LORETA Neurofeedback in College Students with ADHD

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Attention deficit hyperactivity disorder (ADHD) is a psychiatric disorder characterized by behavioral symptoms in three main areas—hyperactivity, impulsivity, and inattention. Per the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-V), there are three subtypes of ADHD—predominately hyperactive/impulsive, predominately inattentive, and combined type—each of which is classified based on a number of behavioral symptoms (APA, 2013). One of the key factors in diagnosing and understanding ADHD is that the symptoms are pervasive across multiple settings. Additionally, the comorbidity between ADHD and other psychiatric disorders is high, with learning disabilities, depression, and anxiety frequently codiagnosed conditions (Decker, McIntosh, Kelly, Nicholls, & Dean, 2001). ADHD is also a neurodevelopmental disorder, which suggests that while the symptoms change as an individual develops, the disorder continues to affect those individuals across their lifespan (Das, Cherbuin, Easteal, & Anstey, 2014; Fredriksen et al., 2014).

Symptomatology in ADHD is multifactorial; thus, ADHD is a heterogeneous disability. Some research has suggested that the subtypes currently used do not fully encompass the spectrum of the disorder (i.e., Barkley, 2001, 2003; Milich, Balentine, & Lyman, 2001). Diagnosis is still highly dependent on behavioral observations and testing. In part, symptomatology is related to normative standards of development. For instance, being "driven as a motor" and difficulty inhibiting behavior is typical for children at a certain developmental level. However, when these difficulties

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continue into late childhood and early adolescence, they become problematic. Additionally, inattentive behaviors often go unnoticed, particularly in younger children when cognitive demands are kept low. Furthermore, the presentation of ADHD can change across development. Regardless of the exact presentation of the disorder, these difficulties with classification have resulted in significant heterogeneity of how individuals are identified as having ADHD. Consequently, research studies that use participants with a previous diagnosis of ADHD likely involve significant confounds involving the origins of symptomatology. As an inevitable consequence, treatment plans for many individuals with ADHD will be ineffective because most treatment options are based on a categorical diagnosis of behavioral symptomatology without specification of individualistic factors or determination of the underlying causes.

Developmentally, ADHD symptoms are present from preschool to adulthood (Guldberg-Kjar & Johansson, 2013; Scholtens, Rydell, & Yang-Wallentin, 2013; Washbrook, Propper, & Sayal, 2013). However, symptom manifestation may differ across developmental ages. For instance, hyperactivity may be the primary symptom in childhood; whereas working memory (WM) deficits may be the primary symptoms in adulthood (Kim, Liu, Glizer, Tannock, & Woltering, 2013). Children with ADHD are often distinguished by an inability to inhibit their behavior despite an awareness of appropriate behavioral standards (Nigg, 2013). Disruptive behavior, which is typically the primary reason for referral, may be paramount despite having an understanding of the appropriate behaviors within a given context. Although they may be able to verbally explain what they "should" do, they may still be unable to actually complete the motor act. Although children with ADHD do not have significant deficits in language, with possible exceptions in language pragmatics (Green, Johnson, & Bretherton, 2014), they may have significant difficulty in using language to regulate their own behavior (Petersen et al., 2013).

CHALLENGES IN TREATING ADHD IN COLLEGE-AGED POPULATIONS

The transition to early adulthood is often tumultuous for individuals with ADHD. In part, life outcomes depend on the environmental and contextual demands for the person. Individuals in fairly structured and repetitive occupations with low cognitive demand may have little interference from ADHD symptoms, once habits are established. However, individuals attempting to enter new environments with new or complex cognitive demands—a common experience in early adulthood—may experience significant distress and difficulty adapting.

The college years are a particularly vulnerable time for young adults with ADHD. Inherently, matriculating to a university is a highly unstructured task. Routines and habits cultivated from adapting to local high school conditions, in which many students had support from teachers and parents, as well as legal protection from an Individualized Educational Program (IEP), are no longer available. Although hyperactive symptoms may persist, WM and executive function (EF) deficits are more prominent (Barkley, 2010). Indeed, WM and EF deficits have been hallmark symptoms of individuals with ADHD (Barkley, 2006; Das et al., 2014), although these symptoms are also prominent in other disability conditions (Englund, Decker, Allen, & Roberts, 2014; Kofler, Rapport, Bolden, Sarver, & Raiker, 2010; Raiker, Rapport, Kofler, & Sarver, 2012). Such deficits become a struggle as complex course content places high demands on cognitive ability. These increased demands on WM and EF are often overwhelming and insurmountable for college students with ADHD, often leading to destructive emotional states. Misuse of prescription, and recreational drugs are a predictable consequence (Culpepper, 2013). Success or lack thereof, in adapting to the demands of higher education will have a tremendous impact on the life trajectories of individuals with ADHD.

