaction time data, known to be described by the ex-Gaussian distribution, the Gaussian with an exponential tail. These surveys also revealed that the discrete errors were not Gaussian-distributed and could in no way yield to parametric analysis, i.e. in terms of mean and standard deviation. Not only were the distributions long-tailed, but in the case of omission errors and reaction time outliers they were ‘all tail’. That is to say, these were power-law distributed over the entire range. Commission error distributions could be fitted to a Gaussian component with a power-law tail.

The practical implications were multi-fold. As parametric analysis of the discrete error data was contraindicated, non-parametric analysis was mandated, with scores expressed in percentiles. To flesh out the entire distribution in order to make percentile scores meaningful, a large sample pool was required. Population-based norms were adopted for this purpose (as is customary in IQ-tests), with only the pathological extremes excluded from the analysis. The resulting norms are therefore relatively free of arbitrary human judgment. They were also fairly low in statistical uncertainty, being based in this case on a sample of 70,000 trials, with a sample size of 500 for each age and gender bin from age six to 70+.

There were theoretical implications as well. All three types of discrete errors were power-law distributed, which indicated that they were all subject to a chaotic failure mode. All three were also statistically highly correlated. They all scaled with dysregulation status. Thus, a common underlying failure mode could be reasonably postulated, enhancing the prospects that a small set of protocols could serve this disparate variable space adequately.

3.0 Results for the Evaluation of Group Data for ILF Neurofeedback

In 2017 a survey of training outcomes was performed in which 12,200 pre-post data sets were evaluated for a nominal twenty sessions of ILF NF. The survey included the contributions of more than a thousand clinicians to the database, and hence are representative of what is actually being accomplished in the clinical realm. The data were analyzed independently of any information about the clients beyond age and gender. Hence the results reflect attentional failure generally in the clinical population, not just among those labeled ADHD. However, that is in line with our present understanding, namely that one or more common failure modes are involved that cut across clinical boundaries.

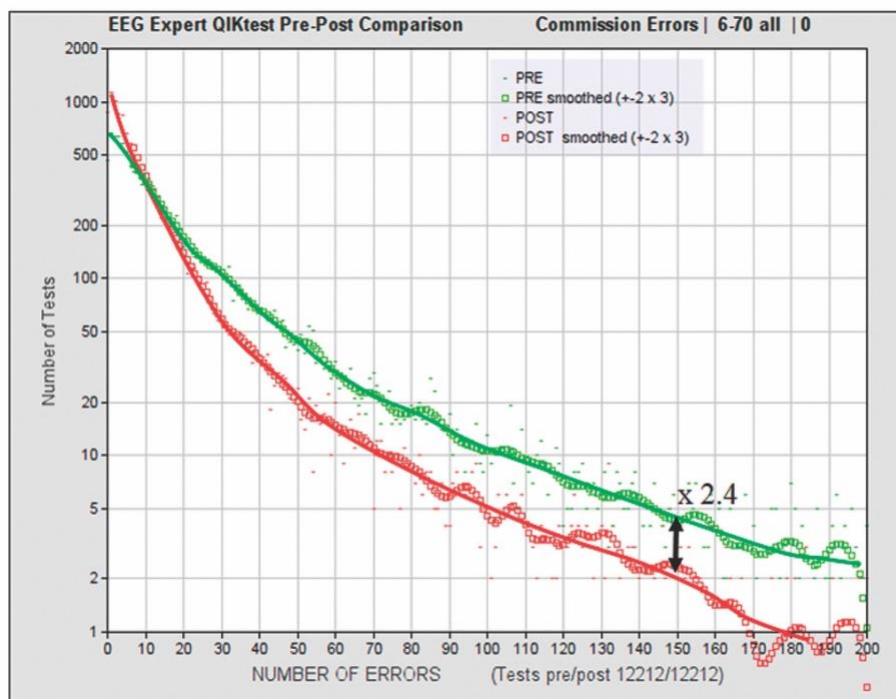


Figure 1. Incidence of commission errors before and after nominally twenty sessions of training. Shown is the number of tests in the database for each value of the number of commission errors per test. The number of tests is plotted logarithmically.

Results for the pre-post distribution of commission errors is shown in Figure 1. The number of tests showing a particular number of commission errors is plotted versus the number of errors. The results indicate that improvements with training are achievable at any measurable level of deficit. If a child is capable of taking the test, improvements with training are the expectation. An average improvement by a factor of 2.4 characterizes the severely deficit region. A similar curve for omission errors reveals an improvement factor of 1.8. For reaction time outliers, an improvement factor of 1.4 is indicated.

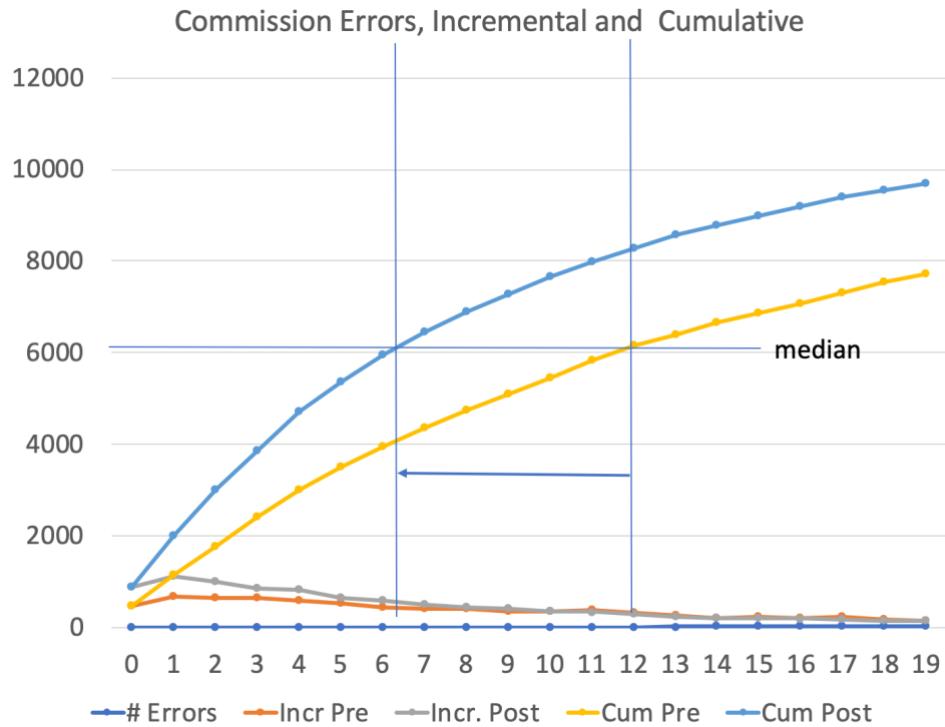


Figure 2. Incremental and cumulative distributions for commission errors, before and after nominally twenty sessions of training. The median score improves from 12 to 6.3, an improvement factor of 1.9.

The cumulative curve of commission errors is more revealing of what happens with the bulk of the population. This is shown in Figure 2. The median number of commission errors declines in training by a factor of 1.9. The comparable factor for omission errors is 2.4, and for reaction time outliers, it is 2.1. The results are summarized in Table 1.

Table 1. Improvement factors for error incidence in highly deficit region, and in median score.

Improvement Factors	Commissions	Omissions	Outliers
Error Incidence	2.4	1.8	1.4
Median Score	1.9	2.4	2.1

The improvement factors are substantial, leaving no doubt about the robustness of the findings. The clinical significance of these improvements is not yet apparent, however. For that purpose, age-segregated data are drawn upon to fill out the picture. The tenth birthday was chosen to divide the early and late developmental periods. The twentieth birthday is the appropriate end point to the developmental phase. Conveniently, the normative data allow the median scores to be interpreted in

terms of equivalent mental age. The results are shown in Figures 3 and 4 for the two age ranges, and for all four of the primary assessment categories of the CPT. Regarded in these terms, the functional improvements are seen to be quite substantial for the discrete errors, albeit more modest for reaction time and variability. That is entirely in line with clinical objectives.

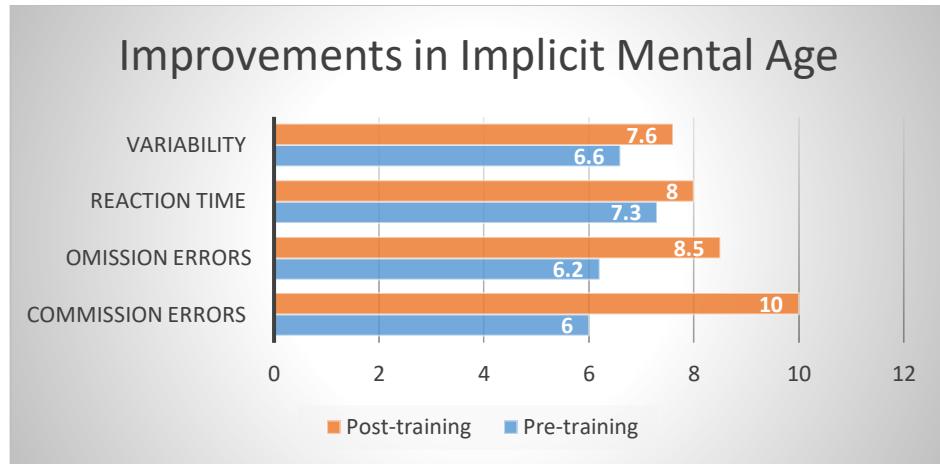


Figure 3. Improvement in implicit mental age for the principal measures of the CPT, based on the median score in the respective cumulative distribution. These data refer to the cohort that had not yet reached the tenth birthday.