Theoretical Explanations

Theoretical approaches are important for understanding and treating the core deficits of the disorder. There are various theoretical approaches to understanding ADHD and different approaches have different consequences. For example, school-based services view ADHD as a medical condition and require identification as Other Health Impaired, similar to other medical disabilities. Unfortunately, this approach places undue emphasis on medical treatments (i.e., medication) despite the fact that ADHD is no more "medical" than other behavioral disabilities (Wodrich & Schmitt, 2006). Historically, minimal brain dysfunction (MBD) was a precursor classification to ADHD as the symptoms of ADHD are similar to individuals with mild brain injuries. However, subsequent research found that many children with MBD did not have any documented evidence of brain injury, which provided the impetus to find the underlying causes of the disability. Hyperkinetic Reaction of Childhood in the DSM-II (1968), and Attention Deficit Disorder with the specifier "with or without hyperactivity" in the DSM-III (1980) were subsequent classifications that have also been discarded. The therapeutic use of stimulant medication, discovered by chance rather than scientific investigation (Baumeister, Henderson, Pow, & Advokat, 2012), is attributed to increases in dopamine in the frontal regions of the brain (del Campo et al., 2013). Because

increases in dopamine alleviate symptoms, "neurochemical" theories have postulated ADHD is caused by a deficiency in neurotransmitters. However, just as migraines are not caused by a deficiency of aspirin in the brain, drugs that increase dopamine may be therapeutic but not necessarily causal of ADHD. Other conceptual approaches range from parenting to food additives (Barkley, 2006).

Without discounting the significant heterogeneity in ADHD causes, contemporary evidence has supported a link between ADHD symptoms and disruptions in specific brain networks, mainly in the prefrontal and subcortical areas of the brain (Silvetti, Wiersema, Sonuga-Barke, & Verguts, 2013; Volkow et al., 2007). Neuropsychological theories (see Sergeant, Geurts, Huijbregts, Scheres, & Oosterlaan, 2003 for a comparison of different models) have emphasized response inhibition and WM deficits as core constructs in explaining ADHD (Barkley, 1997, 2003). Although contemporary models continue to support disruptions in specific brain networks, there has been a shift in the neuropsychological constructs. Indeed, self-regulation (Barkley, 2013) and cognitive control (Nigg, 2010) have increased in importance as core constructs to integrate and unify various symptoms characteristic of ADHD. In contrast to traditional models, these more contemporary models recognize the role of internalized mental processes that regulate goal-oriented behavior. More specifically, self-regulatory process are viewed not as the cognitive functions involved with goal pursuit and obtainment (e.g., goal state representations), but rather as the cognitive processes influencing other cognitive processes that are more directly involved in specific behaviors. Although somewhat undefined, deficits in this area could include self-awareness, inhibition, WM, motivation, and/or innovation (Barkley, 2013).

Self-regulation may provide an important mediating construct to explain treatment outcomes in ADHD. Both behavioral (Pelham & Fabiano, 2008) and psychopharmacological interventions (i.e., stimulant medication) (Bitter, Angyalosi, & Czobor, 2012; Castells et al., 2011; Faraone & Biederman, 2002; Faraone, Spencer, Aleardi, Pagano, & Biederman, 2004) have demonstrated effectiveness in treating ADHD. However, the effect sizes for most of these interventions are modest, few generalize to an ecological context (Van der Oord, Prins, Oosterlaan, & Emmelkamp, 2008), and it is unpredictable why some individuals with ADHD respond to some treatments and not others. Such inconsistency may be a result of unknown, or uncontrolled, mediating variables. Self-regulation is a viable mediating construct that may account for inconsistencies across studies. That is, diverse ADHD interventions work sporadically because each, implicitly, facilitates or sets the occasion for the individual to initiate self-regulation processes, which likely occurs in some individuals and not others. Tasks and situations that require the flexible shifting of attention toward the regulation of internal mental processes

enhance cognitive control, which promote response inhibition and enhance WM. Unfortunately, these interventions indirectly train self-regulation, rather than directly, and result in unpredictable outcomes.

ADHD, Self-Regulation, and Neurofeedback

Neurofeedback may be useful to explicitly treat self-regulation deficits in ADHD. The effectiveness of quantitative electroencephalography (qEEG) in both assessment and treatment of ADHD is well supported (Chabot et al., 1996). Meta-analytical research has concluded qEEG measures are capable of differentiating individuals with ADHD from individuals without ADHD (Arns, Conners, & Kraemer, 2013; Chabot, Merkin, Wood, Davenport, & Serfontein, 1996; Snyder & Hall, 2006). Indeed, the American Academy of Pediatrics, which has historically solely supported behavioral and psychopharmacological interventions for ADHD, has recently added EEG biofeedback as a Level 1 Best Supported intervention for ADHD (American Academy of Pediatrics, 2012). Electroencephalographic methods also have the added benefit of being less expensive and more accessible than other neuroimaging techniques.