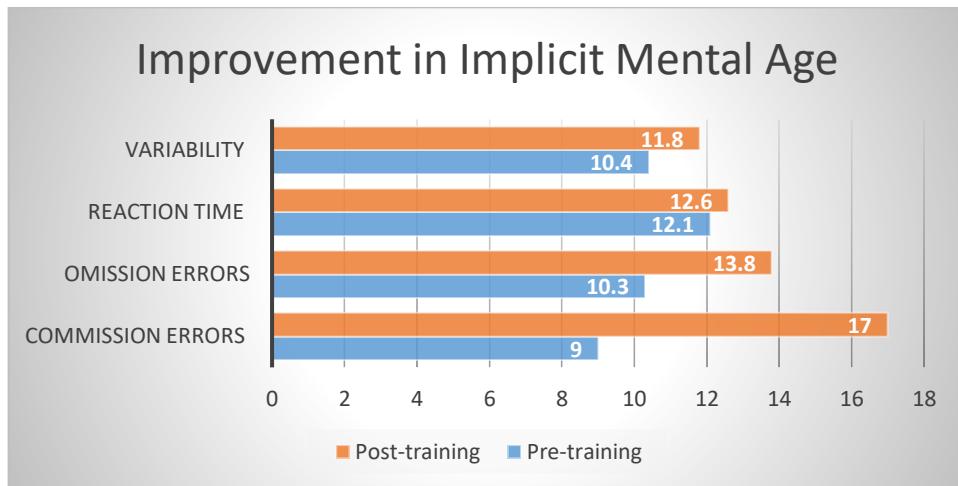


Figure 4. Improvement in implicit mental age for the age cohort of 10-19 years, inclusive, for the principal measures of the CPT.

The problem being addressed is much more the context out of which the action arises, as opposed to the execution of the action itself. The distinction between the two is particularly apparent in Parkinson's, and it's the heart of the story in Oliver Sacks' *Awakenings*. In the CPT we arrange for the most benign of circumstances. The challenge is a minimal one; the task can be readily anticipated; the choice is binary; and there are no external distractors. The failures thus identified therefore trace back to the brain's inability to maintain continuity of state, to random internal disruptors, or to both. These two failure modes are the immediate targets of the ILF NF training process, and substantial success in that undertaking is in evidence.

Finally, it is of interest to inquire whether results have improved over the years with the ongoing refinement of protocols. For this purpose, the ten-year survey was segmented with the dividing line of the end of 2013. Comparisons are shown in Table 2 in the form of ratios of improvement factors for the two epochs. There have been improvements in outcomes with respect to commission errors and reaction time outliers, whereas results have been stable for all the other categories. These results testify to the existence of a robust training protocol with relatively predictable outcomes at least in the statistical sense.

Table 2. Ratios of improvement factors for post-2013 data vis-à-vis pre-2014 data.

Ratios of Factors of Improvement	6 through 9 yrs	10 through 19 yrs	20 years to 70+	Average ratio of improvement factors
Commissions	1.12	1.18	1.09	1.13
Omissions	0.97	1.03	1.03	1.01
Outliers	0.96	1.11	1.22	1.10
Reaction Time	0.98	0.99	1.00	0.99
Variability	0.97	1.02	1.00	1.00

In the remainder of the chapter we shift toward a more clinical perspective on the performance of this work, and the variety of symptom profiles that can be resolved with ILF NF.

4.0 Why Diagnosis is Not Important from a Neurofeedback Perspective

Neurofeedback exercises the brain into self-regulation and improved function, irrespective of the modality. In ILF NF, the training is covert, and hence the exercise is self-generated by the brain in engagement with the signal. With proper protocols, improvements in performance should be achievable irrespective of baseline performance when initiating neurofeedback. There is usually a set of symptoms to work with that cues us with respect to self-regulatory status, and that set of symptoms might or might not fit into the diagnostic criteria for a particular condition. Even if nothing rises to the level of overt symptoms, there are relative strengths and weaknesses that may be discerned. Parents are often concerned that their children won't get the training because they don't have a formal diagnosis, and we quickly lay those concerns to rest. From a neurofeedback perspective, the more important aspect is "who is this person that is presenting with these symptoms? How is that unique brain affected and constrained, given those specific symptoms? How is it dysfunctional and dysregulated?" The symptoms are the observable manifestations of the dysregulation status that we need to learn about and impact with the training.

A symptom is necessarily subjective, observed and appraised by the patient, and typically it cannot be measured directly or with much accuracy. Therefore, the same symptom is going to be perceived differently by any two affected individuals. This is not, however, a lamentable limitation. On the contrary, the specifics of how this symptom manifests in one brain or another is the information we need in order to decide how to train. The particularity and the context of symptom presentation are keys to the underlying pattern of dysregulation.

Having a diagnosis merely orients us towards one set of symptoms or another, but it doesn't give us any specifics on how those symptoms are related at a deeper level, of the pattern of dysregulation that is affecting that brain and body. At the same time, not having a diagnosis simply means we get the

information we need in the form of a list of symptoms the client describes during the intake. We then map those symptom patterns to principal failure modes of cerebral regulation, which in turn map to our training sites. We then lay out a training protocol that will target the identified failure modes, which implies that symptoms are being targeted only indirectly. The client will experience the training and will be able to notice and report on changes in symptoms as well as on shifts in functional status. These changes and shifts help us to understand in what way the training is affecting brain function, and we make continual adjustments to the training protocol to optimize results. Such adjustments may be made several times during one of the early sessions, before the training protocol settles down to a more predictable pattern.

5.0 The ADHD Spectrum in the Clinical Perspective

The clinical model distinguishes between two principal subtypes of ADHD for purposes of structuring a neurofeedback protocol: the *simple subtype* and the *complicated subtype*. The simple ADHD subtype is well described by the cardinal symptoms of ADHD: inattention and distractibility on the one hand; impulsivity and hyperactivity on the other. This pattern indicates the need to train two main areas in the brain: left prefrontal cortex and right parietal. This bi-hemispheric strategy has served us well for many years, going back to the mid-nineties. It is traceable to the model of ADHD of Malone, Kershner, and Swanson,¹⁰ which in turn is based on the model of Tucker and Williamson.¹¹ Representative clinical results obtained with the earlier SMR/beta training protocols at some 32 clinical practices are covered in Kaiser and Othmer.¹²

The left prefrontal cortex is a critical part of the executive control system that refers to directed attention, planning, reasoning and judgment. It is involved in voluntary behaviors such as decision-making, planning and thinking, internally motivated attention and inhibition of impulsive and compulsive behaviors. Good prefrontal function allows us to memorize information while planning and executing appropriate sequences of actions to achieve concrete goals. It also is crucial to good self-regulation by inhibiting primitive and immature reactions while time is allocated to consider possible outcomes and consequences of alternative courses of action. Whenever there is a lack of appropriate prefrontal control the person may have difficulty completing tasks, focusing for a longer period of time, and hewing to longer-term goals; the person may also exhibit poor organization skills, and may even be unaware of his own behavior, and unable to consider consequences of behavioral alternatives.

The right parietal cortex plays an important role in integrating information from our senses to build a coherent picture of the world around us. It is involved in visual-spatial processing, spatial and body awareness, orientation of the body in space, and motor coordination on the macro-scale. Impaired function of the right parietal cortex can lead to a lack of self-awareness and spatial awareness, and it can result in the inability of the subject to control body movement, leading to hyperactivity.

The complicated subtype of ADHD includes the above-mentioned symptoms as a result of poor function of the left prefrontal and right parietal cortex but adds physiological dysregulation and emotional symptoms to the picture. (Strictly speaking, ADHD is a disorder of exclusion, and thus the more complicated presentations should not be labeled ADHD. But in the real world, this is what often happens, and that leads to the diagnostic tunnel vision already alluded to. The more complicated aspects may not be attended to because they don't fit the template.) The lack of emotional control resulting in oppositional or aggressive behavior requires training the right prefrontal cortex while the symptoms of instability including headaches, mood swings, asthma etc. require left-right (i.e., inter-hemispheric)

temporal stabilization. It is tempting to surmise that it is easier to work with the uncomplicated subtype. The problem is that the lack of self-awareness in ADHD makes it difficult for clients to report on changes occurring with the training. For the neurofeedback clinician this presents a challenge in making clinical decisions regarding training protocols. The complicated subtype, on the other hand, involves sensitive, touchy nervous systems with an abundance of symptoms that are easy to report on and easy to track. These people are well aware of what is bothering them. Our clinical adjustments to the training protocols are perceived promptly by the client and this helps the process of finding what is optimal. Fortunately, these clients are plentiful in our clinic.

In a clinical setting like ours, people who seek help have typically already tried numerous other modalities to resolve their issues, and that explains why we tend to see the complex cases, where ADHD-related symptoms are part of a bigger picture. For those with uncomplicated ADHD, modern medicine addresses the problem well enough that further help with neurofeedback is unlikely to be sought. It's mostly when clients really want to avoid medication altogether that we get to see people in this category.