Like other ADHD treatments there are inconsistencies in treatment outcomes using NF. For example, some studies have found NF to be superior to stimulant medication (Meisel, Servera, Garcia-Banda, Cardo, & Moreno, 2013) and other studies have found NF to be less effective (Ogrim & Hestad, 2013). Like other treatments, NF may provide an opportunity for an individual to engage in self-regulation. Progress monitoring provides self-relevant feedback that rewards self-referential thought processes and increases self-efficacy in cognitive control. As demonstrated by recent studies, control beliefs, defined as the individual's expectancy for contingent result of an action, influences NF performance (Witte, Kober, Ninaus, Neuper, & Wood, 2013), as do implicit learning strategies used during NF (Kober, Witte, Ninaus, Neuper, & Wood, 2013). Furthermore, as indicated in an fMRI NF study, effortful attempts to self-modulate brain activity, in both real and sham conditions, bilateral insula, anterior cingulate, and prefrontal regions of the brain became activated (Ninaus et al., 2013), all of which are brain areas involved in attention. Interestingly, there is a high degree of overlap between brain networks involved in attention and self-regulation, which will be discussed next.

Brain Networks Involved in Self-Regulation

In a meta-analysis of 27 MRI studies that incorporated self-referential tasks, three broad brain regions were identified as consistently activated during self-oriented tasks using both factor analysis and then reanalyzed

using cluster analysis (Northoff et al., 2006). These regions include the Ventral Medial Prefrontal, Dorsal Medial Prefrontal, and Precuneus areas of the brain. Together these regions are referred to as the midline cortical structures (MCS) and appear to be a network involved with self-referential cognitive processes (D'Argembeau et al., 2008; Holt et al., 2011; Kim, 2012; Kjaer, Nowak, & Lou, 2002; Northoff & Bermpohl, 2004).

Notably, there is substantial overlap between MCS and brain areas involved with other networks such as the default mode network (Otti et al., 2012; Travis et al., 2010) and attentional control networks (Zhang & Li, 2012). Indeed, a recent study implicated symptoms of ADHD as involving brain areas that are in common to the MCS, default, and attention networks (Posner, Park, & Wang, 2014). Despite the overlap in brain networks, these areas are typically described as attention networks in ADHD research although self-regulation processes, or the lack thereof, could be an alternative viable hypothesis. For example, the anterior cingulate (AC) is repeatedly implicated in both attention and self-regulation studies (Rothbart, Sheese, Rueda, & Posner, 2011). Functionally, the AC has been described as providing a cost-benefits analysis of effortful cognition as mediated by dopamine (Kurniawan, Guitart-Masip, & Dolan, 2011), and is critical for translating intention to motor action (Paus, 2001). Presumably, effortful cognition is metabolically expensive and engaged when there is a mismatch between expected outcomes or rewards and actual outcomes, which is indicated with dopamine signaling.

Increasing activation in brain networks involved in self-referential cognitive processes is thought to increase attentional flexibility, which allows for better allocation of attentional resources across different networks. Thus, the engagement of "self" brain networks may be a critical prerequisite for positive therapeutic outcomes in individuals with ADHD. Neurofeedback may directly amplify self-referential cognitive processes but also directly target the brain networks involved with self-referential thinking. In turn, these brain networks may provide the basis for cognitive control, which in turn leads to changes in brain activity. Mechanistically, the general variables involved with self-regulation NF may involve (i) providing physiological (body related) self-relevant information, (ii) self-awareness of control of cognition and brain activity, and (iii) learning opportunities to facilitate and strengthen strategies for self-regulation to control cognition.