There are a few major differences between traditional treatment options and neurofeedback for ADHD. The most important one is the fact that neurofeedback is non-invasive and doesn't put anything into the system, while medication is invasive and has potentially significant, troublesome, and even lasting side-effects. The other difference is that while medication administration is limited by age, neurofeedback can be done at any age. Last but not least, allopathic medicine will consider the severity of the presenting symptoms when deciding on a treatment plan and a certain dose of medication to be administered. With neurofeedback the severity of a symptom is not important in establishing how to train that brain. We also have the ability to be very specific in terms of which areas in the brain we target with the training and exactly how to fine-tune the frequency in order to obtain best results. Medication effects wear off in hours, while the changes promoted by neurofeedback can last a lifetime if sufficient training has been done.

In our assessment process we include the QIKtest in all cases in which the client is capable of taking it. The CPT is a grueling challenge for the young ADHD child. It is administered at age six and up, provided that the client can understand and follow instructions and is able to stay with the task. The baseline test is a consistency check on what we learn about that brain's way of functioning during the intake. The client should also 'recognize himself' in the test results as they are explained to him. Results are presented in the more familiar form of standard scores rather than percentiles. A parent who feels reassured that her child is scoring at norms would be distressed to find out that this refers to the fiftieth percentile. We live in a Lake Wobegon world. Percentile scores are therefore converted to standard scores via the Gaussian distribution. The conditions under which the baseline test is taken (e.g., with or without any medication), will typically be replicated during the comparison test that is done 20 sessions later. It is expected that the second test will show improvement in most areas, with significant improvement expected in areas of initial deficit. Adverse outcomes in one aspect or another compel the training strategy to be redirected.

Oftentimes attention and impulse control issues are part of a more complex scenario that can present under a different name: PTSD, attachment issues, developmental disorder, addiction, depression or anxiety. Our understanding of brain function allows us to interpret symptoms of inattention, impulsivity or hyperactivity in the context of a certain layered and more complicated picture. The CPT test has historically been associated with the characterization of the ADHD brain. But we have put it to use much more universally. It is a test of nervous system status that is very revealing of the capacity for self-

regulation. Whereas it is not prescriptive of training protocols, it is a truth test of sorts to index our approach to the goal of improved self-regulatory capacity.

6.0 Clinical Case Studies

6.1 Case #1

Christine, a 20-year-old woman, sought help for symptoms related to her ADHD diagnosis. During the intake interview a much more complicated picture was disclosed, a picture that included trauma and a history of addiction. Adopted as an infant, she had a normal early life. Her adoptive parents divorced when she was 12 years old and she was raised by her mother, with whom she never really got along. The relationship with her sister, adopted as well, wasn't close until later in life. As her family life became increasingly more stressful, her academic performance suffered. She had been in several car accidents and had pain in her upper back and muscle tension in her neck and shoulders as a result. She had a history of multiple drug addictions which she was able to overcome – she had been sober for two years when she came to us. She described how anything she put into her system had the power to get her hooked – she had abused cocaine, marijuana, and would even use Adderall to get high. Currently she was struggling with new addictions: food and cigarettes. She had suffered one seizure-like episode with a drug overdose. She complained of poor balance and motor coordination and her sense of direction was not good either. She described having difficulties falling asleep and found it impossible to wake up in the morning despite setting several alarms. She also had a history of sleepwalking.

Between the addictive behaviors and immense anxiety, her terrible sleep hygiene and thus disrupted sleep patterns, the obsessive fears of failure or becoming overweight, and the migraines and headaches and severe PMS, the fact that she came in with the diagnosis of ADHD seemed beside the point. She did have difficulties concentrating and had a hard time staying on task, would often zone out and was both hyperactive and impulsive. But, given the fact that her brain had been through so much, can we understand the latter symptoms as consequences of all the different traumas her brain had suffered or just another set of separate issues that happened to be experienced by the same brain? And, does it really matter?

Our improved understanding of brain organization and function has helped us refine our method of training the brain. It is a well-known fact that trauma to the brain means disruption in early development and interference with self-regulatory processes, particularly affect regulation and autonomic function. When core self-regulation is deficient, a person will be unable to feel safe or comfortable within themselves and in the world. An unmodulated fear response radiates throughout the regulatory regime, affecting regulatory status quite broadly. The right hemisphere is responsible for acquiring this skill, and when things don't go as planned for whatever reason, that puts the brain in emergency mode and makes it work overtime to try and keep the person safe and connected. The more urgent issue is the lack of core self-regulation and its consequences, which can emerge immediately or later in life in the form of anxiety or hyper-vigilance, attachment deficits or reactivity, aggressive or paranoid behaviors, all serving the same purpose of preserving life and assuring personal safety.

With the client described above, not only had she experienced trauma as an infant when she was adopted at four months of age, but then her sense of safety and bonding was shattered yet again when her adoptive family broke up. The self-destructive, addictive behaviors can be easily explained by her lacking a sense of core self, needing external stimuli to cope with life.

During her first QIK test she found it quite challenging to stay awake, missed one target and almost missed 19 others (reaction time outliers), while also pushing the button for the non-target four times. She was slow and variable in her response times and accuracy suffered as well, as shown in Figure 5 and 6.

Results Summary:

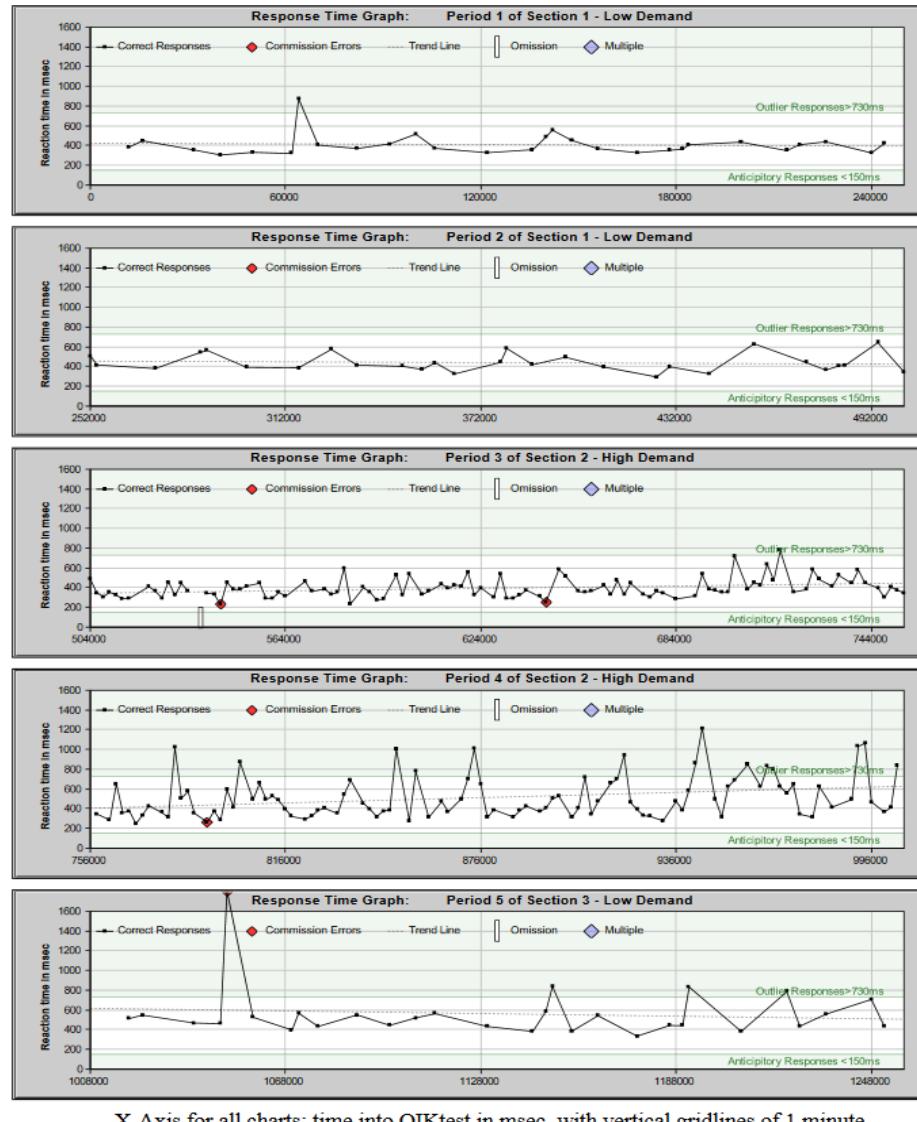


Figure 5. Results summary QIK baseline test, Case #1.

RAW DATA	Period									Total
	1	2	3	4	5	Sect. 1	Sect. 1	Sect. 2	Sect. 2	
	Low	Low	High	High	Low					
Demand Demand Demand Demand Demand										
Omissions(#)	0	0	1	0	0	0	1	0	1	
Outliers	1	0	1	14	3	1	15	3	19	
Commissions(#)	0	0	2	1	1	0	3	1	4	
Response time(ms)	393	440	394	444	482	417	417	482	423	
Variability(ms)	61	91	90	125	83	81	110	83	90	

Figure 6. Raw data, QIK baseline test, Case #1.