Application and Demonstration of LORETA Neurofeedback in Treating College ADHD

Understanding the role of self-regulation as applied to NF may improve treatment of individuals with ADHD. Certainly, improving
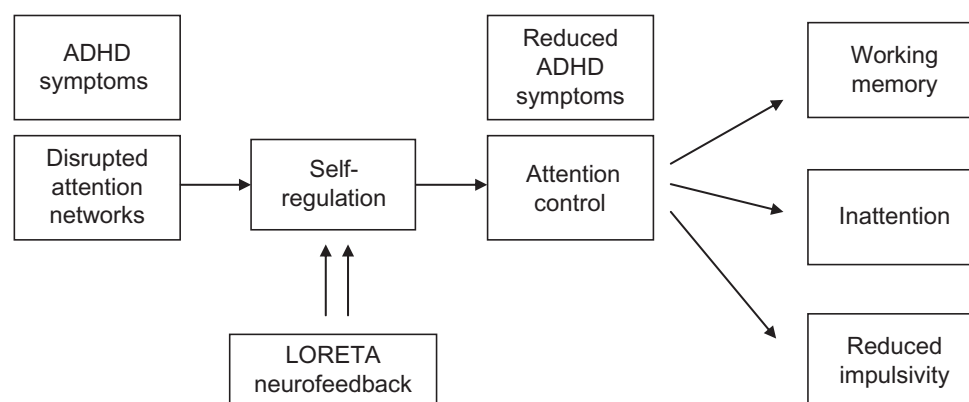
FIGURE 14.1 Theoretical model of LORETA mechanisms of change.

self-regulation skills would benefit college students with ADHD, and should be an integral aspect of treatment.

Figure 14.1 provides a working model of using LORETA NF as a self-regulatory intervention to address ADHD symptoms. As illustrated in the diagram, NF is used as an interactive process to facilitate self-regulation. Effectiveness is determined by linear trends in score change which demonstrate associated contingencies between self-regulatory control and changes in brain activity in prescribed brain networks. The individual's awareness in contingencies between self-regulation cognition and feedback from LORETA estimates provides stabilization and changes in broad attention networks. This "awareness" is a precursor to behavioral change.

LORETA and sLORETA Z-score Neurofeedback

Low-resolution electromagnetic tomography (LORETA)- and standardized LORETA (sLORETA)-based NF approaches are essential for estimating brain areas beyond surface EEG (i.e., areas involving self-regulation). LORETA does so via a mathematical solution to the EEG inverse problem. That is, it provides an estimate of current density in the 3D brain volume, thus providing an estimate of where in the brain the scalp-recorded EEG is being generated. sLORETA is a similar method, wherein the current density estimates are standardized with an estimation of the variability in the data, thus providing pseudo-F statistics as output rather than raw current density estimates. Although source accuracy is not perfect with low-density recordings, mislocalization tends to happen in consistent and predictable ways. Furthermore, LORETA results are consistent. Similar to EEG and quantitative EEG (qEEG), which have been shown to be highly reliable (Burgess & Gruzelier, 1993;

Corsi-Cabrera, Galindo-Vilchis, del-Río-Portilla, Arce, & Ramos-Loyo, 2007; Corsi-Cabrera, Solis-Ortiz, & Guevara, 1997; Gudmundsson, Runarsson, Sigurdsson, Eiriksdottir, & Johnsen, 2007; Roberts, 2012), LORETA results are highly reliable (Cannon et al., 2012).

LORETA Z-score Brodmann areas for attention networks overlap with regions involving the self, as previously identified. For example, selecting attention problems and executive functioning deficits in the symptoms checklist includes 12 Brodmann areas: 7, 8, 9, 10, 11, 19, 23, 24, 33, 45, 46, and 47. Many of these (i.e., Brodmann areas 7, 19, 23) overlap with the default network, and MCS, such as the anterior cingulate and precuneus (i.e., Brodmann areas 23, 24, 33). As previously mentioned, this overlap in "self" and attentional networks forms a core network integrating self-regulation with motivational states involving self-control of cognition. Improving functionality of this core system, although not curative, may mediate symptomatology of most psychiatric conditions, including ADHD.

Applications and Demonstration of LORETA Z-score NF

Case demonstrations will be reviewed from a research project examining the efficacy of LORETA Z-score neurofeedback in college-aged students with ADHD to demonstrate the general approach. We will provide information on ADHD symptomatology as well as neuropsychological test results. Additionally, preliminary results from two individuals who have been treated with LORETA Z-score neurofeedback training will be presented.

Consistent with guidelines for treating ADHD, multiple assessment approaches are used in diagnosing the disorder (American Academy of Pediatrics, Subcommittee on Attention-Deficit/Hyperactivity Disorder & Committee on Quality Improvement, 2001; Chatfield & American Academy of Pediatrics, 2002; Valente, 2001). Such guidelines often specify the need for parent and teacher ratings, which are often irrelevant, or unavailable for college-aged students. Students also often come to college previously identified as having ADHD.

Additionally, consistent with our conceptualization of ADHD as involving self-regulatory deficits, neurofeedback is viewed as an interactive learning process in which a person becomes aware of his or her brain functioning as mediated through EEG technology. The brain−computer interface used in neurofeedback provides the opportunity for an individual to learn and practice self-regulatory skills. However, for this research project, no attempts were made to engage the students in self-regulation.

Performance-based behavioral measures, as well as self-report rating scales, were administered for several reasons. First, such measures

provide a baseline for initial functioning status. Baseline scores can then be used as a comparison for judging treatment success posttherapy. Additionally, ADHD is behaviorally diagnosed disorder. As such, using behavioral measures can provide direct evidence of changes in symptom severity on a clinically relevant scale. For the case studies described below, preintervention baseline behavioral measures included three measures of short-term/WM, and a computerized attention task, which assessed omission and commission errors to provide measures of inattention and impulsivity (i.e., self-regulatory behaviors).

Consistent with the guiding theoretical model presented in this chapter and previous discussion on ADHD, a guiding treatment model for ADHD is to divide the individuals into two broad groups. One group involves individuals with ADHD symptoms who have identifiable disruptions in attention networks in the brain as indicated by atypical neurophysiological measures. This could be operationalized by specific Z-scores beyond a set alpha (typically two or three standard deviations from the mean). The other group represents individuals with ADHD who either have symptomatology not due to dysfunction in brain networks (i.e., typical neurophysiological test performance) or have dysfunction, which is not detected by standard clinical EEG measures.

For diagnostic purposes, it is important that clinical symptoms match the underlying neurophysiological problems causing the symptoms. As a preliminary guideline, the following specific steps are offered to guide the treatment of individuals with ADHD using LORETA Z-score neurofeedback:

1. Perform standard qEEG with eyes open and eyes closed
2. Determine if there are disruptions in attention networks that match symptomatology
3. Administer behavioral measures to quantify the degree of attention dysfunction (inhibition, inattention, etc.)
4. Teach and administer neurofeedback procedures and monitor progress
5. Readminister behavioral measures to determine the extent of progress
6. Readminister standard qEEG measures with eyes open and eyes closed to determine the extent of change in attentional dysfunction.

CASE STUDIES

In order to more fully demonstrate the utility of LORETA Z-score neurofeedback with a college population, cases from a study examining the effectiveness of LORETA Z-score neurofeedback training are

TABLE 14.1   Case Study Scores

	Numbers Reversed			Auditory Working Memory			Memory for Words		
	Pretest	Session 10	Posttest	Pretest	Session 10	Posttest	Pretest	Session 10	Posttest
Student A	18	19	22	27	34	34	20	20	19
Student B	14	15	16	29	31	32	17	19	19

	Omissions			Commisions			Reaction Time		
	Pretest	Session 10	Posttest	Pretest	Session 10	Posttest	Pretest	Session 10	Posttest
Student A	44.87	47.96	44.87	62.87	62.87	61.32	34.64	32.26	30.20
Student B	47.31	52.21	47.31	65.96	70.61	52.04	35.12	35.98	39.48

Note: NR, AWM, MW = Woodcock Johnson Tests of Cognitive Abilities, 3rd Edition; omissions, commisions, reaction time = Conners' Continuous Performance Test, 2nd Edition.

presented below. The study employed a delayed treatment design such that for the first 10 sessions, participants were randomly assigned to either a sham or treatment condition. After the completion of those 10 sessions, all participants received neurofeedback training. As such, two case studies are provided. The first is an individual who received neurofeedback training for the entire study, and the second, an individual who first received the sham condition, followed by LORETA Z-score training. Both individuals were 20-year-old female university students. Both were diagnosed in late adolescence by a general physician, and were prescribed stimulant medication to alleviate their symptoms. As the study took place during the academic year, students were not asked to stop their current medication regimen. The results described below were found in spite of their medication use.

Pre- and posttest behavioral data was collected at three time points—prior to session one, after session 10, after the final session—in addition to EEG data. Given that deficits in WM and self-regulatory processes are common in individuals with ADHD, the behavioral measures selected include three measures of short-term and WM—numbers reversed (NR), auditory working memory (AWM), memory for words (MW)—from the Woodcock Johnson Tests of Cognitive Abilities, Third Edition (WJ III) and the Connor's Continuous Performance Test, Second Edition (CPT-II), which includes measures of inattention and impulsivity. Both behavioral data and EEG data are presented (see Table 14.1) and discussed below.

CASE 1

TREATMENT CONDITION

Student A was a 20-year-old female who was diagnosed with ADHD at age 18. At pretest she reported symptoms of inattention and hyperactivity/impulsivity, though the latter was more prevalent. In terms of her short-term/WM performance, she obtained the following raw scores on the WJ III subtests: NR = 18, AWM = 27, and MW = 20. Raw scores were used because WJ III measures were constructed using item response models and there were minimal sources of developmental variation (Decker, 2008). Student A was also administered the CPT-II at pretest and obtained the following T-scores: omission errors (i.e., measure of inattention) = 44.87 commission errors (i.e., measure of impulsivity) = 62.87, and hit reaction time = 34.64. T-scores were used for this measure, as the raw scores obtained on the CPT are not directly interpretable.