On more detailed analysis as shown in Figure 6 we can see which parts of the test were more challenging. She started the period 1 low demand task and did pretty well, with only one outlier and no other errors, and she was fairly fast and consistent. As we kept boring her in the second period, she remained accurate but slowed down and became significantly more variable in her responses. Entering the third period of the test with the faster pace, she started making more mistakes, missed the target once and also had two commission errors. Maintaining the high demand task was an even bigger challenge for her, as she slowed down and became very inconsistent, and also had a significant number of outliers. Just as she described during the intake, she had a hard time staying on-task, which is obvious when we look how her performance degraded as she needed to maintain a task, boring or challenging. With increased performance pressure her anxiety level increased, and thus when making mistakes in the test she sped up instead of taking her time to consider before acting. Recovery in period 5 was difficult, with three outliers and one commission error, and she was both slow and variable.

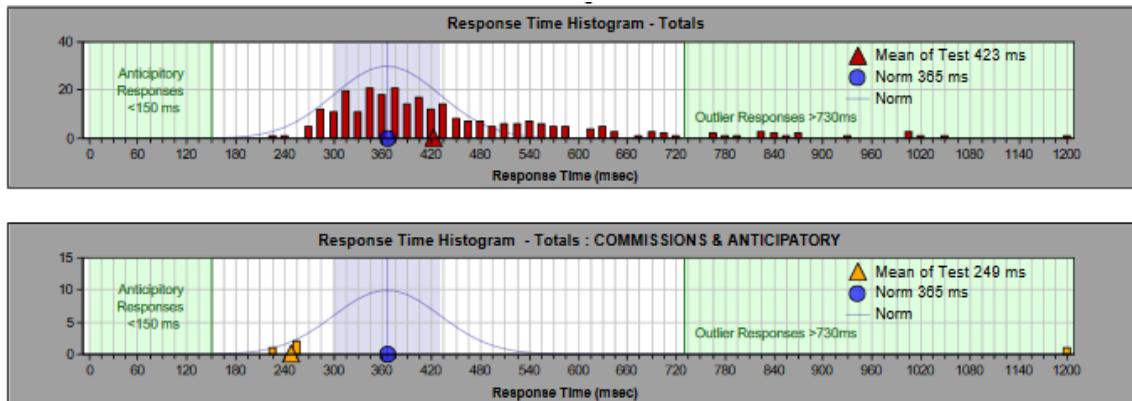


X-Axis for all charts: time into QIKtest in msec, with vertical gridlines of 1 minute

Figure 7. Time series graph of response times, QIK baseline test, Case #1.

The response time series in Figure 7 allow us to see the time course of events, where she made the mistakes, and in which part of the test her performance was better or worse. Clearly this client was able to perform fairly well when under pressure, as long as the stress was of a short duration. When the pressure continued, her performance declined, and with increased stress her nervous system tended to shut down. She almost fell asleep during the test.

Compared to a normal distribution of response times for age group and gender, the distribution of her response times in Figure 8 is much broader, with a mean reaction time of 423ms compared to the norm of 362ms. Comparing different parts of the test, it is evident that the performance decrement increases during the second high demand task, which was the most difficult for her to perform.



Norms: the blue line represents a normal distribution of reaction times for this age and group: compare by form and position to your data in red. The light blue area represents 68.2% (or $\pm 1\sigma$) of a normal distribution.

Figure 8. Response time histograms – Totals, QIK baseline test, Case #1.

She had been medicated in the past, but her sensitive nervous system didn't tolerate the different medications well, or she ended up abusing them, so eventually she just stopped taking them. The only medication still being used when we started our sessions was melatonin to help her sleep.

We started training and one by one we added all the areas in the brain we needed to train in order to target the symptoms she had described. Given the traumatic early life and the addiction history, right parietal training for calming was crucial. At the same time, several instability symptoms indicated a great need for bilateral training at mid-temporal sites to enhance stability. Later, right prefrontal placement was introduced to address the attachment issues, as well as to impact on the addictive behaviors. Finally, when her system settled down, she had fewer headaches, and her sleep had improved, we introduced the left prefrontal training to specifically target concentration, distractibility and impulse control.

The reevaluation (after 20 sessions) revealed that most symptoms had greatly improved: concentration was much better and her ability to stay on task for prolonged periods had dramatically improved. She was no longer zoning out while reading. The anxiety was gone, and her sleep had normalized. She had also stopped taking melatonin. Her sense of direction had greatly improved, but she continued to have difficulties getting to appointments on time. She rarely had any nightmares now and waking up in the morning had become easier. Her obsessive worries had moderated and she was less hyperactive. The headaches and migraines had vanished, and her PMS symptoms were less intense after the training. She felt less urge to abuse substances of any kind and had switched to electronic cigarettes to help her quit, after having already had success in reducing her smoking even before the start of training. The remaining concerns related to weight gain and being successful in life, but she felt like she was more in control of her thoughts and emotions.

Top-level scores after training are shown in Figure 9. Detailed comparison between the first and the second QIK test (Figures 6 and 10) reveals significant changes in her performance. This time there are no omissions or outliers, but interestingly she had seven commission errors, compared to just four earlier, and they are all fast, as shown in Figure 11. One variable that day was her coffee intake: she hadn't had any before the test, which was different from the previous time. Coffee acted like a stimulant for her, waking her mind up and helping her focus, so the fact that she hadn't consumed any probably influenced her performance. Speed and consistency were significantly improved from the first test.

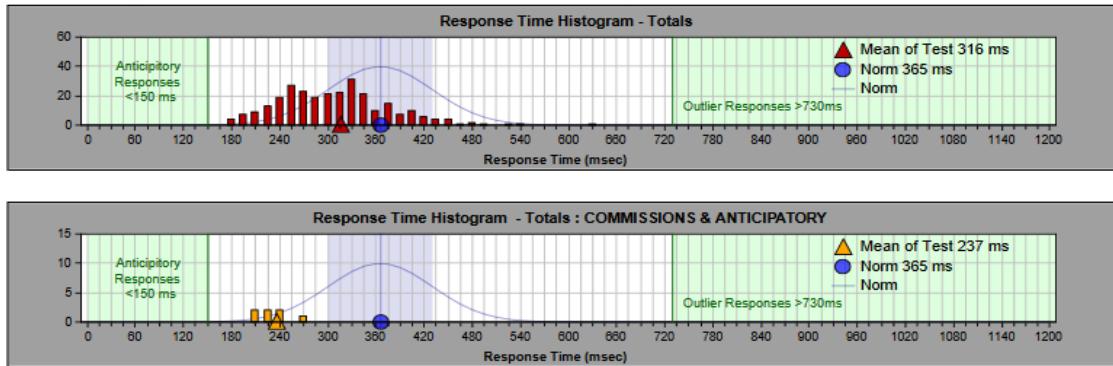
Results Summary:



Figure 9. Results summary QIK test 2, Case #1.

RAW DATA	Period					Period			Period					
	1		2		3	4		5	Sect. 1		Sect. 2		Sect. 3	Total
	Sect. 1	Sect. 1	Sect. 1	Sect. 2	Sect. 2	Sect. 3	Sect. 3	Sect. 3	Sect. 1	Sect. 2	Sect. 2	Sect. 3	Sect. 3	
	Low	Low	High	High	Low	Low	High	High	Low	Low	High	High	Low	
Omissions(#)	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Outliers	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Commissions(#)	0	0	1	5	1	0	6	1	7					
Response time(ms)	379	373	303	280	370	376	291	370	316					
Variability(ms)	68	51	64	51	55	60	59	55	58					

Figure 10. Raw data, QIK test 2, Case #1.