CASE 1   (*cont'd*)

The first posttest was conducted after 10 sessions of NF. Student A's performance on the WJ III measures was as follows, NR = 19, AWM = 34, and MW = 20. This suggests that her performance stayed fairly consistent on two of the three measures, while she made a substantial improvement on the third measure, AWM. Her raw score of 27 at pretest corresponds to a standard score of 97 (M = 100, SD = 15). The 7-point increase in her raw score (AWM = 34) corresponds to a 16-point increase in standard score (SS = 113), which is greater than one standard deviation of change. In terms of the CPT-II, her midpoint scores were as follows, omissions = 47.96, commissions = 62.87, and hit reaction time = 32.26, suggesting that her performance remained fairly consistent, though she was a little faster at responding. At the second posttest (i.e., after the final session), her performance remained fairly consistent on both the WJ III (NR = 22, AWM = 34, MW = 19) and CPT-II (omissions = 44.87, commissions = 61.32, hit RT = 30.20), suggesting she reached a plateau, though the improvements she did make from pretest to the first posttest were maintained.

CASE 2

DELAYED TREATMENT CONDITION

Student B was also a 20-year-old female, who was diagnosed with ADHD at age 17. At pretest, she too reported symptoms of inattention and hyperactivity/impulsivity. However, per self-report, her distress was more equally distributed across the two domains. In terms of her short-term/WM performance, Student B obtained the following raw scores on the WJ III subtests: NR = 14, AWM = 29, and MW = 17. She also obtained the following T-scores on the CPT-II pretest: omission errors = 47.31, commission errors = 65.96, and hit reaction time = 35.12.

At midpoint, Student B obtained the following scores on the WJ III measures, NR = 15, AWM = 31, and MW = 19. This suggests that her performance stayed fairly consistent across all three measures, as would be expected given that she was receiving sham treatment. In terms of the CPT-II, her midpoint scores were as follows, omissions = 52.21, commissions = 70.61, and hit reaction time = 35.98, suggesting that her performance remained fairly consistent as well. While it is of note that her commission errors increased from Time 1 to

Time 2, her scores fell within the clinically significant range at both time points. As such, it is likely this was due to random variation, rather than suggesting that her performance worsened, especially considering the other measures were commensurate with her earlier performance. Given the delayed treatment design of the study, Student B was not expected to make significant gains from pretest to the first posttest measurement. However, it was hypothesized that she would begin to demonstrate changes by the end of the study.

At posttest 2, Student B's scores on the WJ III were as follows, NR = 16, AWM = 32, and MW = 19, suggesting little change in her WM as a result of the training. However, her performance on the CPT-II suggested the opposite. Her posttest 2 scores on the CPT-II were as follows, omissions = 47.31, commissions = 52.04, and hit RT = 39.48. Commission errors are indicative of impulsivity and her performance on this measure decreased from the clinically significant range into the "average" range after beginning the training.

Figures 14.2A and B illustrate the significant change from pretest to midpoint for Students A and B respectively. As shown below, Student A demonstrated significant change in alpha and posterior beta and high beta after completing 10 sessions of neurofeedback. As a caveat, it is possible that the full scalp alpha change could be due to nonspecific changes in alertness between the two sessions. However, the consistency in this change at both posttests suggests that the changes are likely due to more than chance. For example, as suggested by our theoretical model, self-regulation may produce nonspecific effects that account for changes in brain activity. Student B also demonstrated some significant change (i.e., theta, and some bilateral delta and high beta) during the sham condition, suggesting there was somewhat of a placebo effect as well. However, it is prudent to remember that these are individual cases, and as such, individual variation can have an immense impact on analyses such as these.

Figure 14.3A and B illustrates the significant change from posttest 1 to posttest 2 (end of the study) for both individuals. As expected, Student A showed significant changes in theta, alpha, beta, and high beta, as well as some highly localized change in prefrontal delta by the end of the study. This suggests that Student A continued to experience change following the first 10 sessions, likely building on the changes that occurred at the start of the study. This is most notable in the theta and beta (including high beta) ranges, which are often associated with ADHD. Student B also demonstrated additional significant change after receiving the real treatment. Specifically, this change was evident in
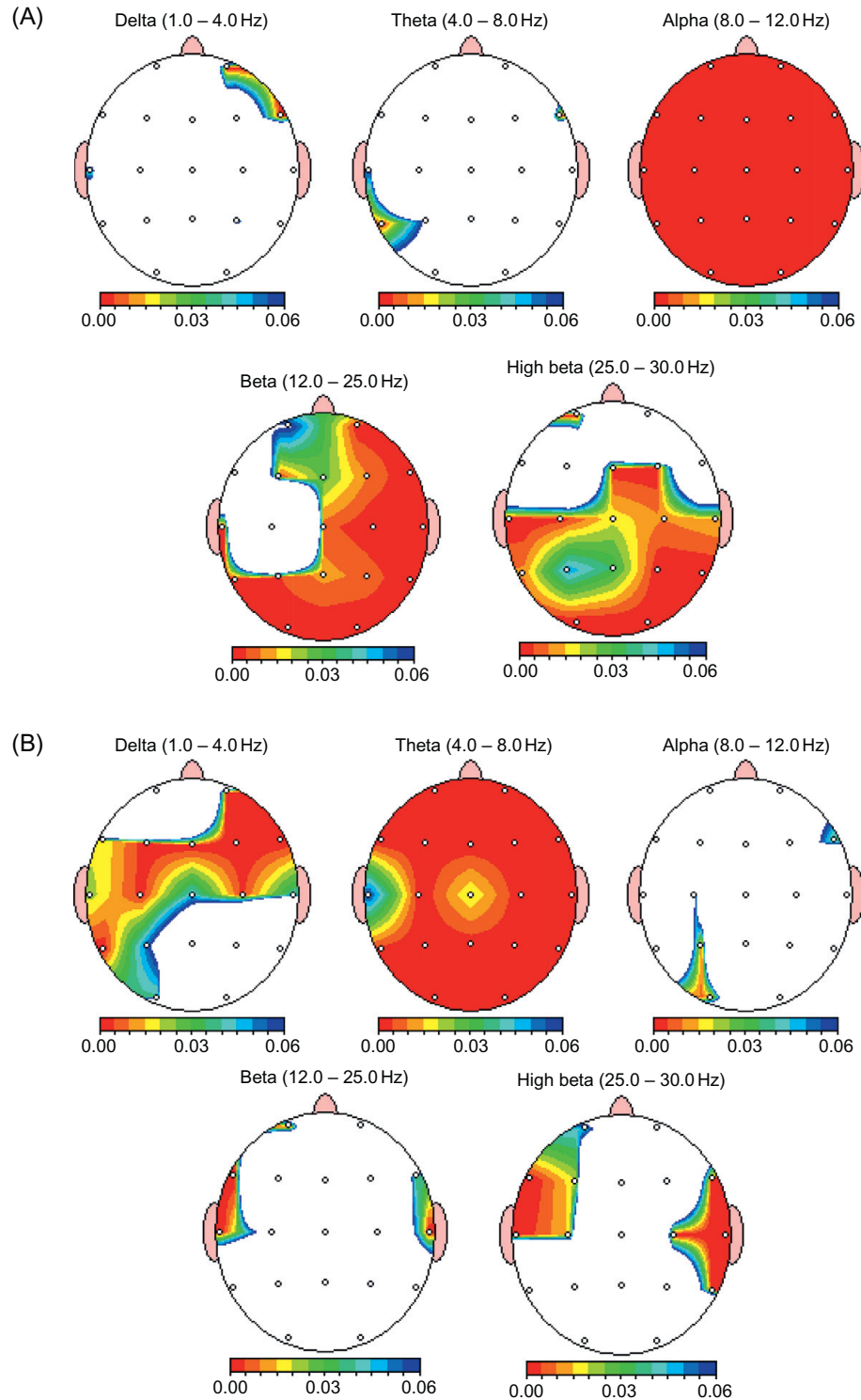
FIGURE 14.2   (A) Changes in absolute power from pretest to session 10 following NF treatment for Student A. (B) Changes in absolute power from pretest to session 10 following NF treatment for Student B. *Note*: Scales at the bottom designate *p*-values.
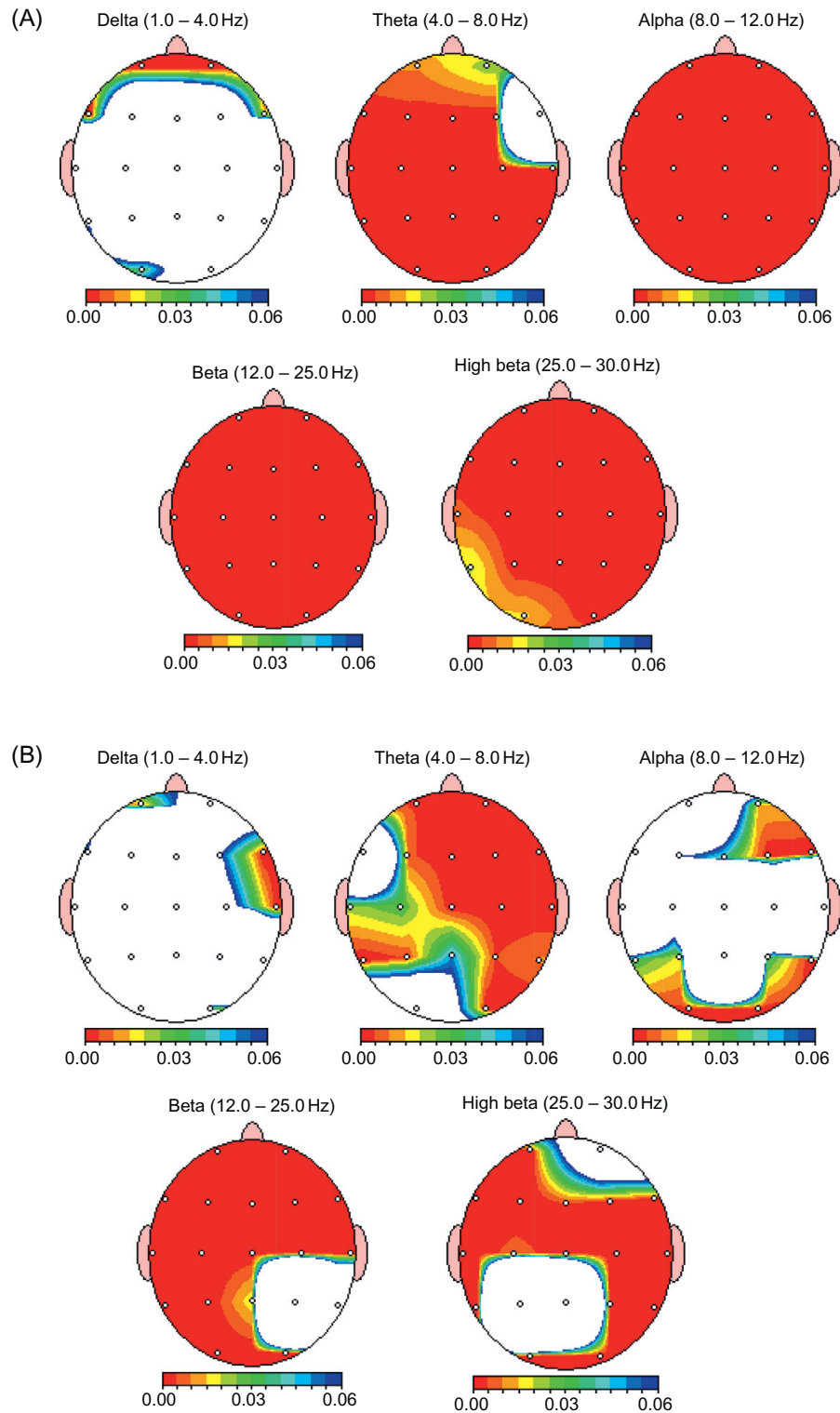
FIGURE 14.3 (A) Changes in absolute power from session 10 to posttest following NF treatment for Student A. (B) Changes in absolute power from session 10 to posttest following NF treatment for Student B. *Note*: Scales at the bottom designate *p*-values.