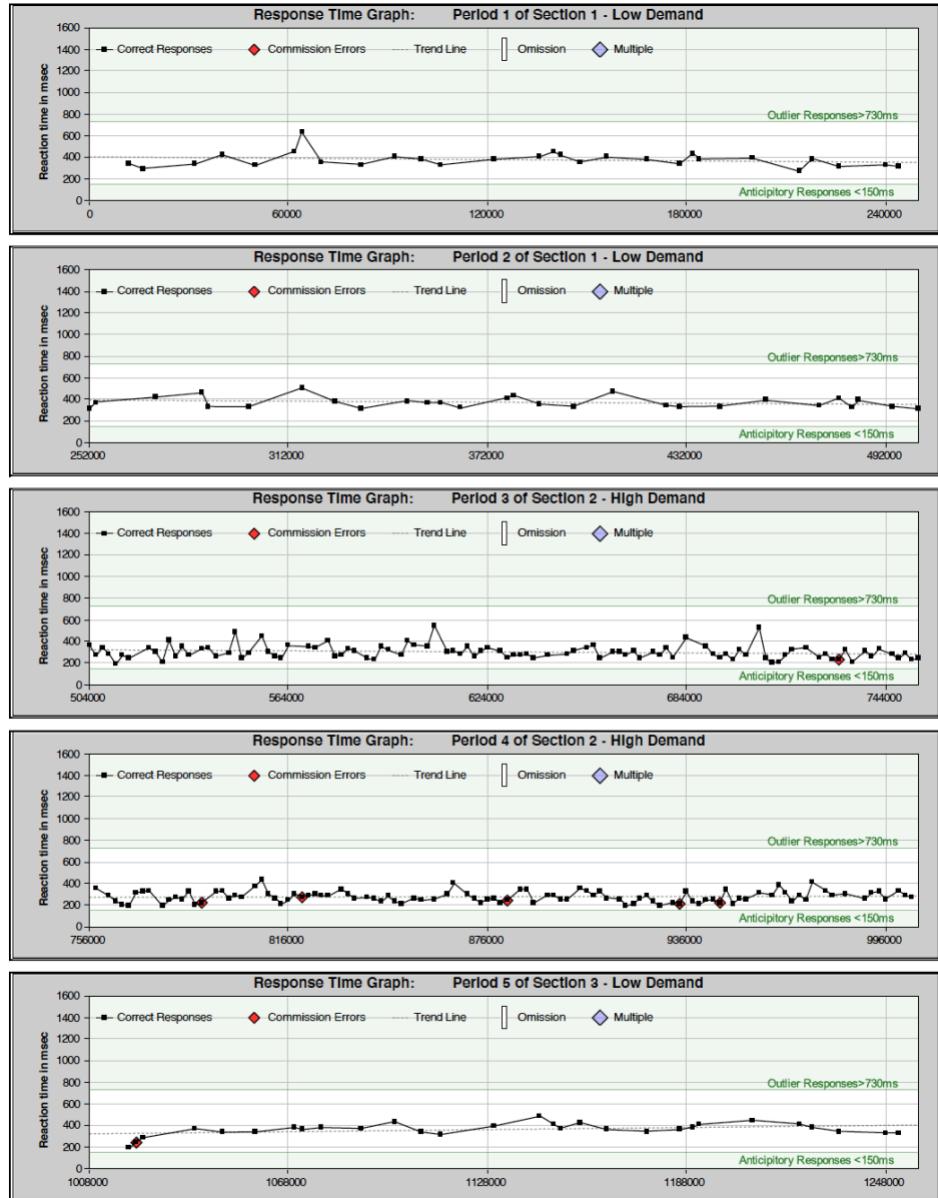


Norms: the blue line represents a normal distribution of reaction times for this age and group: compare by form and position to your data in red. The light blue area represents 68.2% (or $\pm 1\sigma$) of a normal distribution.

Figure 11. Response time histograms, QIK test 2, Case #1.

The greatest difficulty was still to perform under the pressure of the high demand task, she found, especially when maintaining that task (second period). She had similar difficulties during the first test, but this time she was both faster and more consistent during that part of the test, and that might have caused her to make more commission errors. With a mean reaction time score of more than one standard deviation above norms, she was performing at greater risk of commission errors (Figure 12). It's only during the recovery period that she speeds up instead of becoming more careful after the one

commission error she made, but overall her performance had significantly improved (Figures 13 and 14), which is consistent with the perceived changes in her symptoms. All were all reduced in severity to allow for better performance in everyday life.



X-Axis for all charts: time into QIKtest in msec, with vertical gridlines of 1 minute

Figure 12. Time series graph of response times, QIK re-test, Case #1, for comparison with Figure 7.

With such a complex case, 20 sessions is typically enough to see significant favorable change but not enough to be able to say, “we’re done training.” In fact, because of her complicated early life and the addictive behaviors, further training was recommended, and other training modalities were needed to work on resolution of learned habits.

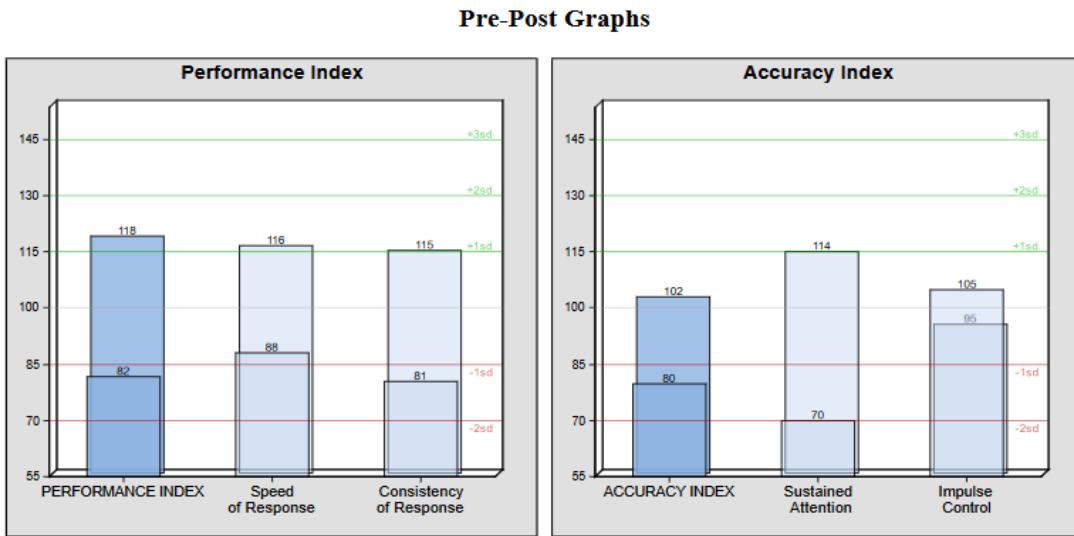


Figure 13. Pre-post graphs of standard scores for the primary measures, Case #1.

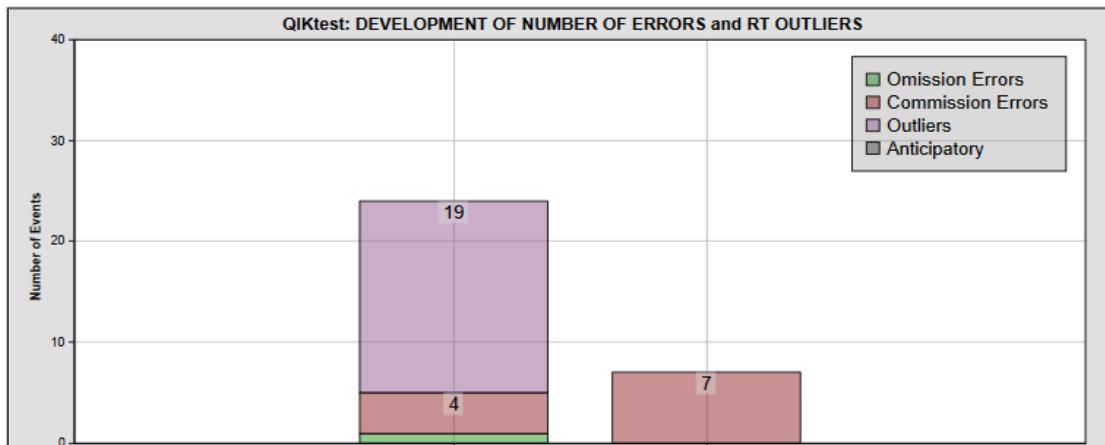


Figure 14. Development of number of errors and outliers, Case #1. Improvement was from the 9th percentile to the 55th.

6.2 Case #2

George, a 33-year-old man, sought help for his ADD symptoms when the medication he was taking created new issues for him such as rebound headaches and palpitations. In other respects, the medications had been helpful.

In his developmental history there was nothing exceptional except for his parents' divorce when he was still an infant. He was raised by his mother and stepfather. In his family history he mentioned ADHD, along with autoimmune disorders, insomnia, depression, anxiety, obesity, alcohol addiction and conduct problems. He was taking Adderall 10 mg/ day and up to 20 mg occasionally when he had to undergo some testing in school.

His main concerns before we started training were difficulties concentrating, getting on task, completing tasks and impulsivity. About once a week he would have a hard time falling back to sleep once he woke up around 2am. He would experience anxiety as tension in his body and obsessive worries. He sometimes had neck tension and ground his teeth. He would overeat with stress and was sensitive to sugar – he would have a sugar-fueled high and then crash later. He had frequent headaches when not drinking coffee or not taking Adderall.

His only QIK test was taken before we started training him (Figure 15), and he complained that it had been difficult to perform because he was getting distracted by the ticking of the clock on the wall. He missed the target three times during the test, twice in the recovery part of the test, period 5, and once during the high demand task, which is consistent with what we already knew about his difficulties performing under pressure and staying on task for long periods of time (Figure 16). His overall performance and accuracy were average; he hadn't taken Adderall the day of the test and didn't take it for the most part while doing the sessions in the clinic.

Results Summary:

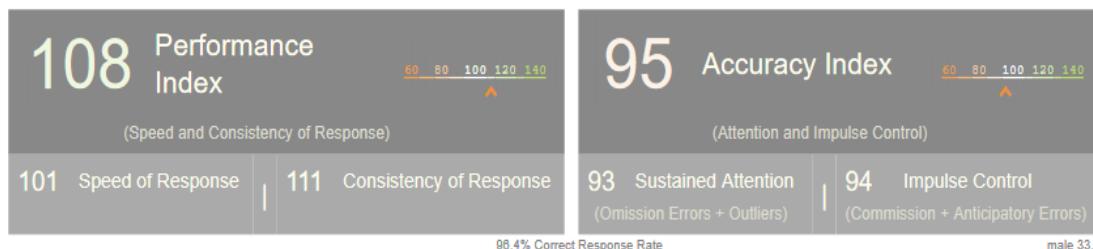


Figure 15. Results summary QIK baseline test, Case #2.