theta, beta, and high beta, as well as some posterior and right frontal alpha. Again, it seems as though the treatment impacted the theta and beta wavelengths the most, which is consistent with previous and current research, suggesting that the ratio of theta and beta waves is often atypical in individuals with ADHD.

The results of these two case studies are promising, as both demonstrated change in the expected directions. More interesting still, is that the impact of LORETA Z-score training was able to indirectly affect the students' performance on more commonly used behavioral measures as well. The implications of this are far reaching as many psychological disorders are diagnosed through the use of such tools, rather than through the use of neuroimaging techniques.

CONCLUSION

ADHD is a developmental disability that impacts individuals through adulthood. However, core deficits from ADHD are different in adulthood than in childhood. A core deficit in college-aged students with ADHD, and as specified in recent theories of ADHD, are deficits in self-regulation. Neurofeedback may not only provide a vital role in addressing self-regulation deficits but may also be viewed as a self-regulation treatment. Although there are terms to describe individuals with reduced sensitivity to visual input (visually impaired), and for individuals with reduced auditory sensitivity (hearing impaired), there is not a term to describe individuals who have reduced sensitivity to internal thought processes. In part, self-regulation fills this void. Case studies of college-aged students with ADHD were provided to demonstrate the general approach to demonstrating the use of LORETA NF to target core attention networks. It is the authors' hope that this chapter, and the presentation of the case studies, provides a basic overview of the use of this technique for those interested in the use of LORETA NF with a college population with ADHD.

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