RAW DATA	Period					Period			Period			Period			Period			Total
	1		2		3		4		5		Sect. 1		Sect. 2		Sect 3			
	Sect. 1	Low Demand	Sect. 1	Low Demand	Sect. 2	High Demand	Sect. 2	High Demand	Sect. 3	Low Demand	Sect. 1	Sect. 2	Sect 3					
Omissions(#)	0	0	1	0	2	0	1	2	3									
Outliers	0	0	0	0	0	0	0	0	0								0	
Commissions(#)	0	0	4	3	0	0	7	0	7								7	
Response time(ms)	398	389	322	349	393	393	335	393	353									
Variability(ms)	37	49	82	65	63	44	75	63	59									

Fig.10.15

Figure 16. Raw data, QIK baseline test, Case #2.

He started noticing positive changes in his distractibility early on with the training and was able to track results as he was studying for exams. It took a while to find the optimal protocol for him; he had a very sensitive nervous system, and because of the Adderall and caffeine variables it was at times challenging to figure out what each was contributing to the reported shifts. He clearly benefited from the sessions and would report improvement in concentration and the ability to deal with stress and deadlines he had to meet, but usually the results faded a day or two after sessions. He didn't finish his 20 treatments and we didn't get to take a second QIK test, so there is no measurable data to gauge brain performance. Given the inability to hold the gains, it was clear that a lot more training would have been needed before his brain would successfully stay on track and perform optimally on its own. One hypothesis for the failure to hold gains is that the infant had in fact been traumatized by the parents' divorce, and that the resulting

impact on nervous system functioning had not yet fully resolved. This case also illustrates what can frequently happen as the client comes to terms with this novel method. An initial healthy skepticism may well transition to its opposite, heightened expectations, as the first good effects are felt. When the training procedure then fails to live up to those new expectations, the effort is abandoned.

6.3 Case #3

Aidan, a 16-year-old young man with a dual diagnosis of ADHD and dyslexia, received intensive neurofeedback training at our clinic. Over a two-week span he received 20 neurofeedback sessions, at a rate of two sessions a day, and he continued with home training.

The main concerns described during the intake with us were hyperactivity and distractibility, anxiety as worries, some frustration and compulsive organization, as well as difficulty with academic classes. Words would move on the page when trying to read. This problem was helped considerably with Irlen lenses. He had headaches with reading or when dehydrated and had some difficulties falling asleep. In the past he had been sleep-walking and had night terrors as well. Sugar sensitivity was described also.

He was born through emergency C-section with his umbilical cord around his neck and was described as a stressed baby. He walked early and talked late and wasn't much of a talker even later on in life. He was accident-prone and had a few falls and stitches growing up and even had a finger reattached at 11 months. Around the time he was four years old his parents separated for a year. In his genetic history insomnia, postpartum depression, anxiety, OCD, dyslexia and Asperger's were present.

Prior to the neurofeedback he had been on 54 mg of Concerta®/day, which he stopped taking while undergoing neurofeedback. His pre-training QIK test (Figures 17 and 18) revealed an average performance index and an accuracy index well below average, with impulsivity scores in the 1st percentile, with a high number of commission errors and a significant number of omissions. The part that he found to be the most difficult was the high demand section, where his performance dropped significantly. After the first two sessions he reported falling asleep faster, and one session later noticed improvement in reading comprehension, although at the time the protocol wasn't yet focusing on reading issues. His mother also noticed him becoming less hyperactive, even though he was off his medication for the duration of the neurofeedback training. By session 10 behavioral issues had subsided to where they would have been when he was on medication. Reading continued to improve, and he actually started reading more with---or even without---his Irlen lenses.

QIK test #1

Results Summary:



Figure 17. Results summary QIK baseline test, Case #3.

RAW DATA	Period	Period	Period	Period	Period				Total				
	1	2	3	4	5	Sect. 1	Sect. 1	Sect. 2	Sect. 2	Sect. 3	Sect. 1	Sect. 2	Sect 3
	Low	Low	High	High	Low	Low	High	High	Low	Low	Low	High	Low
	Demand	Demand	Demand	Demand	Demand	Demand	Demand	Demand	Demand	Demand	Demand	Demand	Demand
Omissions(#)	0	1	4	5	3	1	9	3	13				
Outliers	0	0	0	1	1	0	1	1	2				
Commissions(#)	2	0	18	14	2	2	32	2	36				
Response time(ms)	398	398	280	326	429	398	303	429	335				
Variability(ms)	81	48	91	111	124	67	104	124	91				

Figure 18. Raw data, QIK baseline test, Case #3.

During reassessment the progress made was reported: he had better understanding while reading and was more able to visualize what he read. He described more immediate and detailed imagery and was less fidgety and distracted. His sleep had gotten better with fewer nightmares and less anxiety or obsessive worries. He also noticed being more comfortable when having to deal with traffic.

QIK test #2

Results Summary:



Figure 19. Results summary QIK test 2, Case #3.

RAW DATA	Period	Period	Period	Period	Period				Total				
	1	2	3	4	5	Sect. 1	Sect. 1	Sect. 2	Sect. 2	Sect. 3	Sect. 1	Sect. 2	Sect 3
	Low	Low	High	High	Low	Low	High	High	Low	Low	Low	High	Low
	Demand	Demand	Demand	Demand	Demand	Demand	Demand	Demand	Demand	Demand	Demand	Demand	Demand
Omissions(#)	0	0	0	0	0	0	0	0	0	0	0	0	0
Outliers	0	0	3	0	0	0	0	3	0	0	0	0	3
Commissions(#)	0	0	5	4	0	0	0	9	0	0	0	0	9
Response time(ms)	402	380	337	366	431	391	352	431	368				
Variability(ms)	82	55	84	97	54	71	92	54	74				

Figure 20. Raw data, QIK test 2, Case #3.

The second QIK test (Figures 19 and 20) showed some net improvement in the performance index. His speed of response decreased during the high-demand periods, which allowed for higher consistency of response as well as improved accuracy. In fact, accuracy went from a standard score of 72 to 103, from

the 3rd percentile to the 58th. This kind of spectacular improvement becomes even more relevant in the prevailing context, since he had stopped taking his medication before undergoing training in the clinic.

It was decided that it would be appropriate for him to continue training at home, and he received 4-5 sessions a week while continuing to stay off the medication. A month into home training his mother reported further improvement in concentration and in his ability to make good choices, and he was better able to manage usual day-to-day events.

6.4 Case #4

Michael, an 11-year-old boy, was having a hard time in school. He didn't have a formal diagnosis but exhibited some of the classic symptoms of ADHD. He had a short attention span, was easily distracted, impulsive and disorganized and couldn't sit still in school. Among the presenting symptoms there were some learning difficulties, like understanding math concepts and calculation and writing problems. He was described as being clumsy. He was inflexible and defiant mostly in a school setting; frustration and anger were issues as well. Occasional headaches and stomachaches as well as teeth-grinding and sugar cravings completed the picture. After being adopted at birth, his early life was unremarkable, except for some chronic ear infections that required tubes at age one until age two. As a result, he was sensitive to sound, especially to loud noise.

Taking the QIK test (Figures 21 and 22) the first time proved to be quite a challenge for Michael. He scored below average for speed, consistency and inattention with only one score, impulsivity, within the normal range. This indicates that he was slow and somewhat variable, and unable to stay on task. The response time histogram (Figure 23) reveals a broad distribution of response times with lots of outliers.

Results Summary:

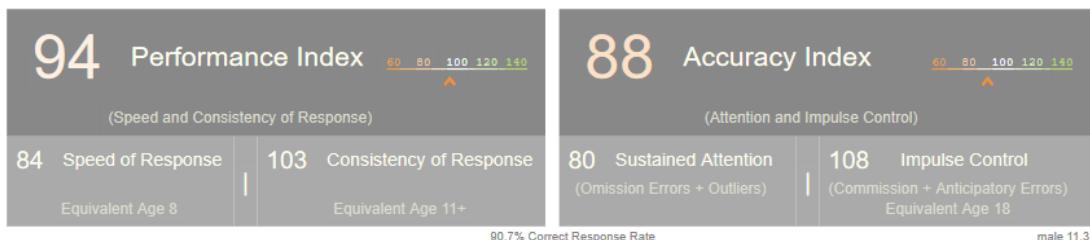
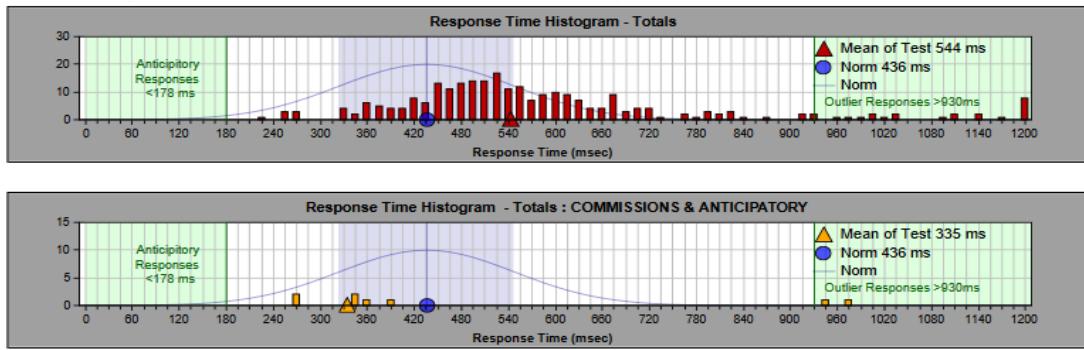


Figure 21. Results summary QIK baseline test, Case #4.

Results	Data	Norm	Score	
M11.3 Total Test	Measured Value	Median of Distribution	Standard Score	Percentile
Omission Errors	18 errors	5 ₀ errors	85	17 %
Outlier Responses	24 errors	4 ₄ errors	77	6 %
Commission Errors	8 errors	12 ₉ errors	108	70 ₅ %
Response Time	543 ₅ ms	436 ms	84	14 %
Variability	113 ₁ ms	112 ms	103	56 %

Figure 22. Results summary data, QIK baseline test, Case #4.



Norms: the blue line represents a normal distribution of reaction times for this age and group: compare by form and position to your data in red. The light blue area represents 68.2% (or $\pm 1\sigma$) of a normal distribution.

Figure 23. Response time histograms, QIK baseline test, Case #4.

His training protocol targeted areas in the brain to promote physical, emotional, and mental calming as well as stabilization. T3-P3 was added for the learning difficulties. Over a five month span he completed 20 sessions of neurofeedback. His statement at the end: "I'm not stupid anymore," conveys his own sense of the progress he had made in just 20 sessions. The child was thrilled about his new way of relating to his peers. This sheds some light on how difficult it can be for people with these symptoms to fit in, how much harder they feel they need to work to keep the pace in school or at work, and how much their disregulated nervous system can hinder function. During his re-evaluation his mother reported improvement in most initial symptoms, and his second QIK test supported that with measurable data.

He was enjoying school and was more optimistic, now that his attention and impulse control had significantly improved. In place of his earlier defiance, he was less frustrated and angry, and much more flexible and cooperative. He was much more organized and improved his writing and math skills. He didn't have any headaches or stomachaches and wasn't grinding his teeth anymore. He was less clumsy and was now able to sit still in school, so he wasn't distracting others as he had been before.

His first test reflected the above described difficulties mostly in attention and to a certain degree in impulse control. Twenty sessions later, a second test showed significantly improved overall scores, with a superb leap towards the upper limit of normal accuracy index scores (Figures 24 and 25). Both sustained attention and impulse control were much better, and the performance index greatly improved as well.

Results Summary:



Figure 24. Results summary QIK test 2, Case #4.

Results	Data	Norm	Score	
M11.7 Total Test	Measured Value	Median of Distribution	Standard Score	Percentile
Omission Errors	0 errors	5.0 errors	>122	max
Outlier Responses	1 error	4.4 errors	115	84 %
Commission Errors	4 errors	12.9 errors	116	86.9 %
Response Time	457.9 ms	436 ms	96	40 %
Variability	108.4 ms	112 ms	106	66 %

Figure 25. Results summary data, QIK test 2, Case #4.

In light of these impressive gains, we suggested retesting Michael after three months to see if the results were holding. This is not always assured after training only for 20 sessions. Although the impulse control continued to improve, the performance index dropped back to the level measured prior to the neurofeedback. The need for additional sessions is indicated by these results (Figures 26 and 27). Since the brain showed itself capable of operating at the higher performance level, it should be able to do so again. Other factors that could explain the failure to hold gains should also be looked for. Figures 28 and 29 show the trends of errors and outlier responses over the three data sets.

Results Summary:



Figure 26. Results summary QIK test 3, Case #4.

Results	Data	Norm	Score	
M11.9 Total Test	Measured Value	Median of Distribution	Standard Score	Percentile
Omission Errors	4 errors	5.0 errors	102	57 %
Outlier Responses	9 errors	4.4 errors	90	26 %
Commission Errors	2 errors	12.9 errors	125	95.5 %
Response Time	549.6 ms	436 ms	83	13 %
Variability	113.3 ms	112 ms	100	51 %

Figure 27. Results summary data, QIK test 3, Case #4.

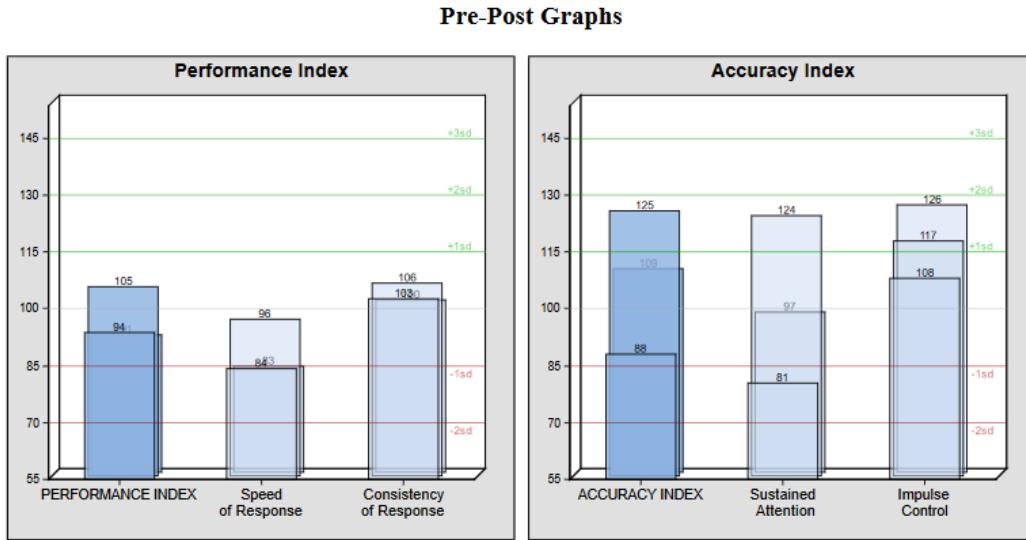


Figure 28. Pre-post graphs, Case #4

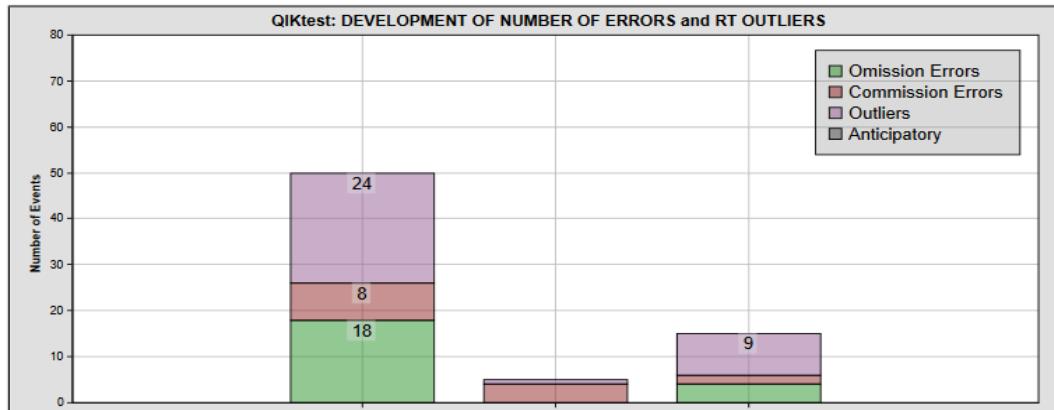


Figure 29. Development of number of errors and outliers, Case #4. The score improved from the 21st percentile to the 95th, and was still above norms at 73rd percentile at the later re-test.

6.5 Case #5

Nicky, an 8-year-old boy, had been diagnosed three years prior to coming to see us for ADHD symptoms. His mother described him as a very smart child who was highly impulsive and hyperactive, had poor self-control and a short attention span. He had difficulties organizing, was distractible and forgetful, and was impatient and easily frustrated. He was always rushing through tasks which led to making mistakes and had some difficulties with math and spelling. He always wanted to do as little as possible to get by and had poor self-confidence. He was playing the class clown in order to feel accepted and would manipulate anyone to get his way by lying and cheating, about which he never exhibited remorse. He was fearless, selfish and careless, and never personally at fault. Most recently he had gotten in trouble in school for aggressive behaviors. He was also biting his nails, mostly when under pressure. He had stomachaches with constipation, and sugar cravings were an issue as well. Bedwetting had been a problem in the past but had stopped a few months back.

Noteworthy is the fact that the birth process had been a breach presentation that required an emergency C-section, and Nicky was born with the umbilical cord around his neck. All developmental milestones were reached on time. His mother described herself as a perfectionist and would push him just as hard as she pushed herself. In consequence, Michael blamed her for wanting him to be perfect.

The genetic history revealed addiction problems, thyroid disorders and bipolar disorder, as well as ADHD. When we started training, he was on 10 mg of Adderall a day, which had been doubled three months prior to the start of neurofeedback treatments, due to lack of improvement in symptoms. Despite the increase in the medication, his symptoms were not controlled, and his parents were concerned he would have to take more and more of it until, eventually, it wouldn't work for him at all.

During the first QIK test (Figure 30) Nicky had a difficult time staying on task and had to be prompted several times to continue, as he was becoming increasingly restless and bored. At the end he was able to report on the number of mistakes he had made. The test report revealed all scores within the normal range, with high scores in sustained attention and consistency of response times, while the speed of response was normal. Whereas his impulse control score was high, he clearly struggled with stopping himself from impulsively pressing the button for the non-target.

Results Summary:



Figure 30. Results summary QIK baseline test, Case #5

In designing his training protocol, we considered basic placements to target most of the described symptoms: stabilization for the sugar cravings; physical calming for anxiety, hyperactivity, self-awareness, constipation; emotional control for self-confidence, frustration, anger and aggressive and manipulative behaviors as well as social-emotional awareness; mental calming to address impulse control, organization, attention and forgetfulness and also attention to details to help with math and spelling. We did a total of 21 neurofeedback sessions in the clinic and after the reevaluation they continued with home training for another two months.

He responded quickly to the training and subtle changes in symptoms were noticed early on. By session 4 he was mellower, less easily frustrated and not getting in trouble as much in school, something that the teacher had commented on. He was less impatient and less forgetful, not rushing through tasks all the time. He also didn't need to be told to stop misbehaving that often. He even was able to report feeling calmer and felt good about not getting in trouble in school much anymore.

Nicky was still taking his medication on school days, but his parents decided to try to stop it on the weekends to observe his behavior without it. While before doing neurofeedback going off the medication on the weekends was impossible due to his being really 'out of control', after just six sessions he did pretty well with the drug holiday. Soon his math improved, and he became more thoughtful and calmer,

while remaining well-behaved without medication during the weekends. During his treatments we adjusted the protocol as needed, according to his response to the training, and took on more areas to work on as we continued to calm and stabilize his nervous system.

The reevaluation revealed significantly improved performance with the QIK test (Figure 31) and, according to his parents, the following changes in symptoms: he was less hyperactive, less impulsive, and less easily frustrated. He hadn't displayed any aggressive behaviors in weeks, was biting nails less, and his mathematics performance had improved considerably. He had been off the medication during the weekends and was observed to maintain good behavior without it. He was doing much better in school both with performance and behavior - a change that his parents and his teachers had noticed. Several symptoms had not changed significantly: he was still rushing through tasks and was still using manipulative behaviors to get his way. Spelling was still problematic, and he needed repetitions and prompts to follow instructions. These concerns, along with the goal to help lower his medications while supporting brain function with neurofeedback, were the reasons we recommended home training.

Results Summary:



Figure 31. Results summary QIK test 2, Case #5

All the scores in the second test were significantly better than the scores in his first test, and interestingly he didn't have any anticipatory responses or outliers in the retest. This reflects a readiness of his nervous system to attend to the task at hand and respond appropriately. Also noteworthy is the fact that although his performance during the first test scored within the normal range, he improved it during the second test in all tested areas (Figure 31). Summary data are shown in Figures 32 and 33.

The home training allows for longer, more frequent sessions, utilizing the protocols that were established in the clinic. By reinforcing the training further on an almost daily basis, the expectations are to see further significant gains and help the brain to hold those gains. They did about four sessions a week in the two months following the treatments in the clinic before returning the system, to a total of 32 sessions in addition to the 21 we had completed here. While doing the home training Nicky was only taking half of his 10 mg of Adderall and he continued to improve. His teacher was impressed with his good behavior, and he even got an award for his creative writing. The teacher pointed out that his spelling had improved, and that he really had taken a lot of time and put a lot of effort into this project, something he would have absolutely not been able to do before neurofeedback. He continued to benefit from the sessions at home, and when they returned the system two months later, he was a different child, according to his mother. During the summertime, while on vacation, he went off the medication and continued to do well without it.

Pre-Post Graphs

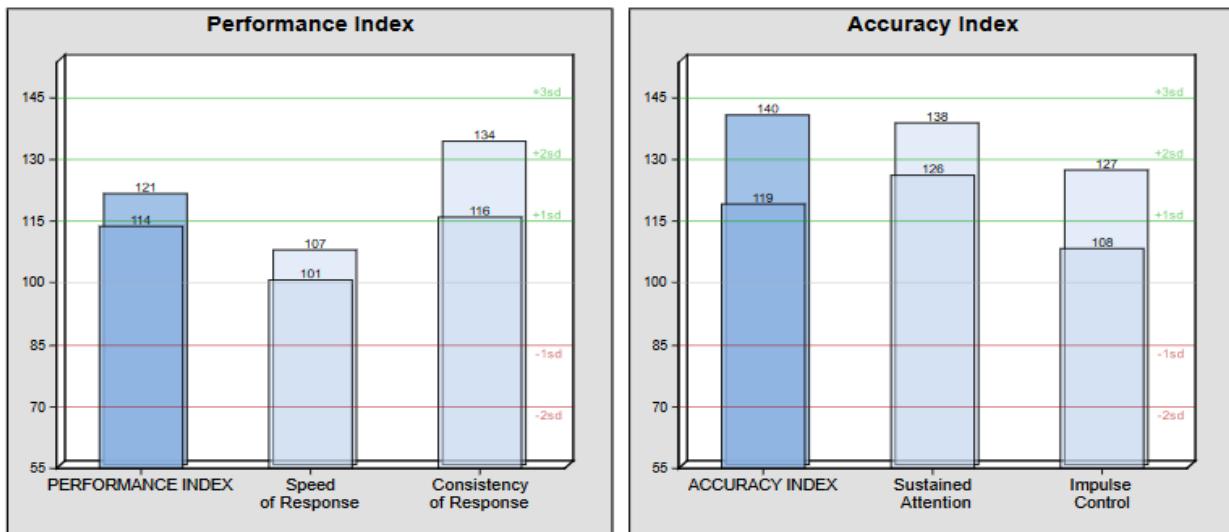


Figure 32. Pre-post graphs, Case #5

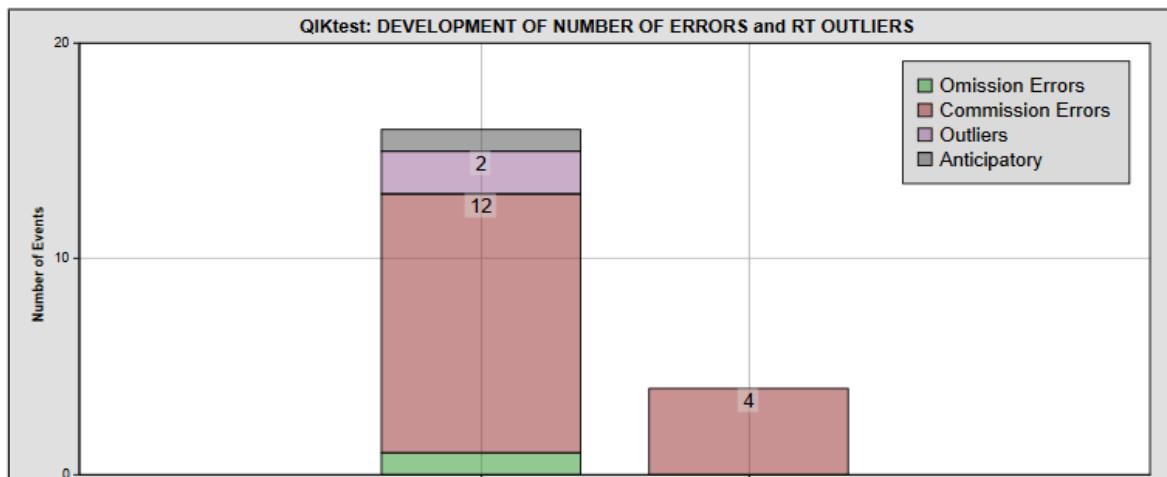


Figure 33. Development of number of errors and outliers, Case #5. The accuracy score improved from the 91st to the 99th percentile.

With all the clinical cases described above, as well as with all the other clients we continue to help with neurofeedback, the particularity and specificity of our method, as well as the individuality of the response for each person we train, become ever more obvious with the progressive refinement of methods. It is within the brain's scope to enhance its own functional capacity if it is merely given information on its own behavior, to which it is normally blind. By facilitating this process, we allow enhanced self-regulation to emerge and to consolidate. Beyond the diagnostic label, what needs to be fully understood is the uniqueness of each case and the many variables that come into play in the guidance of this process.

7.0 Summary and Conclusion

This chapter reflects the evolution in the understanding of the ADHD spectrum that needs to occur. First of all, the behavioral features of ADHD are here embedded in a more comprehensive dysregulation model that draws attention to commonalities rather than distinctions. The responsiveness of the entire repertoire of behavioral sequelae of ADHD to a simple training technique points to a modest set of underlying failure mechanisms. The fact that this technique relies almost entirely on information derived from the extreme infra-low frequency domain implies that we are engaged with the tonic regulation of our intrinsic connectivity networks. The fact that this training occurs while the brain is being minimally challenged argues further that our primary concern is with the internal organization, the functional connectivity, of the task-negative network, the Default Mode. A secondary concern is with the interaction of the task-negative with the task-positive control networks.

The review of the case reports illustrates the individuality with which matters need to be approached clinically, even within the overall homogeneity and commonality of approach. We may be seeing here one of the early exemplars of the coming age of personalized medicine and of functional medicine. In the application of infra-low frequency training, the adaptation of the protocols to each case is obligatory, which justifies reliance on case series to establish clinical effectiveness. Additionally, the statistical data presented at the outset document the breadth of impact of this method on attentional deficits across the clinical spectrum.

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